SURGERY

MODULE 1 SURGERY 1

It has three units:

1. General introduction to surgery
2. Soft tissue injuries
3. Chest conditions

UNIT 1.

Definition. *A branch of medicine which treats diseases, deformities and injuries by manual or operative procedures*

INTRODUCTION

The management of surgical problems includes the following;

- Application of technical skills

- Training in the basic sciences to the problems of diagnosis and treatment and

- Genuine sympathy and love for the patient.

The surgeon must be a doctor in the old fashioned sense, that is,

- An applied scientist
- An engineer
- An artist and
- A minister to his or her fellow human beings.

THE HISTORY

Introduction- At first contact, the surgeon must gain the pt's confidence and give assurance that help is available and will be provided. He must demonstrate concern for the patient as a person who requires help and not just as a "case" to be processed through the surgical ward. Most pts are eager to like and trust their doctors and respond gratefully to a sympathetic and understanding person. Generally what is needed is a conducive atmosphere for the patient.

A formal history must be structured. Do not ask leading questions because the cooperative pt gives the answer that seems to be wanted; and the interview concludes on a note of mutual satisfaction with the wrong answer thus developed.

Building a history; History is detective. Avoid preconceived ideas, snap judgments, and haste conclusions. First determine the facts and then search for essential clues. A pt may conceal the most

important symptoms e.g passage of blood by the rectum hoping that if nothing is inquired about them nothing is serious. Special emphasis should be put on the more common surgical conditions. The symptoms include;

Pain –Analysis of the nature of pain is vital during hx taking.

- ascertain how the pain begun, was it explosive, rapid or gradual in onset?

- establish the character of the pain, whether so severe that it cannot be relieved by medications, is it constant or intermittent?

- Are there classic associations like rhythmic pattern of small bowel obstruction

- How does the pt react to the pain? Remember the overeater's description of pain is usually inappropriate and is described as 'excruciating' in a casual or jovial manner.

Vomiting; -ask what the pt vomited

- How much
- How often
- What the vomitus looked like
- Was it projectile
- Is it necessary for the examiner to look at it?

Change in bowel habits; this is common and usually of no significance, however, a person who has always had a regular evacuation notices a distinct change particularly towards intermittent alterations of constipation and diarrhea, colon cancer is suspected. Emphasis is placed upon the size and shape of the stool.

Haematemesis or Haematochezia; Bleeding from any orifice demands the most critical analysis and can never be dismissed as due to some immediately obvious cause. The most common error is to assume that bleeding from the rectum is attributable to hemorrhoids. The character of the blood is of great significance. Does it clot? Is it bright or dark red? Is it changed in any way, as in the coffee ground vomitus of slow gastric bleeding or the dark tarry stool of upper G.I.T bleeding?

Trauma

Trauma occurs more commonly that it is often difficult to establish a relationship between the chief complaint and an episode of trauma. Children in particular are subject to all kinds of minor trauma, and the family may attribute the onset of an illness to a specific recent injury. On the other hand, children may be subjected to severe trauma though their parents are unaware of it. The possibility of trauma having been inflicted by a parent must not be overlooked.

When there is a history of trauma, the details must be established as precisely as possible. What was the pt's position when the accident occurred? Was consciousness lost? Retrograde amnesia (inability to remember events just preceding the accident) always indicates some degree of cerebral damage. If the

pt is able to remember every detail of an accident, has not lost consciousness, and has no evidence of external injury to the head, brain damage can be excluded.

In the case of gunshot wound and stab wounds, knowing the nature of the weapon, its size and shape, the probable trajectory and the position of the patient when hit may be very helpful in evaluating the nature of the resultant injury.

The possibility that an accident might have been caused by preexisting disease such as epilepsy, diabetes, coronary artery disease, or hypoglycemia must be explored.

Family history

The family history is important in surgical illnesses. Polyposis of the colon is a classic example, but diabetes, peutz-jeghers syndrome, chronic pancreatitis, multiglandular syndromes, other endocrine abnormalities, and cancers are often understood and better evaluated in the light of a careful family history.

Past History

A detailed past history may illuminate obscure areas of the present illness. A pt with a long and complicated h/o diseases and injuries is likely to be a much poorer risk than even a very old pt experiencing a major surgical illness for the first time. It is easier to review the past history by inquiring about each system as you perform the physical examination on that part of the body. The nutritional background of the pt should be considered.

Emotional background

Emotionally and mentally disturbed pts require surgical operations as often as others, and full cooperation between psychiatrist and surgeon is essential. Before or after an operation, a pt may develop a major psychotic disturbance that is beyond the ability of the surgeon to appraise or manage. There are many situations in which the surgeons can and should deal with the emotional aspect of the pts illness rather than resorting to psychiatric assistance. This is particularly important in the care of pts with malignant dxes or those who must undergo mutilating operations such as amputations, ileostomy, or colostomy.

PHYSICAL EXAMINATION

A complete examination of the patient includes physical examination, certain special procedures such as esophagoscopy, laboratory tests, x-ray examinations, and follow-up examinations. Painful, inconvenient, and costly procedures should be avoided unless there is a reasonable chance that the information gained will be useful in making clinical decisions.

The elective physical examination

It should be done in an orderly and detailed fashion. One should acquire a habit of performing a complete examination in exactly the same sequence, so that no step is omitted. When the routine must be modified, as in an emergency, the examiner recalls without conscious effort what must be done to complete the examination later. All pts are sensitive and somewhat embarrassed to being examined. It is both courteous and clinically useful to put the pt at ease. The examining rm and table should be comfortable, and drapes used if the pt is required to strip for the examination. Most pts will relax if they are allowed to talk a bit during the examination, which is another reason for taking the past history while the examination is being done.

A useful rule is to first observe the pts general physique and habitus and then to carefully inspect the hands. Many systemic diseases show themselves in the hands [liver cirrhosis, hyperthyroidism, Reynaud's disease, pulmonary insufficiency, heart disease, and nutritional disorders].

Inspection, palpation and auscultation are the time honored step in appraising both normal and the abnormal. Comparison of the two sides of the body often suggests a specific abnormality. An example is the examination of a female breast when the pt raises and lowers her arms, will often reveal slide dimpling indicative of an infiltrative carcinoma barely detectable on palpation.

Successful palpation requires skill and gentleness. Spasm, tension, and anxiety caused by painful examination procedures may make an adequate examination almost impossible, particularly in children. Another important feature of palpation is the laying of hands that has been called part of the ministry of medicine. A disappointed and critical pt often will say of a doctor, 'He hardly touched me'. Careful, precise, and gentle palpation not only gives the physician the information being sought but also inspires confidence and trust. When examining areas of tenderness, it may be necessary to use only one in order to precisely localize the extent of tenderness. This is of particularly importance in examination of the acute abdomen.

Auscultation is now important in surgery than it is in medicine. Auscultation of the abdomen and peripheral vessels has become absolutely essential. The nature of ileus and the presence of a variety of lesions are revealed by auscultation. Bizarre abdominal pain in a young woman can easily be ascribed to hysteria or anxiety on the basis of a negative physical examination and x-rays of the gastro-intestinal tract.

Examination of body orifices

Complete examination of the ears, mouth, rectum, and pelvis is accepted as part of a complete examination. Palpation of the mouth and tongue is as essential as inspection. Inspection of the rectum with a sigmoidoscope is now regarded as part of a complete physical examination. Every surgeon should acquire familiarity with the use of the ophthalmoscope and sigmoid scope and should use them regularly in doing complete physical examination.

EMERGENCY PHYSICAL EXAMINATION

In an emergency, the routine of the physical examination must be altered to fit the circumstances. The history may be limited to a single sentence, or there may be on history if the pt is unconscious and there are no other informants. Although the details of an accident or injury may be very useful in the appraisal of the pt, they must be left for future consideration. The primary considerations are the following:

- Is the pt breathing?
- Is the airway open?
- Is there a palpable pulse?
- Is the heart beating? and
- Is massive bleeding occurring

If the pt is not breathing, airway obstruction must be ruled out by thrusting the fingers into the mouth and pulling the tongue forward. If the pt is unconscious, the respiratory tract should be intubated and mouth to mouth respiration started. If there is no pulse or heart beat, start cardiac resuscitation. Serious external blood loss from an extremity can be controlled by elevation and pressure. Tourniquets are rarely required.

Every victim of major blunt trauma should be suspected of having a vertebral injury capable of causing damage to the spinal cord unless rough handling is avoided. Some injuries are so life threatening that action must be taken before even a limited physical examination is done. Penetrating wounds of the heart, large open sucking wounds of the chest, massive crush injuries with flail chest, massive external bleeding all require emergency treatment before any further examination can be done.

In most emergencies, however, after it has been established that the airway is open, the heart is beating, and there is no massive external hemorrhage- and after anti-shock measures have been instituted, if necessary- a rapid survey examination must be done. Failure to perform such an examination can lead to serious mistakes in the care of the pt. It takes no more than 2 or 3 minutes to carefully examine the head, thorax, abdomen, extremities, genitalia (particularly in females), and back. If cervical cord damage has been ruled out, it is essential to turn the injured pt and carefully inspect the back, buttocks, and perineum. Tension pneumothorax and cardiac tamponade may easily be overlooked if there are multiple injuries.

Upon completion of the survey examination, control of pain, splinting of fractured limbs, suturing of lacerations, and most of emergency treatment can be started.

LABORATORY & OTHER EXAMINATIONS

Laboratory examination

Laboratory examinations in a surgical pts have the following objectives:

- Screening for asymptomatic dxes that may affect the surgical result (e.g, unsuspected anemia or diabetes,
- Appraisal of diseases that may contraindicate elective surgery or require treatment before surgery(eg, diabetes, heart failure),

- Diagnosis of disorders that require surgery(eg, hyperthyroidism, pheochromocytoma), and
- Evaluation of the nature and extent of metabolic or shock.

Pts undergoing major surgery, even though they seem to be in excellent health except for their surgical disease, should have a complete blood and urine examination. A history of renal, hepatic, or heart disease requires detailed studies. Latent, asymptomatic renal insufficiency may be missed, since many pts with chronic renal disease have varying degrees of nitrogen retention without proteinuria.A fixed urine specific gravity is easily overlooked, and preoperative determination of the blood urea nitrogen and serum creatinine is frequently required. Pts who have had hepatitis may have no jaundice but may have hepatic insufficiency that can be precipitated into acute failure by blood loss or shock.

Medical consultation is frequently required in the total preoperative appraisal of the surgical pt, and there is no more rewarding experience than the thorough evaluation of apt with heart dx or GIT dx by a physician and a surgeon working together. It is essential, however, that the surgeon does not become totally dependent upon a medical consultant for the preoperative evaluation and management of the pt. The total management must be the surgeon's responsibility and is not to be delegated. Moreover, the surgeon is the only one with the experience and background to interpret the meaning the meaning of laboratory tests in the light of other features of the case- particularly the history and physical findings.

Imaging Studies

Modern pt care calls for a variety of critical radiologic examinations. The closest cooperation between the radiologist and the surgeon is essential if serious mistakes are to be avoided. This means that the surgeon must not refer the pt to the radiologist, requesting a particular examination, without providing an adequate account of the history and physical findings. Particularly in emergency situations, review of the films and consultation are needed.

When the radiologic diagnosis is not definitive, the examination must be repeated in the light of the history and physical examination. Despite the great accuracy of x-ray diagnosis, a negative GIT study still does not exclude ulcer or a neoplasm; particularly in the right colon, small lesions are easily overlooked. At times the history and findings are so clearly diagnostic that operation is justifiable despite negative imaging studies.

Special Examinations

Examples include cystoscopy, gastroscopy, esophagoscopy; colonoscopy, angiography, and bronchoscopy are often required in the diagnostic appraisal of surgical disorders. The surgeon must be familiar with the indications and limitations of these procedures and be prepared to consult with colleagues in medicine and the surgical specialties as required.

THE DIFFERENCE BETWEEN SURGICAL AND MEDICAL CONDITIONS

Patients who present with surgical conditions only pose minimal to the surgical team. Their treatment revolves around the surgical condition with which they have presented. Surgical pts who present with associated medical diseases pose a great challenge and therefore require spe cial attention, because other than the surgical condition they have, the accompanying medical condition may complicate the surgical condition or vice versa. Essentially, this is a problem to both the surgeon and the pt himself/herself. The common medical conditions encountered include, DM, Hypertension, Cardiac diseases, Bronchial Asthma, Use of steroids, etc.

Diabetes Mellitus:

Diabetic pts undergo more surgical procedures than non-diabetics and management of the diabetic pt before, during and after surgery is an important responsibility of the surgeon. Control of fluids, electrolytes, glucose and insulin is important in the operating room .Marked hyperglycemia should be avoided during surgery; the greater danger is severe unrecognized hyperglycemia.

Heart diseases and the surgical pt

Anesthesia and surgery present a risk to any pt, but this risk is increased with pre-existing heart disease, whether clinically apparent or undiagnosed. Complications related to heart diseases are the major cause of nonsurgical perioperative deaths. Cardiac diseases may be exacerbated by many of the changes accompanying surgery, including fluctuations in heart rate, BP , blood volume, oxygenation, PH, and coagulability. These may lead to myocardial ischemia due to increased myocardial oxygen demand or reduced coronary blood flow, impaired myocardial contractility. This leads to altered cardiac performance due to changes in preload or afterload. Increased circulating catecholamines or sympathetic nervous system activity may precipitate arrhythmias as well as increase heart rate blood pressure.

Hypertension

CLASSIFICATION OF SURGICAL PATIENTS

Patients can be classified into three major groups;

a) **According to speed of surgical intervention.** This will depend on the condition of the patient. The patient could be;

i) Emergency case – This is the patient whose life is in danger unless immediate surgical intervention takes place. Examples include ruptured ectopic pregnancy, intestinal obstruction, bleeding cut wound and obstructed labor.

Ii) Elective (planned or cold) case. This is a patient who requires surgical intervention but the condition of the patient is not putting his life in danger and can be postponed to another time. Example include uncomplicated lipoma, hernias which are not complicated and patients with extra digits

b) According to whether the patient **should stay overnight in the ward** before operation or come in the morning of the operation.

1) Admitted (or overnight stay) case. This is usually a major case which requires preparation before operation and therefore patient has to be admitted into the ward.
2) Day case. Patient does not require to be admitted. He comes in the morning and is operated. The operation is minor. After operation the patient is observed and can be allowed home provided he is accompanied.

c) According to the **general condition of the patient.** In this group the patient is said to be either;

i) Fit – To undergo anaesthesia and surgery. (ii)Unfit – To undergo anaesthesia and surgery.

PRE – OPERATIVE MANAGEMENT OF A PATIENT

It will depend on the type of patient;

- Admit the patient to the ward through outpatient, SOPC, GOPC or any other special clinic from within the hospital.
- Take complete and relevant history
- Perform physical examination
- Do the basic investigations like the vital signs, grouping and cross matching, full haemogram, urinalysis etc?
- Do specific investigations like ultrasound according to the condition of the patient
- The patient should go to the ward with the results of the investigations to avoid unnecessary delay and time wasting.
- Explain to the patient the nature of his condition, why he should be admitted and the reason for operation
- Counseling to the patient, relatives or guardian
- Let everything that is done to the patient be documented
- Involve the relatives/guardians/caretakers be involved in the management of the patient
- Obtain an informed consent. A person to give consent should be above 18 yrs and mentally sound at the time of signing the consent. The consent is valid for between 24-48 hrs from the time of signing.
- Those who are not legible to sign are those less than 18 yrs, mentally unstable and those already premedicated.

Premedication;

Definition – The administration of drugs in the period of 1-2 hrs before induction of anesthesia and surgery.

Objectives – 1) Allay anxiety and fear by psychotherapy and anxiolytics usually benzodiazepine

2)Reduction of secretions by giving anticholinergics like atropine

3) Reduction of undesired reflexes(vagal reflexes which may produce severe bradycardia) like traction of the eye muscles may lead to bradycardia and arrhythmias known as oculo-cardiac reflex

4) Limitation of sympathoadrenal responses during induction and intubation which may lead to tarchycardia , hypertension and raised catecholamines. The responses are not desired especially in patients with hypertension, ischaemic heart diseases.

5) Produce amnesia (anterograde and retrograde) commonly by benzodiazepines

6) Post – operative anti emetics

7) Reduction of gastric volume and elevation of gastric PH. In patients with risk of vomiting or regurgitation e.g. metoclopromide.

In addition to the above different categories will require their specific pre-medications e.g. diabetics and hypertensive.

POSTOPERATIVE CARE

In modern practice, the patient is monitored and supervised closely and continuously during induction and throughout the operative procedure. However many problems associated with anesthesia and surgery may occur in the immediate postoperative period, and it is essential that supervision by adequately trained personnel is continued during the recovery period. In addition some major and minor complications of anesthesia and surgery may occur at any time in the first few days after the operation.

The early recovery period

Many hospitals have a recovery ward in close proximity to the operating theatre. A large number of recovery areas are closed at night and at weekends; at these times, and in hospitals with no recovery ward, the patient is supervised usually in corridor close to the operating theatre and often by inadequately trained staff. This section describes common problems which occur in the immediate postoperative period and refers specifically to their management in a recovery ward; however the same principles are applicable to recovery in other locations.

The recovery period starts as soon as the patient leaves the operating table and the direct supervision of the anesthetist. All the complications listed below may occur at anytime, including the period of transfer from operating theatre to recovery ward; in some operating theatre suites, the transfer to the recovery ward may last for several minutes, and it is essential that the standard of observation does not diminish during the journey. The patient must be closely monitored at all times.

Systems affected

Central nervous system

Consciousness may not return for several minutes after the end of general anesthesia, and may be impaired for a longer period of time. During this period, a patent airway must be maintained. There is a risk of aspiration into the lungs of any material, e.g. gastric content or blood, which is present in the pharynx. Consciousness may be depressed also in patients who have received sedation to facilitate

endoscopy or regional anesthesia. Excitement and confusion may occur during recovery and may result in injury. Pain may be severe if long acting analgesics have not been given during surgery.

Cardiovascular system

Peripheral resistance and cardiac output may be reduced because of residual effects of anesthetic drugs in the absence of surgical stimulation. Hypovolemia may be present because of inadequate fluid replacement during surgery, continued bleeding postoperatively or expansion of capacitance of the vascular system as a result of increased sympathoadrenal activity after restoration of consciousness, especially if analgesia is inadequate.

Respiratory system

Hypoventilation occurs commonly, usually as a result of residual effects of anaesthetic drugs or incomplete antagonism of neuromuscular blocking drugs. Hypoxaemia may result from hypoventilation, ventilation/perfusion imbalance or increased oxygen consumption produced by restlessness or shivering.

Gastrointestinal tract

Nausea and vomiting are common in the immediate postoperative period.

Staff, equipment and monitoring

The recovery ward should be staffed by trained and experienced nurses; one nurse must remain with each patient at all times. The responsibility for the patient's welfare remains with the anesthetist. In many hospitals, an anesthetist is designated to be available immediately to treat complications detected by the nursing staff. The patient is nursed in bed if a prolonged stay is anticipated, but more commonly on a trolley. All trolleys and beds must have facility to be tipped down. Suction apparatus, including catheters, an oxygen supply with appropriate facemask, a self inflating resuscitation bag and anaesthetic mask, and a sphygmomanometer must be available for each patient. In addition, there should be a complete range of resuscitation equipments within the recovery room; this includes an anesthetic machine, a range of laryngoscopes, tracheal tube , bougies, i.v. cannulae, fluids, emergency drugs, ECG monitor and defibrillirator. Facilities for cricothyroid cannulation, e.g. minitracheotomy set, or for formal tracheostomy should be available. A wide range of drugs should be stored in the recovery area for the treatment of common complications and also emergency events.

All patients should be monitored by measurement of heart rate, arterial pressure and respiratory rate and by assessment of level of consciousness, peripheral circulation and adequacy of ventilation; in some circumstances, minute volume may be measured using a respirometer. At least one mechanical ventilator should be available. Pulse oximetry is valuable, particularly in children, the elderly, patients with pulmonary disease and those with cardiovascular instability. Urine output should be measured routinely in patients who have undergone major surgery.

Wounds and surgical drains should be inspected regularly for signs of bleeding. The patient should n ot be discharged to the ward until;

1) Consciousness has returned fully, and a patient airway can be maintained
2) Ventilation is adequate and stable
3) The cardiovascular system is stable
4) Excessive surgical blood loss has stopped

High risk patients, or those who have undergone major surgery, should stay in the recovery ward for up to 24hrs. If this is not feasible, or if instability persists for longer than 24 hrs, the patient should be transferred to a high dependency or intensive care.

WOUND

Definition: A loss of continuity of skin or mucus membrane as a result of injury; soft tissue and bone may or may not be damaged.

Wound healing

The discontinuity in the surface of the body exposes the deeper tissues to the dangers of bacterial infections. This danger persists until such a time an intact surface has been restored by the healing process. Wound healing can occur in two main ways:

i) Healing by 1st intention
ii) Healing by 2nd intention

Healing by 1st intention

The main objective is to obtain rapid healing of the wound without an infection and with minimum scar tissue formation. This is achieved by accurately joining together the edges of the wound by stitches/sutures. This can be catgut, nylon, silk or vicryl.

Healing by 2nd intention

Occurs under three main circumstances:

1. When the wound edges are not brought together
2. When there is skin loss which is not made up for.
3. When wound becomes infected and breaks open or circumstances are such that it has been left open.

Healing by 2nd intention is the normal way in which an abscess heals after it has been drained. An ulcer heals in the same way. Healing by 2nd intention is much slower than healing by 1st intention and invariably involves formation of more scar tissue. The longer a wound remains raw the greater may be the amount of scar tissue formation and the greater will be the contracture and deformity.

FACTORS INFLUENCING WOUND HEALING

The nature and efficiency of wound healing process are influenced by various factors which may be local or general.

A. Local factors

1) **Presence of necrotic and devitalized tissues:** This produces irritation early and provides an excellent medium for bacterial growth. The leucocytes, the fluid loss and absorption of toxins are increased. This reaction around dead tissues delay wound healing because the tissues are engaged in defense process trying to eliminate the necrotic material and infection.
2) **Infection**: Produces varying degrees of tissue destruction and therefore greater delays healing. This is the commonest cause of delay in healing.
3) **Presence of foreign bodies**: Foreign may be introduced from outside or produced in the tissues themselves like the gallstones or urinary stones. Secretion/excretions escaping from their normal channel into neighboring regions acts as foreign bodies e.g. leakage of urine as in VVF, RVF and feaces acts as an irritant. Abnormal accumulation of blood or lymph and a mass of dead tissue such as sequestrum (dead bone tissue) also acts as Fbs. The reaction of tissue due to the presence of Foreign bodies depends on the physical and clinical nature of the fbs, the nature in which they are placed and the presence or absence of infection. If infection occurs in the presence of fbs there is profuse purulent discharge and unless fb is removed the wound will not heal.
4) **Local blood supply**: An adequate bld supply is absolutely necessary for formation of granulation tissues to form a satisfactory wound healing. An impaired bld supply may slow healing, inhibit fibroblast and weaken defense against infection. The bld supply may be interfered by damage to bld vsls, the initial trauma or presence of scar tissue. The presence of edema, congestion, hemorrhage and infection produces a swelling and also interferes with the blood supply. The signs of retarded wound healing as a result of impaired bld supply are as follows:

a) Delay in formation of granulation tissue which is composed of collagen and ground substance.
b) The tissues are pale and the wound is slow to epithelialise.
c) The line of incision shows little congestion and can be easily separated.
d) The skin flaps separate easily, die, and grafts don't take on such a surface.

5) **Rest**: Increased mechanical stress on the wound delays healing. Rest of the part is important especially to the wound of extremities near joints. Muscular action and joint movement disturb the in which cells are growing and results in extravasations of blood and fluid delay healing. Repetitive coughing, vomiting or intestinal obstruction frequently cause ruptures of abdominal wounds, commoner in obese people.
6) **Inaccurate skin apposition**: or a large tissue loss effect causes a delay in healing until the gap has been bridged.

7) **Hemorrhage**: Beside general reduction in blood volume and production of anemia bleeding into the wound adversely affects healing. Ideally blood coagulation should be just be enough to stick the part of the wound together and to eliminate the dead space in the wound. Collection of the blood as a result of injury or inadequate hemostasis during operation keeps the sides of the wound apart and does not allow the parts to come together. The hematoma increases the tension in the tissues and produces pain, ischemia, necrosis and delay healing. Hematomas are also an ideal media for bacterial growth as they have no circulation the ction cannot be controlled systemic antibiotics. Large hematomas which are slow to dissolve and those which haven't been evacuated surgically sometimes a cavity with rigid walls containing capsulated fluid.
8) **Irritation:** Direct irritation of the wound acts on living tissue and causes death of tissue and therefore there is delay or failure to heal of the wound.
9) **Type of suture**: The nature of the suture and method of suturing play a part in wound healing. Wounds stitched by catgut heal more slowly than those stitched with silk. They are two times stronger than those stitched by catgut.

B) GENERAL FACTORS

1) **Nutrition**: for normal and rapid rate of healing and normal physiological state is essential .If cellular nutrition is not maintained the body is unable to mobilize the defense mechanism and this favors the growth of bacteria.

a) **Protein deficiency**: may lead to delayed repair due to lack of collagen in the wound and dehiscence (bursting) is common in malnourished patients. The essential amino acids, cysterin and methionine are particularly important in wound healing.

b) **Vitamin deficiency**: the liver is concerned wIth metabolism and storage of the vitamins. The surgical procedures and injuries reduce hepatic functions. The vitamins which play a role in wound healing include:

i) **Vitamin C**: required for the production of intracellular cement and collagen tissue. Prolonged deficiency is associated with marked interfere nce of healing process. Deficiency of intracellular substance in the granulation tissue and capillary bed results, in hemorrhage in wound space. As a result of a prolonged lag period fibroblast is retarded and the development of tensile scar tissue is delayed.

II) **Vitamin K**: is essential for the control of hemorrhage. Deficiency results in hypoprothrombinemia which is commonly seen in jaundiced pts or those marked hepatic insufficiency.

III) **Vitamin A**: It combines with proteins to become an essential part of all specialized epithelial tissues. Lack may lower resistance to infections.

IV) **Vitamin D**: an adequate intake of vit. D is required for proper absorption of calcium and it's subsequent deposition in bone. Absorption of vitamin D may be seriously interfered with owing to impaired liver function due to surgical procedures (after).

2) **Anaemia**: There is little evidence that anemia alone interferes with wound healing. And when delay occurs there is an associated hypoprothrombinaemia

3) **Hormones**: administration of large doses of adrenocortical hormones slows the healing of wound because cortisone decreases the formation of collagen. Thyroid deficiency also slows

wound healing. Other infections causing syndrome= hyponatraemia, oedema, leukaemia, jaundice and syphilis. There is no clear cut evidence that any specific factor is involved apart from hypoproteinaemia and vitamin C deficiency.

4) **Age**: The young heal better than the elderly but the specific factors are not clear.
5) **Metabolic disorders:** Diseases like liver cirrhosis, DM retard wound healing. In uncontrolled DM, the tissues have diminished resistance to bacterial infection because the excess glucose provides a good medium for bacterial growth. The resultant tissue destruction in the wound by the organisms delays healing. There are certain obliterating changes in the arteries seen in DM affecting local blood supply.

TYPES OF WOUNDS

1. INCISED WOUNDS: Are wounds with minimal tissue damage. Are sustained as a result of cuts with sharp objects e.g. knives, broken glass etc

Characteristics- Have even margins bleed very easily and are caused by sharp objects which may also cause damage to the structures below e.g. nerves and tendons

Principles of management

They should be treated by suturing all structures including nerves and tendons within the 1st 6 hours of injury (primary suture).

2. **LACERATED WOUNDS**: A laceration or cut is the result of contact with a sharp object . It is the surgical equivalent of an incised wound. Once the cutting tool has gone deep to the dermis, there is less resistance in the subcutaneous tissues and the cut may penetrate to a considerable depth. Include wounds that are sustained in RTAs, industrial injuries etc. They are due to relatively blunt objects and are associated with certain amount of tearing.

Characteristics; -Edges and surface are ragged (not firm)

- Contain moderate amount of devitalized tissue

- Prone to infection

Principles of management

Are treated by wound excision (debridement), and primary suture of the skin within the 6 hrs of injury. Wound excision involves thorough cleaning, removal of all foreign matter and non viable or devitalized tissues. Each layer is tackled in turn and all devitalized tissues carefully trimmed away to expose a healthy bleeding surface. Debridement is aimed at converting the injury from jagged wound into one which is as near as an incised wound as possible.

NB: Certain amount of damaged but recoverable tissues should remain in the wound. For this reason it is unwise to repair deep structures such as tendons and nerves in such a wound. As soon as the skin wound heals (4-6 wks) a formal secondary repair of any divided tendons and nerves. Wound excision is carried out in a pt with a contaminated wound who arrive late foe treatment e.g. after 24 hrs.

Delay in treatment

As a rule treatment should be carried out within 6 hrs of injury. But under favorable conditions this time limit can be extended and the use of antibiotics makes it possible to delay primary suture for as long as 12 hrs. After 12 hrs treatment is best by delayed primary suture.

0-6 hrs = ideal

0-12 hrs = 1st (primary) suture

>12 hrs = delayed sutures

3. **CRASHED & DEVITALISED WOUNDS (WAR WOUNDS)**

Sustained in RTA, industrial accidents and in war e.g. gunshot wound and explosions.

Characteristics

1. Have ill defined margins.
2. Foreign bodies usually lodged in the wounds

The difficulties that exist in this type of wounds.

i. It is hard to differentiate viable from non-viable tissue with curtained.
ii. Because of extensive tissue there may be great swelling of the tissue and if primary suture is performed, the tension of the tissue will be so high resulting in local ischemia (leading to localwhich of tissues) which was previously viable.
iii. The wound may be heavily contaminated by bacteria.

PRINCIPLES OF MANAGEMENT;

Mainly, excision of the wound and delayed secondary suture.

a) Careful excision of dead tissues layer by layer.
b) Any dead muscle should be excised widely because dead muscles provide an ideal environment of multiplication of gas gangrene micro-organisms i.e. Cl. Welchi and Cl. Botulinum.
c) After excision the wound is not sutured but left open and dressed.
d) After 4-6 days the wound is re-examined and if confirmed that all remaining tissues are viable and the edema subsided sufficiently to allow the wound to be sutured without tension, delayed primary suture can be performed. Avoid tension and it is safer to do skin grafting even when

there is no skin loss. When the wound has healed and tension of tissues has returned to normal the graft can be excised and the skin sutured. If there is any doubt whether it is advisable to suture it should be avoided.

WOUNDS WITH SKIN LOSS

In wounds where skin has been lost the pt is in danger of superadded infection until the wound has been completely healed by epthelialization. Healing of skin layer is important because of need for its restoration and also because of the healing of deeper structures can take place in case of intact skin. Apart from that, the longer any exposed surface remains raw, the greater may be the rate of deformity and disability. In all wounds with skin loss the skin loss should be repaired as soon as practicable.

The following is the timing of wounds with skin loss:

* Clean incised wound with skin loss = primary grafting (0-12hrs

* Lacerated wound with skin loss = excision + primary grafting

*Crashed and devitalized wound with skin loss = excision + delayed primary grafting.

SECONDARY SUTURE

It is done by freeing the skin at the edge of a granulating wound then suturing. Now very rarely used but can be done under the following conditions:

i) When the treatment of the wound has long been delayed for one reason or another.
ii) When the wound has been infected and has to be left open for aeration. It has to be delayed until the infection has been controlled and tissues become healthy.

Because of the presence of granulation tissue and young scar tissue the edges of the wound will have become bound. In order to free the skin enough to be sutured it will be necessary to cut the skin edges.

Disadvantages:

i) Results in fresh skin healing.
ii) Even when the skin flaps are indurated and inelastic due to fibrosis and therefore difficult to suture. Secondary suture is encouraged. It is safer and easier to carry out secondary skin grafting and carry out reconstruction surgery later.

4 PENETRATING WOUNDS

Caused by sharp objects and penetrate deep into the body tissues. Wounds are very deceptive in that the sharp object can penetrate many inches with a mere slit in the skin as the only immediate obvious sign. Impaired movement or loss of sensation indicates injury to the nerves or tendons and

haemorrhage may be obvious. Penetrating abdominal wound may be symptomless until internal haemorrhage or peritonitis indicates:

- Swollen viscera
- Bowel damage or
- Damage of blood vessels.

NB: Penetrating abdominal wounds must be explored without delay to rule out peritoneal involvement..

Bruises, contusions and hematoma: A closed blunt injury may result in a bruise or contusion. There is bleeding into the tissues and visible discoloration. Where the amount of bleeding is sufficient to create a localized collection in tissues this is described as a hematoma. Initially this will be fluid, but it will clot within minutes or hours. Later, after a few days, the hematoma will again liquefy. There is a danger of secondary infection. Bruises require no specific management, and no treatment is of proven value. The pt should be advised that the time required for bruising to clear is extremely variable and in some sites, discoloration may persist for months. A hematoma may be evacuated by open surgery if large or causing pressure effects (such as intracranially), or aspirated by a large bore needle if smaller or on cosmetically sensitive sites. It may be necessary to await liquefaction and to perform repeated aspirations, with appropriate antiseptic precautions. A hematoma will generally reabsorb without scarring, but on occasions there may be persistent tethering of the skin. Blunt injury to the breast may result in an area of fat necrosis that be mistaken for a breast lump.

Wounds can also be classified:

a) **TIDY WOUNDS**: They are inflicted by sharp objects and contain no devitalized tissue. Such wounds can be closed primarily with the expectation of quiet primary healing. Examples include surgical incisions, cuts from glass and knives. Skin wound are usually single and clean. Tendons, arteries and nerves will commonly be injured in tidy wounds, but repair of these structures is possible. Fractures are uncommon in tidy wounds.

b) UNTIDY WOUNDS: They result from crashing, tearing, avulsion, vascular injury or burns. They contain devitalized tissue. Skin wounds will often be multiple and irregular. Tendons, arteries and nerves may be exposed, and might be injured in continuity, but will usually not be divided. Fractures are common and may be multifragmented. Such wounds must not be closed primarily. If they are closed wound healing is unlikely to occur without complications. There may be wound dehiscence, infection and delayed healing. Gas gangrene and death may even result. The correct management is wound excision (excision of devitalized tissue to create a tidy wound). Once an untidy wound has been converted to a tidy wound by excision it can be safely closed or allowed to heal by second intension.

WOUND CLOSURE

Wound closure can be achieved by a number of differing techniques. Most tidy wounds that do not involve loss of tissue can be closed directly. Where there is tissue loss a technique to import

appropriate tissue is needed. Reconstructive surgical techniques range from simple skin grafts to complex composite free tissue transfers.

ANTIBIOTICS ON THE TREATMENT OF WOUNDS

T he most important factor in the treatment of wound is avoidance of infection by careful operative treatment .If only healthy and well vascularised tissues remain in the wound edges the defense mechanism will be able to deal with all the organisms except the most virulent ones. In general antibiotics are indicated in extensive wound with much skin damage or if there has been any delay in treatment. They should be in the form of systemic penicillin for a minimum of five days. Don't start with broad spectrum antibiotics. Further antibiotic therapy depends on the result of bacterial examination of the wound pus swab.

NB: Antibacterial drugs must be used with discretion and only when the sensitivity the organi sm to various drugs is known, because indiscriminate use leads to resistance to drugs. Always give t.t to all pts with open wounds at any part of the body.

WOUND INFECTION

Patients complain of itchiness and pain on the wound. On inspection there is redness and swelling on the margin of the wound. Later small abscesses appear in relation to the stitches and small beads of pus can be seen surrounding the emerging stitches (**stitch abscesses**). An infection may spread to the surrounding subcutaneous tissue and this is called **cellulitis**. And collection of pus may form in the layers of the wound. At this stage the pt is usually very toxic with fever. If the infection is particularly virulent **septicemia** may occur.

TREATMENT OF AN INFECTED WOUND

Early stage: If treated before pus formation occurs it may be possible to arrest by use of systemic antibiotics. If stitches are tight, remove them to relieve tension.

Stitch abscess formation: Any infected stitches should be removed and antibiotic therapy instituted. If there are no deep collections of pus then infection may be arrested and the wound may heal.

When actual pus forms: Stitches should be removed and may result in sufficient separation of the wound to allow pus drainage. If drainage is not satisfactory the pt is taken to theatre, wound opened and drained and allowed to heal by second intension. Done under GA.

SPECIAL TYPES OF WOUNDS

a) **Insect bites:** Usually inflicted by wasps or bees. In sensitized people anaphylaxis may occur hence anaphylactic shock, circulatory collapse, coma and death may occur within a very short time (20 minutes) ,if the venom is injected directly into a vein as occurs in stings in the dorsum of the arm. Wasps are fond of alcoholic drinks and therefore picnicking people should their drinks.

Bees leave their stings and poison glands protruding from the wound. Squeezing should be avoided, instead prick them out. Pressure on the gland squeezes poison into tissues. The gland and the sting should be removed by scraping gently. Bee stings are acidic and should be neutralized by local application of methylene blue, ammonium, sodium bicarbonate (alkaline solution).

Wasps' venom is alkaline and should be neutralized by local application of an acid such as vinegar. Antihistamine drugs such as chloramphenicol mileate (piriton) should be given orally. Local application of antihistamine creams e.g. piperamine mileate (Anthesan cream). If an anaphylactic reaction occurs adrenaline 1:1000 0.5 ml should be given i.m every 10 minutes until pts recovers.

b) **Animal bites:** Bites from animals should be treated as any other wounds. Slightest suspicion that the animal is rabid; the wound should be freely excised. If possible the responsible animal should be kept under observation for any mental changes for about 7-15 days. If the is killed the brain should be examined for Negri bodies (rabies)

c) **SNAKE BITE**

Not all snakes are poisonous. In Kenya poisonous snakes are not commonly seen. Cobra, puff adders, vipers are examples seen in Kenya. Snakes usually feed at night since they know that is when their prey is available. They bite when provoked. Poisonous snakes may bite but not envenominate the victim, they have to prepare the poison. Snake poisons are different; some toxic to the brain or muscles or blood vessels, while some are combine. Bites by snakes are not likely to cause any fatal results but vomiting, giddiness and cardiovascular system collapse may occur.

Snake venom action	**mechanism of toxicity**
-Vasodilators e.g. kalibrein	hypotension
-coagulopathies e.g. Russel's vipers	consumptive coagulopathy
Echis. they activate factors v, x, prothrombin	
-Hyalurodinase (all venoms)	spread of venom
- Haemolysins e.g. vipers	bleeding from fang sites
- Haemorrhagins e.g. viper inhibits platelet activity	" " " "
Increasing vsl permeability	

- Eastern diamond black rattle snake is haemotoxic

and myotoxic e.g. rattle snakes Australian tiger snake, vipers

cobras, bothrobs, asper,taipans — local tissue necrosis

- Neurotoxins can* presynaptic, like mamba, puff adder

* Post synaptic like krails and cobras

* anticholinesterase — muscular paralysis Rs Failure

- Cardiotoxins e.g. burrowing asp, some elapids — coronary vasoconstriction Arteriovenous blockage

Venomous snakes	Antivenins
• Atractaspididae e.g. rattle black snakes	* none specific Rx symptomatically
• Colubridae e.g. brown tree snake	* brown snake antivenin
• Viperidae e.g. russell's viper, European adder	* viper/European viper antivenom
Subfamily crotalidae e.g. pit vipers, rattle snakes	* crotalidae polyvalent antivenom
• Elapidae e.g. cobras, coral snakes, mambas	* multivalent coral snake antivenom, Samir polyvalent, Thai red cross Cobra antivenom
• Hydrophidae sea snake	sea snake antivenom

It is difficult to predict which bites will produce symptoms or clinical outcome.

Ask the patient the time of bite, how long ago, the type of snake

- Apply tourniquet; if upper limb don't exceed 30 minutes, if lower limb don't exceed 60 minutes.
- Too tight tourniquet leads to ischaemia and a too loose one is also dangerous because it will allow the venom to go into the blood stream (tourniquet abuse).
- Excision of the wound and application of potassium permanganate should not be encouraged since they cause trauma, bleeding and necrosis. Some people use anti snake venom but the efficacy is not proven more so when the type of venom is unknown.
- Give anti snake venom as you prepare psychologically for resuscitation incase of anything.
- Be ready for any eventuality
- They cause anaphylactic shock and the efficacy is not proven
- Antihistamine can be an antidote to antisnake venom serum.

Management

- Use large bore i.v cannula on the unaffected limb
- Monitor Bp, coagulation, renal function, cardiorespiratory status.

NB; don't use Aspirin as analgesics because it may aggravate bleeding

- In severe coagulopathy with thrombocytopaenia causing DIC, large quantities of fresh frozen plasma, cryoprecipitate and platelets are required if response to antivenom is poor ,.
- Before antivenom therapy, ask for any history of allergy and do intra dermal sensitivity test before injecting, 0.02 saline diluted antiserum at site far from bite, observe injection site for about 10 min. for development of redness, hives, pruritus, other adverse effects.
- A syringe
- Of 0.5ml 1:1000 adrenaline (epinephrine) must be available when antivenom is given.
- If no reaction, give entire initial dose within 4 hrs of the bite.
- In severe envenoming, antivenom given upto 24 hrs after bite to reverse coagulation deficits.

There are three types of antivenom reactions;

a) Early anphylactoid
b) Pyogenic
c) Late.

If instant anaphylactoid reaction occurs, discontinue antivenom administration and give pt oral antihistamines or i.m adrenaline(0.5 ml of 1;1000). Infusion of antivenom can be restarted at slower rate. Corticosteroids are commonly given to treat serum sickness. If pulses are absent, query compartment syndrome and consider surgical assessment.

Indications for antivenom administration in snake bites

- Cardiogenic shock
- Neurotoxocity
- Rapidly progressive extensive local swelling
- Spontaneous systemic bleeding
- Haematuria
- Incoagulable blood
- Other evidence of haemolysis
- Rhabdomyolsis
- Bites on digits by snakes with necrotic venoms.

i) Anaphylactic shock due to reaction to the serum
ii) Efficiency is not proven

Anti-histamines are indicated for: a) Therapeutic effect (b) Antidote for anti-snake venom serum sickness.

d) **Scorpion stings**: common in desert areas. Can cause intensive pain and upsets for long periods. Patient responds well to emetine 65mg for an adult and smaller dose for children.
e) **Human bites**:

- Can be very fatal to life to life or to the limb itself. The wound becomes contaminated by so many types of bacteria including Vincent's organisms from the mouth. A common injury is an incised wound over the knuckles resulting from a clenched fist knocking the front teeth of another person.

Treatment

- Excise
- Antibiotic cover. You may use a broad spectrum antibiotics (high doses) e.g. tetracycline.

FOREIGN BODIES INTISSUES

Whenever an Fb is suspected, an x-ray of the part must be taken in at least two planes i.e. Antero-posterior (AP) and Lateral planes. This is done to radio opaque foreign bodies. For Fbs which are comparatively less radio opaque, personal observation and palpation is of great assistance . When removal is attempted the following are necessary, good light, ample time, bloodless field and electronic locator.

In cases where the Fb has been lodged in tissues recently, a redi sh track due to extravasated blood indicates the path of the Fb.

Te following are Fbs which are likely to lodge in tissues;

- Hypodermic needles
- Domestic needles
- Sewing machine needles
- Fish hooks
- Gravel
- Glass splinters
- Metallic sutures used in operations which sometimes break within a few days of insertion causing pain in the tissue. E.g. patella stout wire.
- Pieces of clotting in wounds caused by gunshots
- Swabs and packs may be found in the abdominal cavity especially during an abdominal operation causing irritation leading to tender palpable swelling within a few weeks. It is therefore important to count all swabs before and at the end of a operation before closure of the wound
- Radio opaque threads especially those in bank notes should be incorporated in all swabs so that if the swab count is wrong you can send the patient for an x-ray.

EXAMINATION OF A LUMP:

A localized swelling may arise from the local tissues like ; the skin, subcutaneous tissue, muscle, tendon, nerve or bone. Some swelling may originate from one tissue but attach to other surrounding ones. The mportant part of the examination is to determine the origin of the swelli ng.

Method;

Inspection

1. Determine the location of the sweeling
2. Describe the size (measure if possibleor relate to a commonly known object like a pea, tennis ball etc)
3. Shape- round or oval
4. Borders – regular or irregular

Palpation

1. Tenderness – asses whethermild, moderate or very tender.
2. Temperature – asses the temperature on the site of swelling and compare with other parts of the body that are equally exposed.
3. Surface –asses whether smooth or irregular
4. Consistency – soft, firm, hard, fluctuant or pulsating

a) Pulsating – place the index and middle fingers over the swelling . if pulsation is present, the swelling will be felt to move with every beat

b) Elicit for fluctuation or transmitted impulses

i. Use the index finger of each hand
ii. Place pulp of the tip of the right finger halfway between the center and the periphery of the swelling. (this is the " watching finger" and is kept motionless throughout the procedure)
iii. Place the left finger upon a point at an equal distance from the center diagonally opposite the first. (This is the displacing finger)
iv. Apply pressure on the swelling using the displacing finger
v. Feel for outward movement of the watching finger.
vi. If the "watching finger" is displaced by pressure exerted by the "displacing finger" in both axes of the swelling then fluctuation is present

c) Test whether it is possible to empty the swelling by compressing it and then noting any refill after releasing the pressure.

5. Mobility – using your fingers asses whether it is possible to mve the lump. Mobility of the swelling is determined by its attachment to the underlyingand or surroundingtissues

a. Highly mobile swellings are usually situated in the subcutaneous tissue space
b. Swelling that moves with the movement of skin or can be pinched with skin originate from the skin.

c. Swelling that move with every contraction or shifting of muscles in any direction originate from muscles or tendons
d. Fixed or immobile swellings –can originate from bone or indicate malignancy

6. Transluscency – to determine whether the swelling is cystic. The procedure of transillumination requires a powerful torch and a slightly dark room.

a. Place the torch on one side of the swelling
b. Observe for sparse illumination of the swelling

Note: Always remember to examine the lymph nodes draining the the area, nerves and distal pulses to the swelling.

ACUTE ABSCESS

Definition: A localized collection of pus as a result of reaction to pyogenic organisms.

Aetiology: Usually caused by invasion of microorganisms like, 1) staph. Aureus (2) Haemolytic streptococci and (3) E. coli in that order

The bacteria may reach any part of the body through three main routes;

By direct infection from out e.g. through penetrating wounds

Local extension from adjacent focus

By the blood stream (haematogenous) or lymphatyic

In the cause of haematogenous spread, there may be a predisposing factor like a bruised muscle causing extravasations of blood which forms a suitable media for multiplication of bacteria. Acute osteomyelitis may occur following a minor injury to a limb. Discharging sinuses are very suggestive of osteomyelitis.

Pathological process

Once the bacteriae have gained entry to the tissues they multiply and produce toxins and inflammation results.

The area is surrounded by a painful zone of acute inflammation which is infiltrated with leucocytes and bacteria.

Polymorphs contain a proteolytic enzyme which causes liquefaction of tissue into pus which is composed of bacteria and dead leucocytes

The tension in the abscess rises owing to the exudation of plasma and may spread along the paths of least resistance to the surface of the body or to a hollow viscus where the pus is eventually discharged.

Occasionally the resistance of the body is sufficient to destroy the bacteria before pus has found its way to the surface. The fluid is absorbed and either fibrosis follows or a cavity containing insipissated pus remains (hard dry pus). Such a condition may occur in the breast when an attempt to cure a breast abscess with antibiotics fails. When this occurs there is a resultant lump which is called an antibioma (antibiotic tumor). In some cases such as staphylococcal abscess of bones remain dormant (quiescent) but gives rise to flare ups of inflammation following local injuries or impaired general health and is called brodie's abscess and is a variety of chronic osteomyelitis. It is a cold abscess.

Symptoms of acute abscess

Patient feels ill and this depends on (a) size of abscess (b) virulence of the organism (c) the tension within the abscess

Throbbing pain which is characteristic of an abscess which becomes more severe if the affected part is dependent like the toes and fingers. This is because of greater bld supply to the area.

Fever

General malaise

Anorexia

Symptoms of acute abscess

General -raised body temperature -rigors may occur in severe

Local of acute inflammation abscess

The five classical signs of inflammation are present

Heat – the inflamed area feels warmer than surrounding tissue

Redness of the skin over the inflamed area due to hyperaemia

Tenderness due to the pressure of the exudates on the surrounding nerves

Swelling due to hyperaemia and inflammatory exudate

Loss of function. The inflamed tissue does not perform its physiological function

Severity of these signs depends on the extent of the inflammation and its proximity to the surface. The swelling is initially firm and edematous but later becomes soft and fluctuant. In some cases such as acute mastitis increasing edema is characteristic of a deep seated pus. If not treated an abscess tends to point. The skin or membrane covering it gives way and its contents are discharged giving relief.

Treatment

When an abscess threatens to form it can sometimes be aborted by antibiotics of adequate doses and long period.

Bed rest

Elevation of the affected part to improve venous return

Once pus has formed incision and drainage must be done. The incision should be made at an independent part (the lowermost part) of the abscess. ? Application of kaolin poultice or short wave diathermy promotes hyperemia.

If the abscess is situated in an area where there is danger of damaging structures (important) such s the neck, groin, or the axilla, a modified Hilton method of drainage should be used. This method consists of incising the skin and superficial fascia and opening the abscess using a pair of sinus forceps or artery forceps. By separating the blades of the forceps a sufficiently large opening can be made. A finger is then inserted and all the locules broken and converted into a single cavity. A drainage tube is inserted and left in situ, so that pus drains freely.

NB

Pus from an abscess must be cultured to determine the severity of the causative organism.

It is wrong to try to cure an abscess by use of antibiotics

Care must be taken not to incise an aneurysm mistaken for an abscess. In an aneurysm the swelling is characteristically pulsatile.

CELLULITIS

Def. Inflammation spreading along the subcutaneous or fascial planes, often as a result of infection by streptococcal pyogens. Formerly used to be called 'Hospital gangrene'. Gangrene may follow, occasionally resulting in widespread sloughing of tissues.

Aetiology

The commonest is streptococcal pyogens

In the pelvis E. Coli and strep. Faecalis may be responsible

This organism may gain entry into tissues through a minor accidental route such as a graze or scratch or as a result of an operation. If the general resistance of the patient is reduced by such condition s like D. Mellitus, alcoholism or renal insufficiency. cellulitis spreads rapidly and extensively. In this case septicaemia and pynemia may develop

Signs and symptoms

Depends on type of organism and extent of infection

Redness

Itching or stiffiness community at this site inoculators

Tenderness

Swelling

The general features of infection fever, malaise, amnesia may also be present.

Treatment

Bed rest

Elevation

Appropriate antibiotics

If pus is suspected incision should be made down to the deep fascia

Diabetic and other condition if present should be re..

CELLULITICS IN SPECIAL SITUATIONS

Cellulitis on the scalp

Usually results from infection of wound in the scalp. The infection may involve cranial bones and giving rise to osteomyelitis and even meningitis.

Orbital cellulitis

It follows wounds or spread of infection from one of the paranasal sinuses in the vicinity. It gives rise to protruding proptosis, impairment of occular movement, oedema of the eyelid and oedema of the conjectiva.The constitutional symptoms are often severe and there are two outstanding dangers of orbital cellulitis.

Thrombosis may extends the ophthalmic plexus of veins to the cavenous sinus and this may lead to meningitis

The globe of the eye may be infected leading to Panophthalmitis.

Owing to the risk meningitis all the wounds of the orbit used careful attention.

Cellulitis of the neck.

It may occur as a complication of infection in the mouth, tonsilitis, mastoiditis. The condition (cellulitis of the neck) is described as Ludwig's angina or submandibular cellulitis. The two main dangers are;

Oedema of the glottis

Downward spread producing mediastinits

Pelvis cellulitis

Commonest course in women is bilateral tear of the Cx during parturision.

In men it may be the result of rapture of bladder or other pelvic organs.

Cellulitis of scrotum

Occurs as a result of extravasations of urine due to rapture of the urethra. The infection spreads rapidly to the Sc tissue of the scrotum and later spreads to the abdominal wall. The Pt is usually very toxic**.**

BOIL (FURUNCLE):

It is an infection of a pilosabeceous unit with perifolliculitis, usually followed by suppuration and central necrosis. A "blind boil" is one that subsides without suppuration. Boils are common on the face, head and neck. Boils are frequently associated with overwork, worry, debility or other undermining influences. They may be presenting symptoms of diabetes mellitus.

CARBUNCLE

This is an infective gangrene of the **subcutaneous** tissue, which often occurs in the nape of the neck. The subcutaneous tissues become painful and indurated, and the overlying skin is red . Unless the condition is aborted by prompt treatment, extension will occur and, after a few days, areas of softening appear, the skin sloughs and discharges pus. Usually there is one central large slough, surrounded by a "rosette" of small of small areas of necrosis.

PYOMYOSITIS

BACTEREAMIA AND SEPTICEAMIA

They are due to the presence of organism in the bld as diagnosed before blood culture. In conclusion the organisms are merely present in the bld but they are not multiplying septicaemia the organism are present in blood and actively multiplying. The organisms must be present in large number so that the condition to manifest.

Clinical features

Usually features of severe infection i.e.

Intermittent temperature

Rigors

Icterus due to haemolysis of RBC or liver damage.

Treatment

Mainly depends on the causative organism (focus).

Specifically

- Blood for culture and sensitivity
- Antibiotics depending on the culture and can be changed if necessary
- Mainly aimed at preventing further formation of emboli.
- Correction of dehydration by infusion of I.V fluids.
- Administration of intensive antibiotics I.V as soon as possible
- I and D of abscesses where possible.
- If the condition is caused by thrombosis of the lateral intracranial sinus ligation and difussion of internal jugular veins may interrupt the stream of the emboli.
- Death usually follows due to abscess formation in vital organs e.g. heart and brain.

PYAEMIA

Def: A grave of septicaemia due to the circulation in the blood stream of septic emboli composed of masses of organisms, vegetation or infected clots.

The septic emboli lodge and grow in distant organs like brain, lungs, heart, kidney to form multiple abscesses. The organisms responsible are usually streptococci and staphlococci.

Predisposing conditions

- Thrombophlebitis
- Acute osteomyilitis
- ```````Infective endocarditis (bacterial endocarditis).
- Acute suppurative otitis media.
- Infection of an intracranial sinus.

Clinical features

- Repeated rigors.
- Intermittent temperature
- Rapid pulse
- Toxic pt and has a dry furred tongue
- Multiple abscesses may form in any part of the body. They are painful and tender but discovered as a swelling.
- Joints are occasionally affected and may become quietly disorganized.

Treatment:

- Mainly aimed at preventing further formation of emboli
- Administration of intensive antibiotics i.v as soon as possible
- Correction of dehydration by infusion of intravenous fluids
- Incision and drainage of abscesses where possible
- If the condition is caused by thrombosis of the lateral intracranial sinus ligation and diffusion of internal jugular vein may interrupt the stream of the emboli.
- Death usually follows due to abscess formation in vital organs.

ULCERS

Def: Discontinuity of an epithelial surface of skin or mucous membrane as a result of progressive destruction of cell by cell. The destruction of surface tissue is microscopic as distinct (different) from death of macroscopic portion such as occurs in gangrene or necrosis.

Ulcers have a tendency not to heal.

Classification

There are three main classes;

1. Non specific ulcers
2. Specific ulcers
3. Malignant ulcers

A. NON SPECIFIC ULCERS:

They are due to, infection of wounds, or physical or chemical agents. These are predisposing causes as in;

- Local irritation e.g. dental ulcer.
- Interference with circulation e.g. Varicose ulcers

Non- specific ulcers include:

- Traumatic ulcers
- Venous ulcers (venous diseases e.g. varicose veins).
- Trophic ulcers

- Ulcers due to arterial dx e.g. thromboangitis obliterans (buergers disease).

i) VENOUS ULCERS

Occurs either in connection with varicose veins or following D.V.T in which canalization of the deep vein has occurred but the valves are either destroyed or incompetent due to the damage. The underlying cause is venous stasis which leads to local tissue anoxia. Nutrition of the tissue is reduced and it breaks down leading to an ulcer.

Signs

- Medial aspects of the lower 1/3 of leg.
- Ulcer is superficial with a pale or hyperpigmented floor.
- The surrounding tissues and skin are hard
- It worsens in condition which cause impairment of venous return e.g. pregnant

Treatment:

Conservative treatment

- Admission
- Bed rest
- Elevation of the leg
- Daily dressing with lotion such as Eusol
- Exercises
- Pressure bandaging applied from the toes to the tibial tubercle

Surgical`1111111111111111111111111111111111111111111111111111111111111`1111

- Skin grafting when a good venous return has been established
- Stripping off of a varicose vein.

ii) TROPHICAL ULCER

They are due to impairment of nutrition which normally depends on adequate blood supply.

Caused by ischaemic and anesthesia

Trophic ulcers of the finger tips occurs in chronic vasospasms of the (Raynaund's disease, syringomyelia). Raynauds disease occurs in women and mainly the upper limbs. It is attributed to an abnormal sensitivity of arterioles to cold when exposed to cold the vessels go into spasms.

Syringomyelia is uncommon progressive dx in the nervous system seen mainly in adults. For an unknown reason there is cavitation and fibrous tissue reaction in the upper spiral cord and the brain stem and this

interferes with sensation of pain and temperature especially in the hands. As a result of loss of sensation to pain the pt neglects any minor injuries in the skin and this later turns into ulcers.

In the leg ischemic ulcers occurs around the ankle or around the dorsum of the foot.

They are very painful and resistant to local treatment.

The common causes of trophic ulcers due to anesthesia are;

- Leprosy

- D.Mellitus

- Spina bifida

- Tabes dorsalis

- Peripheral nerve injury

In leprosy and d. mellitus there is neuritis which leads to loss of sensation and ulceration.

Trophic ulcer due to anesthesia are called perforating ulcers.

They start in a cone and penetrate into it. The suppuration may involve the bones and joint and spread along facial planes upwards and even involve the cuff muscles.

Rx

Treat the underlying cause

iii) ULCERS DUE TO ARTERIAL DISEASE

Thromboangitis obliterans (buergers dx)

Condition characterized by occlusive dx of small arteries e.g. radial, tibial and ulna arteries.

Thrombophlebitis of superficial or deep veins

Found in male pts about 20 -30 years of age (under 30).

It associated with, excessive smoking.

It does not occur in women or non smokers.

Histologically localized inflammatory changes occurs in the walls of arteries and veins leading to thrombosis. Thrombosis leads to ischaemia and nutrition of the tissue is reduced finally leading to ulceration. Pt complains of pain on walking (intermittent claudication)

Pain on the cuff muscles while resting and limb ulcers due to buerger dx commonly progress to gangrene of toe and fingers.

Rx

Total abstinence from smoking which arrests the dx but does not reverse the established damage.

Administration of peripheral vaso-dilators e.g. presacolin

Lumber (or cervico dorsal) symphatectomy surgical excision of sympathetic nervous system. This is the most useful surgical procedure because it result in healing of the ulcers, skin nutrition is improved and relieves pain

Amputation maybe required.

B. SPECIFIC ULCERS

Syphilitic ulcers

Occurs in any of the three stages of the disease

Primary stage

A sore or chancre appears as the site of entry of the treponema pallidum. Occurs 10 – 90 day after infection.

A small indurated papule 1st appears. It becomes eroded and results in a classical ulcer known as hunterrian chancre. This is an indurate, rounded usually single painless with a slight raised well defined hyperaemic margin.

It exudes a serous discharge which is rich in treponema. Usually heals spontaneously in a few weeks.

In females the lesion may be situated at the

Fourchette

Clitoris

Labia majora

Cervix

In this situation it is not obvious and may pass unnoticed.

In males

Mucous surface of the prepare

Glans penis

Shaft of the penis (occassionally)

Scrotum

Lower abdominal walls

Contact with the early mucous lesion in the mouth may result in an extra genital chances in the lip and the tongue or the nipple.

The primary chancre is usually accompanied by a discrete mobile shotty (ball like) enlargement of the associate lymph nodes.

Secondary

Mucous patches may occur anywhere in the mouth or on the moist surfaces of the genitalia. These are small rounded, often transient superficial erosions. In the mouth the lesions join together to form the snail track ulcers.

Tertiary (5-15 YRS)

The characteristic lesion is called gumma. This is a mass of granulation tissue with central necrosis. Sloughing of subcutaneous gumma leaves a painless, punched out ulcer whose base has a wash leather appearance. On healing the ulcer leaves a silvery "tissue paper" scar.

Treatment

1. Saline compresses. No local Rx should be applied until the exudate has been examined for a negative syphilis by darkground microscopy. 3 successive days specimen should be taken

2. Anti syphilitic therapy

Procain penicillin 9000 units 1.m (7-14/7). Late syphilis Rx can be continued to(14-21/7).

Benzathene penicillin ("panadur"). A long acting penicillin dose 10cc (4.8 m.u) 5cc per buttock. i.m stat. or small doses weekly x3 doses.

TUBERCULOUS ULCERS

Commonly seen in the axilla, neck, or groin due to the breaking down of a tuberculous lymph nodes. Tuberclous ulcer of the tongue occurs in pt with advanced PTB or pharyngeal ulcers. The two cases are this day. It does not occur in pt under Rx.

A typical tuberculous ulcer is shallow and painful. The edges are undermined, irregular and bluish. The floor is covered with pale granulation. The base is soft and the discharge is thin and watery.

The regional lymph nodes are inflamed and matted together.

When a tuberculous ulcer of the skin or mucous membrane becomes chronic and will show small granulation in the subcutaneous tissue often referred to “apple jelly nodules”.

Long standing tuberculosis ulcers may sometimes undergo malignancy resulting in a haemangioma, squamous Cell Carcinoma (S.C.C)

Treatment

Bed rest

Good nourishing diet

Specific anti tuberculous therapy.

C. MALINGNANT ULCER

Squamous cell carcinoma (epithelioma)

Basal cell carcinoma (Rodent ulcer)

Squamous cell carcinoma

Predisposing factors

Burns

Contractures

Ulceration (chronic) due to e.g. tuberculosis and trophical ulcers

Sites:

Commonly lower 1/3 of the leg

But may occur on the scalp or anywhere in a burnt area especially those exposed to the sun

Prepuce of the penis

Characteristics of a typical squamous cell carcinoma.

Outline-irregular

Edges-Raised and ererted

Base-indurated has cauliflower appearance and later becomes attached to the deeper structures

Palpation-Friable (crumbles and bleeds easily).

Blood stained discharge occurs and decreases in amount when there is 2nd injection.

Region lymph nodes are enlarged and fixed.

Treatment

Biopsy for histology for confirmation of Dx

After confirmation of Dx wide excision should be done followed by skin grafting

Partial or total amputation of part involved e.g. la penis

DERMATITIS ARTEFACTA

Self mutation e.g. by application of irritants such as corrosive (hysterical temperament by doi ng something intentionally) or litigation (law suit) may be involved.

The ulcer is usually square and has straight edges.

Heal if protected by an undisturbed dressing.

TROPICAL ULCER

Def: An acute specific localized necrosis of a skin and subcutaneous tissues.

Occurs, as a result of, persistence of an acute stage of a known specific ulcer.

It is endemic in tropical region (India, Africa, in America)

etiological factors

1. Trauma: commonly initiated by very slight trauma e.g. thorn brick, scratch, mosquito bites of feet or leg.

2. Malnutrition: prevalent in people of poor diet, rare in agricultural commercial and city dwellers, therefore, a disease of the poor.

3. Fusiform bacilli and Vincent spirochaete found in the discharge in the early stages of the ulcer.

Flies: contaminate minor traumatic wounds thus formation of tropical ulcer.

Summary of the aetiological.

4 fs

Friction (trauma)

Food (malnutrition)

Fusiform bacillis

Flies

Clinical features

Commonest in males. Exposed to trauma than females

Age incidence 10 – 20 years.

Ulcer in commonest in the lower 1/3 of the leg (ankle joint).

Spreads rapidly and has a grey foul smelling slough.

Usually solitary but occasionally may be more than one

Ulcer is painful

Edges are raised and surrounding tissues edematous

Systemic upset general malaise for example.

Pathological process

Stage 1:

There is a painful lesion which is tense, accompanied by systemic upset e.g. malaise

A blister develops and breaks down with a characteristic foul smelling discharge.

Stage 2:

Tissue dies giving rise to a black slough

Slough separates, leaving an ulcer. Ulcer has slight raised edges. Floor is covered with granulation tissue which bleeds easily.

Ulcer penetrates and muscles, tendons and bones may be exposed on the floor of the ulcer.

Stage3:

Swelling of surrounding tissues subsides.

Pain subsides

Smell disappears

If no healing the ulcer becomes a chronic tropical ulcer.

Complication

Chronicity

Cancerous osteoma due to ulcer irritating the periosteum which reacts to the irritation and forms new bone.

Contracture due to a reduction in skin tissue and excessive scar tissue for formation which leads to

Malignancy to squamous cell carcinoma due to instability of the epithelium and frequent trauma.

Rx

Acute stage

Systemic penicillin x10/7

Bed rest

Raise foot on pillow

Daily dressing

In stage 2 the edge of the ulcer are excised and the floor of the ulcer curated

Local antibiotics are applied when healthy granulation tissue appears. Skin grafting can be done.

Chronic stage

If there is exostosis (overgrowth of bone tissue) the bones is chiseled back to its normal contour.

If there is death bone, sequestrectomy is done (surgical removal).

Contractures are released by plastic surgery.

Amputaion is done incase of malignancy.

Prevention

Encourage footwear (shoes)

Encourage balance diet

Destroy fly breeding places

Antiseptic in dressing new wounds.

LIFE HX OF AN ULCER

Consist of three stages

Extension stage

Transition stage

Repair stage.

Extension stage

Floor covered with exudates, sloughs while the base is indulated

Edge of the ulcer is sharply defined

Purulent discharge which may be blood stained.

Transition stage

Mainly or preparation for healing.

Floor become cleaner

Slough separates

Induration diminishes

Discharge becomes more serous

Small areas of granulation tissue appear at the floor and this areas join up until surface is covered.

Repair stage

Granulation tissue is transformed to fibrous which gradually contracts to form a scar.

Edge becomes more shelved and epithelium gradually extends from it to cover the floor at a rate of 1mm/day.

The healing age of the ulcer is composed of three zones:

Outer zone: consist of epithelium and looks white.

Middle zone: consist of granulation tissue covered by few layers of epithelium and is bluis h in color

Inner zone: Consist of granulation tissue covered by a single layer of epithelium cell and the zone is reddish in color.

CLINICAL EXAMINATION OF AN ULCER

Should be done in a systematic manner.

Aspects to be considered(S,S,S,E,F,B,D,L,P,D).

Site:

95% of rodent ulcer on the upper part of the face

Ca typically affects the lower lip where as a 1^0 chancre of syphilis usually affects upper lip

2. **Size:**

Length of Hx should be regarded e.g. ca extends more rapidly than a rodent ulcer but extends more slowly than an inflammatory ulcer.

3. **Shape:**

A rodent ulcer is usually circular shaped.

A gummatous ulcer is usually circular due to the joining together of multiple ulcers.

Square area or straight edge is suggestive a dermatitis artefacta.

4. **Edge:**

A healing non specific ulcer has shelving edge.

Tuberculosis ulcer is undermined

A rodent ulcer has rolled edge

Syphilitic ulcer has a vertically punched out or a square cut edge

Raised and everted is typical of a malignant ulcer.

5. **Floor:**

Is that which is seen by an observer e.g. may be watery or “apple jelly” granulations are typical in a tuberculous ulcer.

A granulation ulcer has a slough with a wash leather appearance.

6. Base:

Is what can be palpated under the floor.

It is indurated in a carcinoma or syphilitic chancre

May affect deep structure.g. a venous ulcer may be attached to the tibia.

7. Discharge

Prevalent shows active infection

Bluish greenish discharge suggests infection with psuedomonous pyocyaneus.

Watery discharge is typically found in TB

Blood stained discharged is typically found in extension phase of a non specific ulcer.

NB: Bacteriological examination of the discharge is necessary because there may be colonization of micro organism.

Spirochaete are found in 1 chancre.

8. Local lymph node: are not enlarged in rodent ulcer

In squamous cell Ca they may be enlarged, hard and fixed.

The inguinal lymph nodes draining a syphilitic chancre of the penis are found to be firm and shotty

The submandibular lympnodes that drain a chancre of the lip are grossly enlarged.

9. Pain:

Non specific ulcer in the extension and transition stages are painful with only one exception of anaesthetic (trophic) ulcer.

Tuberculosis ulcers vary e.g. a tuberculosis ulcers of the tongue are very painful.

Syphilitic ulcers are usually painless but anal chancre of the homosexual are painful.

10. Depth:

Should be recorded in MM or CM and anatomatically by describing the structure it has penetrated.

11.State of local tissue

Pay attention to local blood supply and nerve supply coz many ulcers especially those in the limbs are secondary to vascular or neurological dx

12. General examination.

Extremely important coz most systemic dxes present with skin lesion and ulcers as the only signs. The examiner must look for any evidence of debility, H/failure, anemia, diabetes mellitus.

A gummatous ulcer may be associated with other stigmatic conditions such as chronic glossitis.

13. Pathological examination

Are of value and must be done on pt with ulcer by:

Taking a biopsy to r/o cancer

Blood for kahn test/vDRL or wasserman test to r/o syphilitic.

Mantoux test R/o tb.

Sputum for A.A.F.B R/o TB.

Urinalysis to R/o diabetes mellitus

Bld for full haemogram

T

D

E.S.R chronic condition

Hb

Platelet count

Film

WBC

Discharge for bacteriology

Other- CxR R/o Tb or malignant metastasis.

X-ray can confirm or R/o.

Blood for H.I.V

LOCAL (TOPICAL) TREATMENT OF NON SPECIFIC ULCERS

Any underlying cause should be treated e.g. varicose veins, diabetes, arterial diseases. Many lotions and non-adhesive tapes applications are used to aid separation of sloughs, hasten granulation tissue and stimulate epithelialisation. The basic requirements of an ideal dressing are;

Maintain a high humidity between the wound and the dressing.

Remove excess exudates and toxic compounds

Permit gaseous exchange of oxygen, carbon dioxide and water vapor;

Provide thermal insulation to the surface and be impermeable to microorganisms;

Be free from particles and toxic wound contaminants

Allow easy removal with no trauma at dressing change

Be safe to use and be acceptable to the patient

Be cost effective

Antiseptics and topical antibiotics

Antiseptics can do more harm than good when used inappropriately. They can interfere with the normal healing process, are toxic to fibroblasts and may permit more virulent organisms to dominate. The routine use of antiseptics and hypocloride solutions should be avoided. If a wound needs cleaning, this can be achieved safely and more economically with normal saline

SUMMARY OF CHRONIC LEG ULCERS

NAME OF ULCERS	SITE	TREATMENT
1.Sickle cell anaemia	Medial malleolus	Bed rest Dressing Skin grafting Antibiotic if septic
2.Venus (varicose) ulcer	Medial malleolus	Bed rest Dressing Elevation Exercises

		Pressure banding Skin grafting Stripping of varicose veins
3.Squamous cell carcinoma	Variable	Wide excision after a biopsy Skin grafting Partial or total amputation
4.karposis sarcoma	Begins on the foot but can spread to other areas	Chemotherapy Radiotherapy Amputation
5.Malignant melanoma	Lower leg and sole of the foot (most common) but may affect front and back of trunk.	Surgical excision and skin grafting if confirmed early
6.Venous gangrene	Variable	Dressing Skin grafting Amputation
7.madura foot	Whole foot	Anti fungal agents: local & systemic Tab grisofulsion Amputation.
8.Yaw(Framboecia) by treponema pertinue	Lower leg	Penicillin as in syphilis
9.Leprosy(trophic ulcer)	Sole of feet Tip of fingers }	Anti tuberculous drugs Rest P.O.p Footwear Amputation Plastic surgery
9.Diabetess mellitus(trophic) ulcers form of gangrene	Toes and feet	Control diabetes Amputation if necessary.

SINUSES AND FISTULAS

Sinus: It is a Latin word meaning hollow. This is an abnormal blind track which is usually lined with granulation tissue. It leads from an epithelial surface into the surrounding tissues. It may be congenital like the periauricular sinus. The acquired forms follow inadequate drainage of an abscess. For example, a peri anal abscess may burst on the surface and lead to a sinus.

Fistula: It is Latin word meaning pipe or tube which is an abnormal communication between the lumen of one organ and the lumen or surface of another, or between vessels. Most fistulas connect epithelial lined surfaces. It may be congenital or acquired. Forms which have a congenital origin include brachia, trachea-esophageal and arteriovenous fistulas. The acquired type follow inadequately trained abscess, Example is when a peri anal abscess opens into the canal and the surface of the perineum.

Persistence of a sinus or fistula;

1. A foreign body or necrotic tissue is present e.g. a suture, hairs, a sequestrum, faecolith, or even a worm.

2. Inefficient or nondependent drainage

3. Unrelieved obstruction of the lumen of a viscus or tube distal to the fistula

4. High pressure, such as occurs in fistula-in-ano due to the normal contraction of the sphincter which force fecal matter through the fistula

5. The walls have become lined with epithelium or endothelium

6. Dense fibrosis prevents contraction and healing

7. Type of infection, e.g. tuberculosis or actinomycosis

8. Presence of malignant disease

9. Drugs, e.g. steroids, cytotoxics.

10. Malnutrition

Interference e.g. artefacta

Irradiation as in RVF for the treatment of ca cervix

Crohn's disease

High output fistula

Treatment

The remedy depends on the removal or specific treatment of the cause.

Proper drainage of abscesses

Scrapping is sometimes necessary to destroy an epithelial lining of a sinus or fistula

Rest or immobilization of the affected part.

BIOPSY

Definition: Excision of a tissue from a living body for microscopic examination.

Indications:

Tumors

For distinguishing a benign from malignant tumor. Useful for growths which initially are benign and have a tendency of becoming malignant e.g. rectal polyp.

For confirming the diagnosis before an extensive mutilating operation.

For guidance as regards the best line of treatment. The choice of treatment whether surgery or RT is to be employed may depend largely on the microscopic differentiation of the tumor. The choice may depend on radio sensitivity of the tumor.

Enlarged lymph nodes

Generalized lymphadenopathy with splenomegally due to various causes can be distinguished by a lymph node biopsy to make a correct diagnosis. For cases with generalized lymphadenopathy biopsy from the inguinal l'nodes should be avoided because this l'nodes can be enlarged due to any other cause and this might mislead the diagnosis. Neck or axillary l'nodes are advised (one or both).

Regional l'node biopsy can be used to diagnose TB of the joints in the vicinity.

Scalenic node biopsy: is the biopsy of the paratracheal and inferior deep cervical l'nodes used to confirm carcinoma of the bronchus. If they are invaded then it means that it is advanced and a radical surgery is contraindicated. It also helps in the diagnosis of carcinoma of the post nasal space.

Other conditions:

For investigation of endocrine disfunction of the endometrium by serological examination of the currated material.

Tissue biopsy is being used frequently to study the liver, kidney and mucosae of the G.I.T.

Types of biopsy:

Incisional (simple) biopsy

Consists removal of a small piece of tissue and take for examination

Usually done under local anesthesia

In case of suspected malignant tumor (growth), a tissue must be adequate and should be taken from the edge of the growth and include a bit of normal tissue to be representative to reveal the presence or absence of malignant cells. A wedge shaped piece of tissue should be incised. The piece of tissue should be picked with a needle and put in formation 40% (formaldehyde solution).

Excisional biopsy:

The whole mass is excised and submitted for histopathology. Done when the growth is small e.g. growth in the breast or a lymph gland. On the breast all growths are malignant until proved otherwise.

Needle biopsy:

It is done in two ways;

Aspiration of the tumor using a large bore lumina needle and submit the aspirate (smear) for histopathology.

By obtaing a core of tissue from tumors or solid organs by using certain specialized needles. The most commonly used are e.g. silverman needle and meghini needle.

The method is used in obtaining biopsy from the;

Liver

Spleen

Kidneys

Prostate

It is of value in confirming the diagnosis of inoperable ca and pts without superfi cial condition from which you can take an incisional biopsy.

Punch biopsy: Carried out in circumstances where tumors are deeply seated, e.g. vocal cords, esophagus, bronchus, urinary bladder and rectum.The tumor is visualized using an endoscope and a piece of tissue is removed by use of a special type of punch forceps.

Exfoliative (brush) biopsy: Valuable where pathological lesions are not readily accessible. For example tumor of the liver, esophagus, respiratory tract and body of the uterus, cervix. Smears are made from aspirated materialand cytological examination carried out e.g. ca esophagus - lavage of the esophagus followed by examination of fluid has led to the discovery of the carcinomaearly when radiology and esophagoscopy have been found negative. Exfoliative bx is rapidly gaining popularity but the only disadvantage is that it requires technical training and even if the smear is positive confirmation must be sought for.

Drill biopsy: An example is sterna puncture where bone marrow and bone tissue i s obtained by use of drill e.g. leishmaniasis and any bone cancer or blood cancer.

Biopsy by major operation: Example is laparatomy. I n this case a lesion in the abdomen such as a lymphoma is removed and sent to laboratory for histopathology.

Suction biopsy: Especially used for the study of the endometrium. The classical method is D&C and then followed by suctioning the curettage. If properly done it heals the endometrium oftenly. Its advantage is that it can be done in the office and can be performed in a greater number of pts.

Dangers of biopsy:

BURNS

A burn is a tissue injury from thermal (heat or cold) application, or from the absorption of physical energy or chemical contact. Each has its own distinctive features and management problems.

Burn depth depends, in thermal injury, upon:

- The temperature of the burning agent;
- The mode of transmission of heat;
- The duration of the contact.

Skin anatomy:

- Epidermis;
  - is the most superficial layer of the skin
  - It provides the waterproofing layer
  - It is constantly replaced from the basal layer
- Dermis;
  - Is the thicker underlyind area that supplies the strength and integrity of the skin
  - Is rich in blood supply from the subdermal capillary network
  - It contains adnexal structures – hair follicles, sebasceous glands and sweat glands. These adnexal structures contain epithelial cells that can proliferate and heal a partial thickness wound by epithelialization.

HEAT INJURY

Scalds –A burn caused by moist heat. Usually caused by hot water. It is the commonest cause of burns. The temperature of boiling water (100 degrees Celsius) or steam is constant and the major determinant of the severity of injury is the duration of contact. As with all burning accidents, those least able to protect themselves the very young, the very old and the very drunk) are particularly vulnerable. In most cases it causes partial thickness (dermal or superficial) or full thickness (deep) burns.

Fat burns – Cooking fat or oil has a much higher temperature (180 degrees c.) than boiling water and hot fat cools slowly on the skin surface. Spills therefore cause deep burns.

Flame burns – A wound caused by coagulative necrosis. They usually cause partial and full thickness burns. They cause coagulative necrosis. Flame burns have a varied etiology: house fire, clothing fires, spills of petrol on the skin, butane gas fires .They often occur in confined spaces and may be associated with inhalation burns.

Electric burns – The passage of electric current through the tissues causes heating that results in cellular damage, Heat produced is a function of resistance of the tissues, the duration of contact and square of the current. Bone is a poor conductor of electrical current, whereas blood vessels, nerves and muscles are good conductors. Low voltage such as from a domestic supply, causes significant contact wounds and may induce cardiac arrest, but no deep tissue damage. High voltage burns cause damage by two mechanisms: flash and current transmission. The flash from an arc may cause a cutaneous burn and ignite clothing, but will not result in deep damage. High voltage current will result in cutaneous entrance and exit wounds and deep damage. Electric burns therefore cause, full thickness burns, may cause ventricular fibrillation and death. Avoid touching the patient if the current is still on.

COLD INJURY Tissue damage cold can occur from industrial accidents due to spills of liquid nitrogen or similar substances. The injuries cause acute cellular damage with the possibility of either a partial thickness or full thickness burn. Severe cooling can freeze tissues and ice formation is particularly likely to cause cellular disruption. Frostbite is due to prolonged exposure to cold and there is an element of ischaemic damage. It causes coagulative necrosis.

FRICTION BURNS: The tissue damage is due to a combination of heat and abrasion. Examples include tight fitting wear or contact between surfaces like between the thighs of an obese subject.

PHYSICAL DAMAGE (Ionizing irradiation): irradiation may lead to tissue necrosis. Such injuries are extremely rare if industrial and medical safety precautions are working. Tissue necrosis may not develop immediately. These injuries are generally limited in area and surgical excision, and flap reconstruction may be appropriate management. Of greater significance is the long term cumulative effect of ionizing radiation in the induction of skin cancers and other tumors.

CHEMICAL BURNS This are caused by acids and alkalis. They cause inflammation, tissue necrosis and allergic skin responses.

CLASSIFICATION OF BURNS:

Can be classified as superficial or deep burns;

Superficial burns:

- They have the ability to heal themselves by epithelialization.
- ➢ Epidermal burns

- look red,
- are painful,
- Have no blisters
- Heal rapidly without sequelae
- ➢ Superficial dermal burns;
- Have blisters
- Are painful
- Heal by epithelialization within 14 days without scarring
- May sometimes leave long term pigmentation changes.

Deep burns:

- Have lost all adnexal structures and if left can only heal by second intension with scarring
- Deep dermal burns may be blistered and have blotchy (discolored spots) red appearance with no capillary return on pressure and absent sensation to pin prick.
- Full thickness burns have a white or charred appearance.
- Sensation is absent
- The charred layer consists of denatured , contracted dermis and is called an *eschar*.

ACCORDING TO SEVERITY

Extends through the epidermis as far as the moist superficial dermis

Are less than 15% in adults

Characterized by reactive process e.g. erythema, blistering

They may alter pigmentation of the healed scar.

Second degree (deep dermal) burns:

Involves the dermis but the skin structures are maintained

May heal spontaneously

Usually result in scarring

Can be defined as superficial burns of 15-25 degree burns or 10-20 in children or <10% in adults.

Third degree or full thickness burns or major burns

- They destroy all dermal elements

- Skin is white charred and waxy

- There is loss of sensation

Fourth degree burns : Involves the muscles and tissues of deeper structures

Fifty degree burns : They involve the bones

Sixty degree burns they involve internal organs.

Burn shock

The factors contributing to burn shock include;

Inflammatory reaction which leads to

Vast increase in vascular permeability

Loss of water, electrolytes, and plasma proteins from the intravascular to the extravascular spaces (hypovolaemia). Hypovolaemia occurs through

Loss in blister fluid

Loss in exudates

Oedema

Evaporation

The volume of the fluid loss is directly proportional to the area of burn.

Above 15% of surface area the loss of fluid produces shock

Overwhelming sudden and intense pain.

SEVERITY OF INJURIES DUE TO BURNS

There are four factors which determine the severity of burns; Extent of the body surface involved:

Patients with burns require intravenous fluid resuscitation if the injury is >10% in children or > 15% in adults.

Patients with smaller areas may require I.V fluids but not I.V resuscitation

Wallace's rule of nine (9) is used to estimate burnt area in adults i.e.

Head + neck = 9

Chest, posterior and anterior = 9 x 2 =18

Trunk, posterior and anterior = 18x2 = 36

Upper limbs = 9x2 = 18

Perineum = 1

Lower limbs =9x2 = 18

= 100

NB: The rule of 9 is inaccurate in children and as a rule of the thumb use the following: up to the age of 1yr the head is 18% and each leg 14%, for each following yr subtract from the head 1% and to each leg 0.5%

NB: The above rule does not apply to infants and the below 14 years because their head and neck are nearly 20% of the body surface area.

1year child's head+neck = 15%

Years child's head + neck = 13%

Therefore for children and adults use the patient's own palm which is equal to 1%.

The depth of burn and causative agent:

Burns can be partial thickness or full thickness;

Superficial Burns

These will heal spontaneously by epithelialization and can be divided into:

Epidermal Burns, these affect only the epidermis and examples are minor flash injuries and sun burn. Hyperemia occurs due to the production of inflammatory mediators; they are painful and heal within 7 days.

Superficial Dermal Burns (= partial thickness superficial), these affect the epidermis and the superficial part of the dermis. The blister is the most important feature. The exposed dermis is pink to white. The sensory nerves are exposed and the wound is therefore extremely painful. They heal within 14 days.

Deep Burns

These are more severe and will only heal after a prolonged period with significant scarring. They are divided into:

Deep Dermal Burns (= partial thickness Deep), there may be some blistering but there is the appearance of blotchy red discoloration. The important feature is the absence of capillary refill. The dermal nerve endings are destroyed resulting in loss of sensation to pinprick.

Full thickness Burns, both layers (epidermis and dermis) are destroyed; may penetrate underlying structures. They have a white, waxy or charred appearance. The important feature is the leathery appearance which is called eschar. There is no pain sensation. Examples of full thickness burns are, those from burning clothes, electricity and mottled metal.

Diagnosis of burn depth

Depth	color	Blister	Capillary Refill	Sensation	Healing

Epidermal	Red	No	Present	Present	Yes
Superficial	Pale	Present	Present	painful	yes
Dermal					
Deep Dermal	Blotchy	+/-	Absent	Absent	No
Full thickness	White	No	Absent	Absent	No

Age of the patient: Individuals of extreme ages the prognosis is poor. NB Not all burns in children are accidental (battered child syndrome). So be careful with the mother's explanation. They satisfactorily defend it.

Associated illness or injury: Concurrent illness increase mortality. Existence of cardiovascular, renal and metabolic disorders must be established (r/o). Any associated fractures increase fluid requirement and the fractures should be treated by conservative method (closed) e.g. manipulation under G.A. Open methods should be avoided to make the skin intact to prevent infection. Crystalloids e.g. hartman's solution(Ringer's lactate) are suitable because they are close to the plasma.

OTHER METHODS OF ESTIMATION OF BURNS

Rule of 5 (for >1 yr) – Head 15%

Head 15%

Chest A/P 15%

Upper limbs 30%

Abdomen A/P 15

Lower limbs 30%

The rule of 10(< 1yr)

Head 20%

Chest a/p 20%

Upper limbs 20%

Abdomen 20%

Lower limbs 20%

Fingers and palm

For small burns and it is approximately 1%

The lund and Browder chart (the most accurate and takes into account the age of the patient)

Check from Adobe

Respiratory burns

All patients with suspected respiratory burns or smoke inhalation should receive humidified oxygen with mask and regular breathing exercises. Administration of crystalloid fluids (e.g. normal saline).Dextrose should be restricted to amounts that are necessary to maintain life.

Satisfactory hydration and renal function. Patients with oral or nasal burns who develop stridor and respiratory distress may have laryngeal oedema. Endotracheal intubation should be done before total obstruction occurs. Tracheostomy should e carried out if intubation is impossible or prolonged lower RS tract burns cause edema and bronchiospasms. Treatment may include administration of aminophylline and if no relief then steroids are indicated. Severe cases may develop respiratory failure in which case mechanical ventilation's indicated.

IMMEDIATE CARE OF BURN PATIENTS

Pre-hospital care

Ensure the safety of the rescuer

Stop the burning process, stop drop and roll(to extinguish a burning person).

Check of other injuries ABC followed by rapid secondary survey to rule out other missed items.

Cool the wound – to provide analgesics and to delay microvascular drainage.

Give oxygen- anyone involved in a fire in an enclosed space, any one with altered levels of consciousness.

Elevate- to reduce swelling and discomfort.

HOSPITAL CARE

NB: Before hospital admission cover with a clean sheet

In hospital either expose or dress with one of the available topical agents.

Avoid use of tight dressings in limbs with compromised circulation

Admission criteria

Age

Neonates	always (often deep burns)
Babies (< 1yr)	TBSA >5%

Children	>8%
Adults	>15%

Site

Head and neck

Hands and feet

Groin and axilla

Perineum

Circumferential burns of the chest and limbs

Depth

Full thickness burns >5%

Special burns

Electrical, chemical and inhalational burns

Others

Social indications. If in doubt admit overnight and reassess next day.

Immediate general evaluation and treatment

Patient should be assessed in a closed warm room and treated as follows;

Emergence sedation

Reassure the patient and keep in a quiet place

Remember full thickness burns are painless

Partial thickness are relieved by cold water compresses and dressing

Opiates like morphine may cause vomiting and respiratory depression and should be used with caution and given in small doses i.v. In children the sedative of choice is trimeprazine tartarate (vallergen).

Fluid resuscitation

Untreated patients can develop hypovolemic shock because of fluid loss from burn wounds or by inflammatory edema.

The volume of fluid loss is proportional to to the surface area. The larger the area, the more the fluid loss. The rate of loss is maximal immediately after burns. The rate diminishes during the first 36hrs. The fluid lost resembles plasma and mainly electrolytes. The fluid should be replaced by colloid solutions like Dextran. Calculation of the rate of administration of fluid formulae are many and here are some of the examples;

PARKLAND (BAXTER) FORMULAR

Crystalloids are used in the early phase of fluid management. Ringer's (Hartman's) solution is the one internationally accepted. The advantage is that it has potassium, sodium and calcium. Use 4mls of crystalloids/kgbwtx burnt area for the first 24 hrs . For example a 70 kg man with 30% burns.

4X70x 30 =8400mls. Half of this amount should be given during the first 8hrs i.e4200mls.

NB: The first 8hrs start at the time the patient was burnt. If the patient comes 2hrs after the burn, you must still give 4200mls in the next 6hrs. ¼ of the remaining fluid (2100 mls) is given in the next 8hrs while the remaining 4200mls is given in the next 8hrs.

Ringer's lactate is preferred because it is balanced and most close to the extracellular fluids. It contains sodium, calcium and potassium chlorides which are beneficial in eliminating lactic acidosis in untreated cases of burn shock. The total body surface is what matters in the formular. During the next 24hrs emphasis should be restoration of plasma volume. Hartman's soln is still the fluid of choice. By this time the integrity of the capillary wall has been restored. At this point colloids become useful. The amount of colloids (plasma) to be administered depends on the extent of burns. In general burns less than 50% of the body surface area rarely require more than 700ml of plasma.

Burns > 50% of the body surface area may require about 1500ml of plasma. Ringer's is continued to maintain adequate sodium and adequate urine output.

BROOK (BROOK ARMY HOSPITAL) FORMULA

In the first 24hrs;

Bwt in kg x % burns x 0.5 ml = amount of colloids e.g. dextran, plasma or blood.

Bwt in kg x % burns x1.5 ml (crystalloids) e.g. Hartman's/ Ringer's lactate

Normal insensible loss should be taken care of (replaced) by administration of dextrose in water r(5% dextrose. Adults= 2000ml, Children up to 1yr =8o ml/kg bwt, 5yr= 60ml/kg body wt and 8yr= 40ml/kg bwt.

First half of the calculated amount of brook formula id given in the first 8hrs. The remainder is given in the next 16 hrs. i.e.

½ = first 8hrs

½ = next 8hrs

½ = next 8hrs

In the next 24hrs the amount required is about ½ of the first 24 hrs.

NB: I n this formula calculations are used on 50% body surface as the maximum % to avoid overhydration.

Example: 70kg man with 60% burns

crystalloids =70 x50 x1.5 = 5250 ml.

colloids = 70 x50 x0.5 = 1750 ml.

insensible loss in adult = 2000 ml.

Total = 9000 ml

Summary of Brook army formula – 0.5ml = colloids

1.5 ml = crystalloids

3. EVANS FORMULA

Total amount of fluid needed per day is calculated in kg x % burn x2

50% = crystalloids

50% = colloids

Insensible loss = 5% dextrose

Example: 70kg man with 20% burns

70 x20 x2 = 2800ml= 1400ml crystalloids

= 1400ml colloids

Hourly assessment of the patient must be met and fluid therapy adjusted accordingly.

ADEQUACY OF FLUID REPLACEMENT

Several parameters are important in gauging success in treatment of shock. Over hydration may lead to CCF, pulmonary edema or both.

Laboratory parameter

Serial hematocrit (PCV) are used to guide in the plasma volume. Hematocrit of > 45% suggests either low plasma volume which can be corrected by giving additional albumin or plasma. Normally in male is between 45-54, female 36-37 % and children 32-475.

Blood urea, electrolytes and creatinine clearance.

Urine for electrolytes.

Clinical parameters

Hourly urine output .It is desirable to have a urine output of about 20-30 ml/hr and should not exceed 100ml ml/hr (i.e. 0.5 – 1ml/kg/hr) at any age.

Normal urine output;

15 yrs old 15-20ml/hr

5-10yrs 10ml/hr

Infants up to 5ml/hr

Catheter should be inserted to drain urine. When output exceeds 100ml/hr the rate of fluid administration should be decreased.

Pulse rate

Blood pressure

Temperature

Jugular and peripheral vein filling.

NB The most important is the urine output. The clinician should be aware of the early decrease in GFR. Also check urine clinically for glucose since a large urine output may be due to diuretic osmosis secondary to glycosuria. You must be certain that the patient is not in one of the phases of renal failure.

CRITERIA FOR ACUTE ADMISSION.

Suspected airway or inhalational burns

Any burns likely to require fluid resuscitation

Burns of any significance to the hands, face, feet, or perineum.

Any burns likely to require surgery

Any suspicion of non accidental injury

Patients of extreme ages

Patients whose psychiatric or social background makes it inadvisable to send them home

Any patient with associated potentially serious sequalae including high tension electrical burns and concentrated hydrochloric acid burns.

INITIAL CARE OF BURN WOUNDS

Good care of wound is one of the most demanding and challenging problems. The objective is to obtain complete healing in the shortest time possible .Initially the burn wound is sterile due to thermal destruction of the flora of the skin. Within 24 hrs the burn wound is colonized by a mixture of flora in which gram positive cocci dominate and the common cultured from the burn wound are;

Staph. Aureus

Staph epidermidis

Diphtheroids

All in the first to third day.

Group A beta haemolytic streptococci is the most virulent and can easily convert a partial thickness burn to full thickness burn. A prophylactic low due of penicillin the first 5 days especially in extensive burns should be given Erythromycin can substitute penicillin. Broad spectrum antibiotics should not be given unless there is systemic infection to avoid resistance.

About 4-5 days following burns there is a gradual change of organism that colonize the wound such as Gram negative- klebsiella, pseudomonous aerogenosa, enterobacter, E. coli, providential stuarti (all in the 4^{th}-5^{th} day).

In the second group pseudomonous is the main offending organism (pseudomonous aerogenosa). The majority of burn wounds don't require cleaning, but adherent clothing, dirty of foreign bodies should be removed by gentle rinsing with warm sterile water. It is not necessary to rupture clean blisters because wound healing is more rapid in an intact blister than a broken blister. If a full thickness burn encircles a trunk or limbs (circumferential) escharotomy (eschar – slough forming after burns and is anesthetic) then the dead skin should be incised along the axial lines to prevent the trapped edema producing a tourniquet effect and eventually there will be ischemia leading to ischemic contractures or gangrene. Escharotomies release tourniquet effects but may cause hemorrhage which may necessitate transfusion.

Before starting any form of wound treatment it is essential wound cultures of the burn area to be carried out. Burn wounds can be treated by: -

Closed method

Open method

Excisional method

CLOSED METHOD

Tulles: - this is gauze dipped in paraffin or antibiotics.It may be oil based tulle e.g. sofratulle or water based creams. It may contain agents such as nitrofurantoin, soframycin or chlorhexidine . The burn is cleaned with a mild antiseptic or normal saline then a layer of tulle is applied. This is followed by an absorbent dressing then a bandage is applied.

Topical antibacterial agents :- There are various agents such as; (a) mafenide (sulphamyci), (b) 0.5% silver nitrate, (c) 1% silver sulphadiazine. The use of these agents has posed a remarkable reduction in the invasive burn wound infection. It also reduces chances of conversion of partial thickness to full thickness by infection which has led to a reduction in the amount of skin grafting being carried out. The use of topical antibacterial agents does not nullify daily inspection of the wound, debridement, care, cleaning as opposed to tulles. These agents have proved to be the most effective to control pseudomonas aerogenosa.

Silver nitrate 0.5%: Is present as acquous solution of 0.5 %

Advantages	Disadvantages
>Effective against pseudomonas	> solution is hypotonic which can lead to Marked decrease in serum electrolytes like Na+, k+, cl and decrease in absorption of water
>Clinical bacterial resistance is unknown	So monitor serum electrolytes 6-8 hrly
	>penetrates the eschar poorly because silver Salts are precipitated immediately they come into Contact with the minerals of the body
>Doesn't impair epithelialization	>precipitated silver chloride causes marked Staining of the skin, dressing, beddings etc
Allergies are rare	
There is significant reduction in the amount Of evaporation, water loss leading to a decrease In metabolic requirement.	

Technique of application:

Initial cleaning and debridement of loose tissue (non viable). Gauze dressings which have been saturate with silver nitrate are applied on the burnt area followed by bandage dressing. Saturate the dressing

with warm silver nitrate every 2hrs. The patient is then covered with a dry sheet or blanket to diminish water loss. The dressings are changed once to thrice daily depending on the degree of infection, drainage and the amount of necrotic material present in the wound.

1% silver Sulphadiazine

This is the newest topical agent in the control of bacterial proliferation.

Advantages:

No pain when being applied

Does not precipitate and penetration of eschar is better. It combines rapidly with DNA and RNA hence more effective.

No bacterial resistance.

Disadvantages:

Skin rash in some cases

Neutropenia in some cases

Technique of application:

Initial debridement and cleaning of the wound

Apply to burnt area with sterile gloved hand

The layer of cream should be 3-5 mm in thickness

Followed by a layer of gauze and then bandages.

Advantages of closed method:

Comfort

No external contamination of the wound

Decrease in the amount of evaporative water from the wound

Tulles promote drying of the wound

Absorbent dressing prevent accumulation of serous or purulent exudates in the wound.

Splinting is possible

Disadvantages of closed method

In addition to the disadvantages of the specific topical agents, the following are the disadvantages of closed method.

Tulles tend to adhere to the wound causing pain during change of dressing

Tulles, topical agents and gauze are expensive

OPEN METHOD

It is ideal when dealing with burns of the face, perineum, unilateral burn to an extremity or trunk, burns in children when dressed are difficulty to apply topical antibacterial agents. Aim of the method is the development of a dry surface that retards bacterial proliferation. This depends on the coolness, dryness and exposure to light. This factors tend to inhibit bacterial proliferation.

Procedure

The burnt area is cleaned and the patient left alone. Serum oozes onto the burnt area and dries up to form a dry cast called eschar. The eschar acts as a physiologic dressing for the wound and protects the underlying structures from contamination and promotes rapid healing. If breakages occur in the eschar, it should be treated by applying topical agents. Areas in which pus is noted should be debrided.

Advantages of open method:

Cuts down the amount of medical attention

Chances of interference by clinicians and nurses are reduced and chances of external contamination are reduced

Method is economical

Patient is spared the pain experienced when dressings are changed

No blood loss as occurs when dressings are being changed.

Disadvantages:

Body temperature is not controlled

Flies access the wound and contamination it to cause infection

CONTRAINDICATIONS TO OPEN METHOD

Circumferential burns of the whole trunk

Burns of the hand because tissues of the hands are loose and burns of the hand are often accompanied by massive edema. If the edema is not corrected then the edema fluid organizes into fibrous tissue with contracture of the digital joint capsule and stiffness of fingers.

Burns of the hands should be treated by:

Closed method

Elevation

Active exercises

All those are aimed at reducing edema and formation of contractures and full movement of the joints to prevent contracture and stiffness.

III) EXCISIONAL METHOD

It is better accomplished on small full thickness burn wounds.

Advantage:

Early rehabilitation of the patient. Areas of up to 15% of body surface can readily be excised so long as good supportive care is available. Donor sites provide partial thickness skin grafts (which contain epidermis and capillary dermis) the excision should involve area of 2 degree burns surrounding the obvious full thickness burns. The early danger of this method is secondary infection causing rejection of the grafts. To minimize this possibility excisional therapy should be instituted as soon as possible after resuscitating with fluids.

NB: Donor skin must not be used unless it has been confirmed that the source is HIV negative. Agood example in which this method is used is the dorsum of the hand. In this case early rehabilitation of the hand is essential to gain maximum. 2-3 days following burns the burnt areas of the hand can be excised and then covered with partial thickness skin graft. The hand is then splinted at a position os right angled flexion of the metacarpophalangeal joint and with the finger joint flexion. This kind of splinting serves two purposes:

Provides early motion of burnt hand

Gives excellent cosmetic result.

Excision and skin grafting may be associated with significant hemorrhage. Carefulbacteriological monitoring of the wound should be done before skin grafting especially a swab for c/s. If staphylococci pyogens are isolated the patient is given cloxacillin or erythromycin..

Tetanus prophylaxis:

It should be given to all burn wounds. If within the last 3yrs the patient has had TT give only one dose of 0.5 ml.

Nutrition:

Blood vsls of the GIT undergo vasoconstriction within the first 24hrs following burns. During this period absorption from the GIT is poor and therefore nutritional requirements should be given through the IV line for the first 24hrs after which the clinician switches to oral route

Patients with severe burns occasionally develop paralytic ileus. So for the first 12hrs oral intake should be restricted to 50ml of water per hr. There after the volume may be increased with half strength milk followed by a liquid diet. An N/G tube should be passed in severely burnt patients to:

Test for gastric ileus in the early stages. Much secretion aspiration is suggestive of ileus. It should be tested every hrly.

Provides a route for additional calories and protein in the later stage.

Burns of the face

The majority of facial burns heal spontaneously and should be treated by open method(exposed method). Daily application of 1% povidine iodine lotion.

Burns of the eyes and eyelids

The eye should be examined for corneal damage as soon as possible before edema of the eye lids makes it difficult. Burnt eyelids should be done skin grafting before significant ectropion has developed. Refer the patient to eye clinic as soon as possible.

Cold injury

Frost bite may be associated with hypothermia. If the body temperature falls below 32 degrees the patient should be kept warm and they are likely to have cardiac arrhythmias. Oxygen should be administered together with 5% dextrose IV. The areas should be compressed with turbid water of about 40 degrees celcius and then treat them as ordinary burns.

Radiation burns

Acute radiation produces damage to the skin and local tissues. The injuries resemble thermal burns. The difference being that acute radiation burn necrosis sets in more slowly and more deeply than the initial erythema. Surgery is contraindicated until the wound has passed into subacute stage which is characterized by:

Disappearance of erythema

Disappearance of edema

Sub acute and chronic burns should be treated by skin grafting. The timing of skin grafting is dictated by the appearance of the wound.

SUBSEQUENT CARE OF BURNT PATIENT TO ACHIEVE HEALING

Nutritional support

Patient with burns exceeding 30% require HPD and high caloric diet which should be given through NG tube in additional to a normal diet. Sugar and milk based products should be given by Sutherland method.

Adult= proteins (1gmxbwt in kg + gm x % burns) for 24hrs

Calories (20 kg calories x bwt x % burns) for 24hrs

Children = proteins and calories should be equal that which the child normally receives at his age. Make up for any deficiency caused by starvation for general anaesthesia. These additional requirements are reduced as healing progresses.

Immunology and antibiotic therapy

Septic complications are the major causes of death in patients with burns because these patients' immune reserves should be reinforced by the intermittent administration of fresh frozen plasma to provide antibiotics. The pt's Hb should be monitored and maintained by fresh blood transfusion.

In general patients with burns should be treated with fresh frozen blood accompanied with antibiotics if the organisms are present. Antibiotics should be used with caution because they may cause resistance. Burnt patients are also susceptible to viral and fungal infection. For severely burnt patients isolation and barrier nursing are important.

Physiotherapy

Regular breathing exercises are essential to all patients with significant burns. The patient should be encouraged to use their main muscle groups regularly.

Psychological support

Burnt patients may become severely depressed and require constant reassurance and positive encouragement to avoid them from becoming lethargic and anoxic.

Follow up and reconstructive surgery

Patients must be carefully encouraged to return to normal life. Deep areas of burns eventually give rise to severe and permanent scars whether grafted or not. The scars are called hypertrophic scars and partially troublesome in children. They are hard, dark, raised, irregular and pruritic. The process of maturation of these scars, i.e. softening, flattening and return to normal skin takes many months or years to come to a completion. During this period scar tissue contract especially those on flexion areas.

Complication of burns

They are divided into:

Early (immediate)

Intermediate

Late

Early complications

Fluid loss: Mainly plasma and especially electrolytes as the main components. This is mainly due to oozing of fluids from the burnt area. The greatest amount loss of fluid occurs in the first 18 hrs. In major burns more than 30% of patients develop gastric distention and paralytic ileus due to loss of electrolytes. For this reason the burnt patient should be given nothing by mouth because of the increased chance of vomiting and aspiration. As a routine, pass an Ng tube for several days until the gastric function has returned to normal. If the patient is alert, no vomiting, and no signs of abdominal distention, other fluids, calories and proteins can be given orally. If paralytic ileus develops after 5-7 days after burns suspect infection (sepsis).

Hypothermia: peripheral blood vessels constrict and blood is deviated to vital organs. This leads to reduction of body temperature. Hypothermia also comes as a result of skin destruction.

Acute renal failure: The patients who are likely to develop renal failure are; (i) Pts with electric burns (ii) Those with other forms of trauma e.g. crash injuries (iii) Pts in whom fluid resuscitation is delayed. These pts have a raise in bld urea and creatinine levels, low GFR, and lack of response to a bolus of fluid (sudden large amount of fluid).

Renal failure is prevented by an early restoration of blood volume by fluids and maintenance of fluid balance. Treatment for renal failure is like for any other type of renal failure. The clinician must ensure that no overload of fluid by strict maintenance input and output chart. Acidosis and al kalemia must be detected and treated appropriately .

d. Haemoglobinuria: It is caused by coagulation of RBC of the burnt area, haemolysis of RBC by toxins, release of myoglobin from burnt muscles.

e) Gastroduodenal erosion and ulceration (curling ulcers): The incidence is about 11% of patients but varies with the % of burns and the presence of sepsis especially in patients with burns less than 50%. Curling ulcers are acute and are thought to be due to stress especially excessive secretions of steroids. They develop within the first 48hrs. The lesions are primarily gastric in location. They may develop anywhere in the body and fundus of the stomach and they are multiple but occasionally single or multiple ulcers in the duodenum. The common clinical sign of the ulcer is the gastrointestinal hemorrhage with hematemesis and malaena with dark stools. Hematemesis occurs more frequently than malaena (x3 more frequently than malaena). About 1% of the pts will require blood transfusion. 10% of the ulcers go on to perforate. The first signs of perforation may be;

- Sudden abdominal pains

- Paralytic ileus

-Abdominal distention

Initial management of ulcers: It is conservative. The stomach should be decompressed with NG tube by aspiration. Check the aspirate for blood. Antacids or milk should be given. Blood transfusion depending on amount of loss.

Indication for operation in a patient with curling ulcers;

Perforation

Uncontrollable hemorrhage. Other curling ulcers should be treated conservatively. If surgery is necessary then the procedure of choice is antrectomy and vagotomy.

Curling ulcers can be prevented by prescribing drugs like cimitidine (tagamet) which are H2 receptor antagonists –Ranitidine (zantac).

A cute toxaemia: This is due to infection in the burnt area by bacteria. Bacteria produce toxins which are absorbed into the blood stream. The patient may go into toxic shock.

Local edema

Intermediate complications of burns

ANEMIA: It frequently follows major burns and is due to the following;

Coagulation of circulating blood in the burnt area .

Haemolysis of RBCs because of absorption of tissue breakdown products from the burnt area. Up to 10% of the RBCs mass may be lost due to haemolysis.

Direct destruction of the RBCs by the burning agent.

Infection may set in the burnt area and give rise to bone marrow depression.

Gastro- duodenal bleeding (curling's ulcers).

Infection: It may come from the following sources;

Patient himself

Medical staff

Patient's environment e.g. bacteria which are resistant to many antibiotics are common in the wards in which the patients are congested.

Conversion of superficial burns to deep burns: may be due to two major reasons;

- Infection
- Improper assessment by the clinician

Death of underlying structures: Increased blood vessels leads to severe hemorrhage leading to anemia and death of underlying structures.

Osteomyelitis due to exposure of bone.

Late complications of burns

During convalescence acute duodenal ulcers may occur in some % of patients and this sometimes becomes chronic (chronic DU)

Chronic .renal failure

Protein losing enteropathy causing hypoproteinaemia leading to edema causing delayed wound healing.

Contractures which follow scar tissue formation. Contractures occur in special sites i.e.

Lower eyelids to ectropion

In the chest wall and will interfere with respiratory movements

Over joints. They may be contracted in flexion or extension position. May be so severe , as to cause dislocation.

Hypertrophic scar formation: The burnt area heals with excessive scar tissue formation and the area rises above the level of the skin. Hypertrophic scars are common in young people. Scar tissues form in phases. Both the intensity and duration are of the active phase of scar formation and is increased more than the normal. There are 3 main stages through which a scar passes.

Stage I: 0-4 wks, the scar is fine, soft, not contracted nor strong.

Stage ii: 4-12 wks, the scar becomes red, hard, thick, and strong and tends to contract

Stage iii: 12- 40 wks, the scar gradually softens, becomes supple, white and tends to relax. Even in cases where severe hypertrophy occurs, scars do not become worse after 12 wks. The scar is thick, red and often itchy and this .persists for about 3-6 months. After this it usually regresses.

Compression treatment of hypertrophic scars:

Using elastic appliances to cause flattening and maturing of the scars. It can be in a form of;

Stockings

Gloves,

Armlets

Body pieces

Helmets for head / face/ neck etc.

Keloids are common in Asians and Africans. The common sites for keloids include;

Shaving areas of the face

Earlobes especially the pierced

Sternum

Back

Difference between keloids and hypertrophic scars:

Hypertrophic scars never get worse after 6 months but keloids continue to get worse even after one yr. Some keloids may progress for even 5-10yrs.

In a keloid the process of maturation and stabilization takes longer than hypertrophic scar.

Keloids are very difficult to treat because excision and resuturing is followed by recurrence. Previously superficial epithelial irradiation was done but the results haven't been certain

Better results are being obtained by shaving away of the excessive tissue but care shoul d be taken not to extend the excision into normal tissue at any point because it is thought that keloids can spread to normal tissue that was not affected by burns. After shaving away the tissues the area is resurfaced by a thick skin graft over this area. Some excellent results have been obtained by injection of steroids into the keloids.

Marjolin's ulcers: It follows burns of lower limbs. This burns typically heal with thin paper like scar formation when the area undergoes frequent trauma. Squamous cell carcinoma may result.

Deficient temperature regulation as a consequence of chronic scarring.

Hypo pigmentation. The scar remains pale in color.

Tracheal stenosis following respiratory tract burns.

Esophageal stricture following swallowing corrosive agents.

Syndectomy: Joining of two or more fingers occurring due to poor dressing of fingers.

Chondritis of the scarred cartilage.

SUMMARY OF BURNS MAGEMENT

First aid

Fluid resuscitation

Wound care

Rehabilitation.

PRE-MALIGNANT CONDITIONS OF THE SKIN

LEUKOPLAKIA: A white thickened patch occurring in the buccal membrane especially in the tongue can also occur on the lip or tongue. Ussually occurs in middle aged males.

Etiological factors

Smoking(esp. pipe)

Syphilis(tertially)

Sepsis

Sharp edge of teeth- irritation trauma

Ingestion of hot spirits

Idiopathic

Pathological process: It is slow and progressive. If left untreated about 20% of all leukoplakia patches undergo malignancy.

SENILE (SOLAR KERATOSIS): Occasionally occurs as multiple lesions on the faceand back of the hand in persons past middle age. The important predisposing factor is ultra violet rays . The lesion is 1 cm in diameter and hard dry scale. Squamous cell carcinoma may develop from senile keratosis.

RADIO DERMATITIS:

In the early stages – Erythema occurs and this progresses to pigmentation and disquamtion. If the dose is very high ulceration may develop.

In the late stages – Atrophy, irregular hyperpigmentation and telangiectasis and hair loss. Eventually squamous carcinoma may develop.

CHRONIC SCAR

For example marjolin scar following burns. A carcinoma that develops in the scar has the following characteristics;

Grows slowly because the scar is relatively avascular

Painless because the scar has no nerves

Secondary deposits don't occur in the regional lymph nodes because the lymphatic vessels have been destroyed.

BOWENS DISEASE

Is an intradermal pre-cancerous condition of the skin in which a brownish induration with a well defined edge appears on the skin. Microscopically large clear cells are seen sooner or .later ca develops.

Treatment – Wide excision

PAGETS DISEASE OF THE NIPPLE

A persistent emphysema like condition that usually starts in the nipple of a patient of above 50 years. It does not respond to treatment. The nipple is eroded and eventually disappears. As the disease progresses the areolar becomes involved and the erosion continues to spread peripherally for about 2 years until the site becomes manifest. It has been agreed that it a slowly growing duct carcinoma which infiltrates the epithelial covering of the nipple.

Microscopically: Infected area is characterized by presence of large vacuolated cells with small deeply staining nucleus in the epidermis. In the majority of cases an exhaustive research reveals malignant changes in the duct. Sooner or later a ca of the breast itself develops.

DDX

Eczema – But is an area of erythema, vesicles and oozing of fluid after rupture and responds to treatment.

Eczema	Pagets
* bilateral	Always unilateral
* occurs during lactation	occurs after menopause
* vesicles are present	no vesicles
*responds to treatment	doesn't respond to treatment

Treatment

Always mastectomy; Skin, breast, underlying pectoral muscles and all lymph tissue in the axill are removed.

The patient should not be subjected to radio therapy alone because it is radio resistant. Radio therapy is important post operatively.

MALIGNANT CONDITIONS OF THE SKIN

BASAL CELL CARCINOMA (RODENT ULCER)

Common in whites than blacks

A common tumor of low grade malignancy

Exposure to the sun is a predisposing factor and therefore the rodent ulcer is common in the tropics

Also occurs in the skin which has been exposed to acids like arsenic

Common in the middle and late age groups

Site- 99% found on the face usually above a line drawn from the lobe of the ear to the corner of the mouth. The commonest site is the inner canters of the eye. Although referred to as a rodent the lesions are non- ulcerated with a dark translucent color as if containing water. There is a network of red blood vessels on the surface of the ulcer.

TYPES

Nodular

Cystic

Ulcerative

A unusual type called “field fire” basal cell carcinoma in which the ulcerated type has a typical appearance. A raised rolled edge like a car tyre with a central ulceration. Temporal healing often takes place but is followed by further lesion with serous discharge with bleeding.

The patient gives a history of a spot which never heals. Although they are slow growing when they ulcerate they involve deeper structures e.g. muscles, cartilage and bone and develop severe disfiguration (hence rodent ulcer). Rarely the basal cell carcinoma changes to squamous cell carcinoma.

MICROSCOPICALLY

Masses of darkly staining cells with a characteristic arrangement in which there is an outer layer of columnar cells surrounding a central mass of polyhedral cells. Cystic spaces are seen.

NB: Dissemination by lymphatics or blood stream does not occur in basal cell carcinoma.

TREATMENT

Surgery or radio therapy is necessary. Excision allows the whole lesion to be examined. It is essential to excise a marginal healthy tissue around the ulcer and do histopathology of the lesion. A skin graft is required to cover the lesion.

SQUAMOUS CELL CARCINOMA

Less common than a rodent ulcer

More malignant and grows more rapidly.

Can occur on its own (independently) " De Novo" in the skin of the face of elderly people but more often occurs in:-

Pre existing skin conditions or

As a result of past irradiation. Can arise from, long standing venous ulcers, chronic lupus vulgaris (TB of the skin), prolonged irritation of the skin by various chemical e.g. tar, dyes, soot etc.

Characteristics of typical squamous cell carcinoma

Outline is irregular

Edges are raised and everted

Base is indurated and sooner or later becomes attached to the deeper structures

A black stained discharge occurs and increases in amount when infection sets in.

The regional lymph nodes may be involved and the deposits may undergo mucoid degeneration to which infection may be added.

Treatment

As soon as the diagnosis is confirmed by biopsy wide excision with skin grafting

Radio therapy which will depend on; * condition of patient, * size of the tumor, * involvement of underlying structures, * and availability of RT facilities.

If lymph nodes are enlarged and mobile block excision should be done i.e. dissection of all lymph nodes. Make sure the enlargement is not secondary infection and they should be fixed. Give antibiotics before excision which helps to confirm that the enlargement is due to tumor. Fixed enlarged lymph nodes are not viable but some regression may be retained by subjecting the lymphnodes to RT.

MALIGNANT MELANOMA

It has been agreed that it is a melanocarcinoma and may arise in two ways:-

From preexisting benign melanoma (pigment naevi)

Can arise De novo.

Etiology

Not known

It is common in light skinned people exposed to hot climates (ultra violet rays)

Clinical recognition

Almost unknown before puberty. It should be suspected after puberty under the following circumstances:-

When a previously existing mole (naevi) begins to enlarge, itches, weeps, scabs or becomes deeply pigmented.

When a pigmented lesion appears in an adult and grows progressively

When a rapidly growing ulcerated skin tumor appears which looks as though it may be malignant. Some malignant melanomas are amelanotic (without melanin) and are called amelatic melanomas.

Site

The commonest site is in the lower leg. In males, it occurs at the foot or back of the trunk.

In black Africans it is common on the sole of the foot.

For reasons not known malignant melanomas don't originate from black parts of the body e.g. eye, meninges, or mucocutaneous junctions e.g. the anus.

DDX

Histiocytoma

Pigmented basal cell carcinoma

Basal cell papilloma

Carvenous haemangioma

Spread

By local extension

By lymphatics

By blood stream

Tumor cell may reach lymph nodes by embolism but spread by lymphatic herniation. It can also occur producing local satellite nodules and the in transit deposits (i.e. between primary growth and the regional lymph nodes). Secondary lymphedema may occur (distant metastasis to lungs, liver, brain, bones, skin, breast and intestine.

Staging and prognosis

Stage I- primary tumor only

Stage II enlargement of lymph nodes or satellite deposits or in transit no des + stage I

Stage III widely disseminated disease

Approximately 70% of patients in stage I survive for 5 years

Approximately 25% patients in stage II survive for 5 years

Most patients in stage III die within 1 year.

The outlook is better in females than males. Pregnancy makes pigmentation moles darker and sometimes longer but does not change the course.

Treatment

Stage I surgical excision because malignant melanoma is non sensitive to treatment. How wide to extend depends on the thickness of the tumor. The surgical defect of the excision is non extensive. If the extent of the excision and laxity of the tissues doesn't allow suturing then skin grafting is done.

NB: Moles should never be cauterized or curreted because this destroys vital evidence if the lesion is malignant the disease may be disseminated.

Management of lymph nodes;

When the regional lymph nodes are involved clinically they must be dissected . When clinically not involved decision must be made whether to dissect or watch and wait until they become enlarged for a thin lesion the rise of lymph spread is slight. For thicker lesions prophylactic dissection of lymphnodes is radiant especially for patients who cannot be regularly be followed up . When regular follow up is possible dissection of lymph nodes should not be done until they clinically involved.

Other methods of management

Cytotoxic agents are of little value especially when the lesion is disseminated but they do not help in the control of locally advanced disease in the limbs if administered by a specialized technique i.e. isolated perfusion where high local concentration without generalized side effects.

Immunotherapy has been tried but not effective.

Malignant melanoma of colloid

This usually presents with blurring of vision treated by enucleation of the eye because they eye has no lymphatic drainage, it spreads by blood to the visceral deposits which can be enormous especially in the liver. The metastasis may not appear clinically for many years after removal of the eye.

BOIL (FURUNCLE):

Definition: An acute staphylococcal infection of a hair follicle with perifolliculitis which usually proceeds to suppuration and central necrosis.

A painful and indurated swelling appears and this gradually extends. After 2-3 days the center of the boil softens and a small slough is discharged with a bead of pus and in the large majority of pts the condition subsides. A boil whch subsides without suppuration is called “a blind boil”.

Boils are generally common in the back of the neck because of presence of hair follicles. A stye is a special boil due to an infection of an eye lash also called hordelum. Infection of a peri anal hair with suppuration is likely to result in a sinus. Infection of hair follicle around the ear meatus causes furunculosis which is very painful because the skin attaches the underlying cartilage and also considerable tension.

Complications:

Cellulitis especially in debilitated pts.

Infection of lymph nodes draining the part.

Secondary boils due to infection of neighboring hair follicles.

Treatment:

Improve the general health of the pt because boils are frequently associated with overwork, worry, debility, examinations. Incisions are not necessary (contraindicated).

A touch of iodine or liquid phenol on a skin pastule hastens necrosis of the overlying skin so that the pus can escape.

If softening occurs around the hair follicle especially on eye lash, removal of the hair allows ready escape of pus.

Determine the sensitivity of the organism and use appropriate antibiotics

Washing the surrounding skin BD with a suitable disinfectant to discourage development of secondary boils.

A paste consisting anhydrous magnesium sulphate 24 parts + glycerine 2 parts is valuable in the treatment of boils because it has osmoticeffects.

CARBUNCLE

Definition: an infective gangrene of the subcutaneous tissue due to staphylococcal infection.

Uncommon below the age of 40yrs

Males are the usual sufferers

DM may be present

Sites

Nape of the neck where the skin is coarse and rough and ill nourished.

Clinical features:

Pain and stiffness of the site

Sc tissue is tender and indurated

Overlying skin is red hick pus sloughs

Extension if in treatment and after 3 days areas of softening occur, the skin giving way and usually there is one central large slough surrounded by smaller areas of necrosis.

Infection sometimes extends widely and fresh openings appear on the surface and they then coalesce with those openings that were previously formed. Scarbuncle of the cheek or upper lip is dangerous because carvenous sinus thrombosis may form.

Treatment:

General treatment and identification of organisms is similar to that of boils

Many carbuncles can be aborted by use of antibiotics

LUPUS VULGARIS (Tb of the skin)

Occurs at the age of 10-25 yrs

Common on the face

One or more nodules appear with congestion of the surrounding skin

Nodule is usually painless

When pressed with a glass slide the nodule appears like apple jelly.

Extension occurs very slowly but suppuration occurs later.

Resulting ulcer tends to heal from the area of origin and migrates. The mucus membrane of the nose and mouth are attached either primarily or extension from the face. Edema occurs if fibrosis caused by lupus obstructs normal lymphatic drainage.

Infection in the nasal cavity may be followed by necrosis of the underlying cartilage.

Epithelioma is prone to occur in lupus scar.

Treatment

General health of the pt

Anti-Tb drugs

Excision if healing is slow.

CYSTS

Definition: A swelling consisting of a collection of fluid in a sac lined by epithelium.

Broad categories:

True cysts

False cysts

True cysts:

They are lined by epithelium or endothelium. If infection sets in then the lining may be composed of granulation tissue and the fluid, usually serous or mucoid, and varies from being brown stained as a result of altered bloodto almost colourless. In epidermoid, dermoid and brachial cysts the contents are like porridge as a result of secretion of desquamated cell (also toothpaste like). Cholesterolcysts are often found in them.

False cysts:

Certain collection of fluids which are not necessary true cysts. They are usually exudation cysts.

Examples include

Pseudocyst of the pancreas- An encysted collection of fluid in a lesser part of the pancreas.

Tuberculous peritonitis- Fluid may be walled off in a cystic form by adherent coils of intestine.

In the center of a tumor due to hemorrhage or due to colliquative necrosis can also happen in the brain as a result of ischaemia ending up with apoplectic cyst.

Classification of cysts.

Congenital

Acquired

Parasitic

Congenital cysts:

They are due to dermoid cells being buried along the lines of closure of embryonic clefts and sinuses by skin fusion, hence the term sequestration dermoids. The cyst is lined by epidermis and contains paste like desquamated material. An example is the brachial cyst.

Brachial cyst:

It usually arises from the remnants (rudimentary remnants). The 2^{nd}, 3^{rd} and 4^{th} clefts may persist. Brachial cyst is usually lined by squamous epithelium and contains clear or paste like fluid. It appears in young adults but occasionally in later stages of life. It protrudes from beneath the anterior border of the upper 1/3 of the sternomastoid, and appears as a fluctuant swelling and may transilluminate but not necessarily a brachial cyst whether positive or negative. If infection has occurred it is difficult to differentiate a brachial cyst from a tuberculous abscess. If the aspirated fluid contains cholesterol crystals it is a brachial cyst. A rare variety of brachial cyst may be found lying near the pharynx and is lined by columnar epithelium which may contain mucus.

Tubulo-dermoid cyst (Tubulo- embryonic)

Occurs in a track of an ectodermal tube in development. Example is the thyroglossal cyst which arises from the thyroglossal duct during growth. The thyroglossal cyst – Due to persistence of the thyroglossal duct which extends from the foramen caecum to the base of the tongue to the thyroid gland. After descending the duct should close but may persist. The thyroglossal cyst may persist in any part of the tract (duct).

Sites for thyroglossalcyst

Area just below the hyoid bone

Region of the thyroid cartilage

Area above the hyoid bone

Clinical features:

As a rule a fluctuant swelling in the mid line of the anterior part of the neck. The only exception is when it occurs at the region of the thyroid cartilage where the thyroglossal tract is pushed to the left.

The swelling moves upwards when patient protrudes the tongue or swallows because the thyroglossal duct is attached to the foramen caecum. It moves down when the patient returns the tongue to the mouth. Even the one at the sides behaves the same.

Treatment

Surgical excision, including the entire tract up to the base of the tongue. Infection is inevitable because the wall of the cyst contains nodules of lymphatic tissue which communicates with the lymph nodes of the neck through lymphatics.

NB: Infected cysts may be mistaken for an abscess and incised. When this happens a thyroglossal fistula arises.

Cysts of embryonic remnants:

They arise from embryonic tubules and ducts which normally disappear or present as remnants. Example : Urachus may persist forming a cyst. The urachus connects the umbilicus to the urinary bladder. If there is complete failure urine escapes through the umbilicus.If lower end remains patent then a diverticulum of the bladder forms. If the middle remains patent then urachial cyst forms leading to suppuration then forming an abscess. The treatment is surgical excision. Postnatally it is supposed to become a fibrous cord from the apex of the bladder to the umbilicus and is called median umbilical ligament.

ACQUIRED CYSTS:

Retention cyst -- This is due to accumulation of secretions of a gland forming obstruction of a duct.Example is a sebasceous cyst (wen) which follows obstruction of the mouth of a sebasceous duct. Patologically it is classified as a dermoid cyst. Common sites are the face and the scalp but can occur anywhere where there are sebasceous glands except the palm and soles.

Clinical presentation:

It appears as a hemispherical swelling, firm or elastic in consistence with no definite edge. It is also adherent to the skin especially if was previously inflamed or subjected to pressure. The punctum of the obstructed can occasionally be seen on the cyst and sebasceous material can be expressed from the cyst. An uncomplicated cyst contains yellowish material composed of fats and epithelial cells. It has putty like consistence and can therefore be indented by a finger tip. Rarely there is a minute worm called dermodex folliculorum seen in the cyst.

Treatment:

Incision - avulsion under local anesthesia. An incision is made through the skin and cyst. The contents are squeezed out and the cyst wall is torn away (avulsion).

Dissection is necessary for cysts which have previously been inflamed. A skin incision is made, wall identified and cyst dissected and removed intact. Unless the wall is completely removed recurrency is possible

Distension cyst:

Occurs in the thyroid as a result of dilatation of acini or in ovary from a graafian follicle.An example is the;

Cystic Hygroma:

At about the 6th wk of embryonic life there are primitive lymph sacs develop in the mesoblast (middle layer of cells)

The main pair is situated at the neck between the jugular and the subclavian vein. They are known as jugular lymph sacs. Sequestration of a portion of jugular lymph sacs from the lymph system leads to formation of cystic hygroma.

Manifestation:

Occurs during early childhood

Occasionally present at birth when it is so large to cause obstructed labor.

Swelling occupies lower 1/3 of the neck and enlarges upwards towards the ear.

Often it is the triangle of the neck involved

Due to intercommunication of this compartment a cystic hygroma is softly cystic and partially compressible.

Increases in size when child cries or coughs

The characteristic feature which differentiates it from the others is that it is brilliantly transluscent

Other sites include; Neck,axilla.

Clinical course of cystic hygroma:

It is uncertain in infancy and is not possible to determine what will happen (prognosis). Sometimes the growth is rapid and occasionally causes respiratory difficult and this demands immediate aspiration of the contents of the cyst. It may also be necessary to do a tracheostomy . At other times there may be infection of the nasopharynx and the cyst may swell or regress on its own.

Pathology:

The swelling consists of an aggregation of cysts which look like a mass of bubbles of soap. The larger cysts are nearer the surface while the smaller lie deeply and tend to infiltrate muscle planes.Each cyst is filled with clear lymph and is lined with a single layer of endothelium.

Treatment:

Excision of cysts at an early age

Often advisable to give injections of sclerosing solutions or even boiling water into the cysts at weekly intervals. The purpose is to reduce the swelling in size and the cyst wall becomes more fibrous and this facilitates easy dissection.

Exudation cysts1

Occurs when fluid exudes into an anatomical space which is already line by endothelial or epithelium. Examples are:

Hydrocele of tunica vaginalis

Bursa- a fibrous sac with synovial membrane filled with synovial fluid.

When a collection of exudates becomes encysted (they are false cysts)

Cystic tumors

Examples include dermoid cysts of the ovary or cystic teratomaj

Implantation dermoids

They arise from squamous epithelium which has been driven beneath the skin by a penetrating wound e.g. in knife grinders, black smith where heavy metal sparks are likely to fly off and cause injury and a small portion of skin is eventually driven into the puncture developing a cyst. They are therefore traumatic and are commonly seen in fingers, palms, legs, cornea.

Classically found in fingers of women who sow a lot. The contents are desquamated cell debris which may undergo mucoid degeneration

Treatment:

The entire cyst should be excised and this gives permanent cure.

Traumatic cysts:

A hematoma may resolve into a cyst. This sometime happens to the muscle masses of the loin and the antero-lateral aspect of the thigh or on the skin. This are located between muscle planes or subcutaneous spaces . They contain brown colored fluid containing cholesterolcrystals.They beco me lined by endotheliumand calcium crystals may be laid on them. Aspiration is only of a temporally value. Only completeexcision gives complete cure.

Degeneration cysts

PARASITIC CYSTS

They are encysted forms of the life cycle of various worms e.g. Hydatid cyst of ;

tinea echinococcus

Trichiniasis of trichuria spiralis affecting muscles

Cystercosis taenia solium of the pig. Rarely affects man but if they occur in man, they calcify and cause effects depending on where they occur especially in the brain. Eisonophillia is usually present. Only those cysts that are causing symptoms should be excised. Hydatid cyst is the most important of the three.

Effects of a cyst.

The effects depend on the size and site of the cyst;

May compress ducts and blood vessels e.g. renal cyst or H. cyst may obstruct a common bile duct.

An ovarian cyst may compress the pelvic veins leading to varicose veins.

The sheer size of an ovarian cyst may increase intra abdominal tension so much so that the patient may come to hospital with symptoms of hiatus hernia.

Complications of cysts

Infection: The cysts become tense, painful and adheres to the surrounding tissue. An abscess may form and discharge onto the surface and result in an ulcer or sinus. Healing does not occur unless the whole lining has been excised.

Hemorrhage: Sudden hemorrhage as may occur in a thyroid cyst. This causes a painful increase in size. There may be difficult in breathing.

Torsion: In cysts attached to neighboring structures by vascular stalk (pedicle). Ovarian cysts sometimes presents this way as acute abdominal emergencies. The cyst turn purple due to cut of blood supply.

Calcification: This follows hemorrhage or infection and as a result of reaction to a parasite e.g. hydatid cyst.

HAEMANGIOMA

Definition: A developmental abnormality of the blood vessels

Not a true tumor

It is an example of a hermatoma

Most common in the skin or subcutaneous tissue but can occur at any tissue of the body.

A haemangioma can be capillary, venous (carvenous) or arterial (plexiform)

Capillary haemangioma

There are several varieties;

Salmon patch: - it is present at birth often at the forehead in the midline and over the occip.. It disappears on its own at the age of 1 yr.

Pot wine stain: presents at birth but changes very little throughout life. Only the color may change a little and it may become nodular in some areas. Treatment only for cosmetic reasons. The texture of the skin is normal. If it occurs in ladies it can be disguised with application of cosmetics. In a boy treatment by excision and skin grafting may be considered. Radiotherapy and any other of destructive therapy is of no value.

Strawberry angioma: Acommon lesion and has a typical history. The baby is normal at birth but after 1-3 wks is noted to have a red mark which increases rapidly for some weeks or even up to three months until the typical straw berry like swelling is reached. Clinically the sign of emptying may be demonstrable. The lesion is composed of immature vaso formative tissue. The subcutaneous tissue as well as the skin are often involved and in severe cases muscles may also be affected. Sub mucosal strawberry angiomas can cause hemorrhage which can be very alarming in some cases. From the age of 3 months to 1 yr, the naevus grows in the child then ceases to grow. Eventually the color fades and then flattening occurs such that at the age of 7-8 yrs involution is complete. Final result is better. Better allow them heal on their own. Radiotherapy is risk of disturbing of growth.

Carvenous:

Are relatively uncommon

Present at birth

Consist of multiple channels of varying caliber

They show no tendency of involution and may become larger and more troublesome later.

Sometimes a whole limb or some parts of adjacent to the trunk are affected.

Occasionally associated with lipoma and in this case is called a naevo-lipoma.

In some cases arterio-venous communications are present.

The skin overlying the naevus may be atrophic and besides being a danger of severe hemorrhage from trauma, the pt may suffer from septicaemia if organisms gain entry.

Antibiotics are urgently indicated

The treatment is generally conservative. Repeated injections of hot water and other sclerosing agents may help but generally not employed.

Arterial (flexiform) hemangioma:

A type of arterio-venous fistula. Presents as a swelling of arteries and arterialized veins and often referred to as cirsoid aneurysm, which is rare and difficult to treat. Capillary may occur in the skinand beneath this abnormal arteries communicate directly with ascended vein. Most commonly the superficial temporal artery and its branches are affected. Cirsoid aneurysm tends to enlarge slowly and hemorrhage occurs if ulceration occurs.

Spider neavus:

A type of capillary hemangioma. It may occur in association with cirrhosis of the liver, especially if located on the skin over the manubrium sterni. May also occur innocently (independently). It shows signs of emptying. Spider naevi usually occur on;

Face

Neck

Shoulder

Upper arm

They physiologically proof to be an overgrowth end artery with branching arterioles.

LYMPHANGIOMA

A congenital localized clusters of dilated lymph sacs in the skin and subcutaneous tissue. The lymph sacs are never connected to the normal lymphatics. The lymph sacs are connected togeth er by a connective tissue. They can occur in association with hemangiomas and are called haemolymphangiomas.

Etiology: Obscure. Likely cluster of lymph sacs that fail to form because they are embryological.

Types:

Capillary lymphangiomas: Are composed of capillary lie lesions in the skins. They are brownish papules or wart like. On examination, tense small vesicles which are lymphatic naevi can be seen. They occur especially on the skin but may be found in internal organs.

Carvenous (cystic) lymphangiomas: Already discussed (refer)

GANGLION:

Definition: A localized tense cystic swelling containing clear gelatinous fluid. It is usually painless. It often communicates with a tendon sheath or capsule of a joint.

Aetiology: The exact origin is not known but the following factors are thought to be etiological;

May arise as a mucoid degeneration of connective tissue. It may arise as a consequence of leakage of synovial fluid through the capsule of a joint.

Trauma.

Site: Simple ganglion; dorsum of the foot and dorsum of hand.

Occasionally minute ganglia can be found on the flexor aspects of the fingers and are extremely painful and tender.

Treatment:

Frequently disappear spontaneously

Removal by surgical excision.

When exploring a ganglion on the flexor aspect of the wrist care must be taken because the radial artery is closely related to the ganglion.

LIPOMA:

Definition: A cluster of fat cells which have become overactive and so distended with fat that they become palpable. It is the commonest of non-connective tissue tumors.

Pathology: Consists of adult fat cells with minimum stromal tissue. It has a capsule from which fibrous strands extend into the growth dividing it into lobes. They undergo necrosis and calcification but never become malignant.

Classification: May be single or multiple. Single lipomas are similar to multiple lipomas in all aspects.

Age incidence: Affects all ages but generally rare in children.

Sites: The commonest is the subcutaneous plane. Lipomas are commonest in the subcutaneous plane of the shoulder, Neck, Buttocks but may occur anywhere in the body where there is fat.

Clinically;

Soft circumscribed, lobulated swelling

Swelling may sometimes be penduculated

The swelling is pseudocystic and pseudoflactuant and freely movable on deep palpation.

Fibrous strands pass from the capsule to the skin. Because of this attachment at more than one site and there is dimpling (in drawing) especially when the tumor is moved under the skin.

NB: A lipoma can be distinguished from a true cystic swelling clinically by a feel of the solid edge as the examiner tries to slip the edge under the fingers.

DDX

Sebasceous cyst but it is attached to the skin at only one point.

Treatment:

Intracapsular removal by making an incision in the capsule and shelling tumor from the capsule (enucleation).

PREMALIGNANT CONDITIONS OF THE SKIN

Bowen's disease:

It is an intradermal pre-cancerous condition of the skin in which a brownish induration with a well defined edge appearing on the skin.

Microscopically:

Large clear cell are seen sooner or later.

Treatment: wide excision.

paget disease of the nipple:emphysema

a persistant like condition that usually starts in the nipple of women over 50yrs. Does not respond to treatment. The nipple is eroded slowly and eventually disappears. As the disease progresses the areolar becomes involved and the erosion continues to spread peripherally for about 2 yrs and at this time a carcinoma on this site becomes manifest. (it has been agreed that it is a slowly growing duct carcinoma which infiltrates the epithelial covering of the nipple).

Microscopically:

The affected area is characterized by presence of large vacuolated cells with small deeply staining nucleus in the epidermis. In the majority of the cases, an exhaustive research has revealed malignant changes in the ducts. Soon or later a cancer of the breast itself develops.

DDX:

eczema. But it is an area of erythema, vesicles, and oozing of fluid after rupture and it responds to treatment.

Eczema	pagets
bilateral	always unilateral
occurs during lactation	occurs after menopause
vesicles are present	no vesicles
responds to treatment	doesn't

Treatment:

always radical mastectomy- the skin, breast, underlying pectoral muscles and all lymphatic tissues in the axilla. The pt should not be subjected to radiotherapy alone because it is radioresistant. RT is important post operatively.

Leukoplakia:

A white thickened patch occurring in the buccal membrane and especially on the tongue. Can also occur on the lips or genitalia. Usually occurs in the middle aged males.

Etiological factors:

Pipe smoking

Syphilis

Tertially Sepsis

Sepsis

Sharp edge of tooth causing irritation trauma

Hot spirit ingestion

Hot spices

Susceptibility(idiopathic)

Pathological process:

A slowly progressing hypernecrosis which if left untreated, 20% of all leukoplackic patches undergo malignancy.

senile (solar) keratosis:

it occasionally occurs as multiple lesions on the face and back of the hand in persons past middle age. The important predisposing factor is ultra violet rays. The lesion is 1cm in diameter and hard dry scale. Squamous cell carcinoma may develop from senile keratosis.

Radio dermatitis:

In the early stage, erythema occurs and this progresses to pigmentation and desquamation. If the dose is very high ulceration may occur.

In the late stages, there is atrophy, irregular hyper pigmentation and telangiectasis and hair loss. Eventually squamous cell carcinoma develops.

Chronic scars(like marjolins scars following burns):

A carcinoma that develops in the scar has the following characteristics:

Grows slowly because the scar is relatively avascular

Painless because scar tissue has no nerves

Secondary deposits don't occur in regional lymph nodes because the lymphatic vessels have been destroyed.

If the ulcer invades normal tissue surrounding the scar it extends at normal rate and thus lymph nodes are liable to be involved.

MALIGNANT CONDITIONS OF THE SKIN

Basal cell carcinoma:

It is also known as a rodent ulcer.

It is common in whites than blacks

It is a common tumor of low malignancy

Exposure to sun light is a predisposing factor and therefore the rodent ulcer is common in the tropics

Also occurs in the skin that has been exposed to acids like arsenic acid.

Age:

Middle or late age.

Sites:

99% is found on the face usually above a line drawn from the lobe of the ear to the corner of the mouth. The commonest site is the inner canthers of the eye. Although referred to as a rodent ulcer, the lesions are non-ulcerated with a darkly transluscent color as if containing water. A network of red blood vessels on the surface.

Types:

Nodular

Cystic

Ulcerative

Unusual type called " field fire" basal cell carcinoma in which the ulcerated type has a typical appearance. It has a raised rolled edge like a car tyre with a central ulceration. Temporally healing often takes place but is followed by a further lesion with serous discharge with bleeding. The pt gives a history of a spot which never heals.

Although they are slow growing, they ulcerate they involve deeper tissues e.g. muscles, cartilage, and bone develop severe disfiguration(hence a rodent ulcer). Dissemination by lymphatics or blood doesn't occur in basal cell carcinoma. Rarely the basal cell carcinoma changes to basal cell carcinoma.

Microscopically:

Masses of darkly staining cells with a characteristic arrangement in which there is an outer layer of columnar cells surrounding a central mass of polyhedral cells. Cystic spaces are seen.

Treatment:

Surgery or Rt is necessary. Excision allows the whole lesions to be examined. It is essential to excise a marginal tissue around and underneath the lesion. Skin graft is required to cover the lesion.

Squamous cell carcinoma:

It is less common than rodent ulcer.

It is more malignant and grows more rapidly

It can occur on its own(independently) "de novo" in the skin of the face of elderly people but more often occurs in a pre-existing skin condition or

As a result of past irradiation.

It can also arise from;

Long standing ulcers

Chronic Lupus vulgaris

Prolonged irritation of the skin by various chemicals e.g. tar, dyes, soot.

Characteristic of a typical squamous cell carcinoma:

Outline is irregular

Edges are raised and everted

Base is indurated and soon or later becomes attached to the deeper structures

A black stained discharge occurs and increases in amount when there is secondary infection.

Regional lymph nodes may be involved and the deposits may undergo mucoid degeneration to which infection may be added.

Treatment

As soon as the diagnosis is confirmed by a biopsy, wide excision with skin grafting.

Radio therapy depending on;

Condition of the patient

Size of tumor

Involvement of underlying structures

Availability of radiotherapy facilities

If lymph nodes are enlarged and mobile, excision should be done i.e. dissection of all lymph nodes. Make sure that the enlargement is not due to secondary infection and they should not be fixed. Gi ve antibiotics before you excise and this helps to confirm that the enlargement is due to tumor. Fixed enlarged lymph nodes are not removable but some regression may be retained by subjecting the lymph nodes radiotherapy.

Malignant melanoma:

It has been agreed that it is a melanocarcinoma. It may arise in two ways;

From a pre-existing benign melanoma(pigmented naevi)

Can arise de novo

Etiology:

Not known

Common in light skinned exposed to hot climates (ultra violet rays) of the sun.

Clinical recognition:

Almost unknown before puberty

Should be suspected after puberty under the following circumstances;

When a previously existing benign mole (naevi) begins to enlarge, itches, weeps, scabs, or bleeds or becomes deeply pigmented

When a pigmented lesion appears in an adult and grows progressively

When a rapidly growing fleshy ulcerated skin tumor appears as though it may be malignant. Some malignant melanomas are aemlanolitic (without melanin) and are called amelanotic melanomas

Site:

Commonest site in women is the lower leg

In males the foot or back of the trunk

In black Africans, sole of the foot

For reasons not known, malignant melanomas don't originate from black parts of the body e.g. eyes, meninges, or mucocutaneous junction like the anus.

DDX:

Histiocytoma

Pigmented basal cell carcinoma

Basal cell papilloma (sebhoreicwart)

Carvenous hemangioma.

Spread

Local extension

Lymphatics

Blood stream

Tumor cells may reach lymph nodes by embolism but spread by lymphatic herniation, can slso occur producing local satellite nodules and the in-transit (i.e. between primary growth and the regional lymph

nodes). Secondary lymph edema may occur (this shows distant metastasis). Metastasis is to the lungs, liver brain, bones, skin, breast, small intestine and heart.

Secondary deposits are typically black but sometimes they contain little or no melanin even when the growth is deeply pigmented.

Staging and prognosis:

Stage I – Primary tumor only

Stage II – enlargement of lymph nodes or satellite deposits or in-transit nodes + stage I.

Stage III – widely disseminated disease

Approximately 70% of pts in stage I survive for 5yrs 25 yrs for stage II . Most pts in stage III die within the 1st. The outlook is better in females than in males. Pregnancy makes pigmented moles darke r and sometimes larger but does not affect their course.

Treatment:

Stage I- Surgical excision because malignant melanoma is non sensitive to radiotherapy. How wide to excise depends on the thickness of the tumor. The surgical defect is closed by a primary suture if the excision is non extensive. If the extent of excision and laxity of tissues don't allow primary suture then skin grafting is done.

NB: moles should never be cauterized or curratted because this destroys vital evidence and if the lesion is malignant the disease may be disseminated .

Management of lymph nodes:

When the regional lymph nodes are involved clinically they must be dissected. Wh en clinically not involved, decision must be made whether to dissect then watch and wait until they become enlarged. For a primary lesion (thin), the risk of lymph spread is so slight. For thicker lesions prophylactic dissection of lymph nodes is indicated and especially for pts who cannot be regularly followed up. When regular follow up is possible, dissection of lymph nodes should not be done until they are clinically involved.

Other methods of management:

Cytotoxic agents are of little value especially when the lesion is disseminated, but they do help in the control of locally advanced disease in the limbs if administered by specialized technique i.e. isolated perfusion. When high local concentrations without generalized side effects.

Immunotherapy has been tried but not effective.

Malignant melanoma of cholloid:

This usually presents with blurring of vision. It is treated by enucleation of the eye. Because the eye has no lymphatic drainage, it spreads by blood to the visceral deposits which can be enormous especially in the liver. The metastasis may not appear clinically for many yrs after removal of the eye.

CIRCUMCISION

Definition: Excision of a circular portion of the prepuce.

It is usually preferred on young boys to allow the prepuce to be drawn back over the glans penis to facilitate urination and cleaning the penis.

Indications:

In infants and young boys

Request by parents (religious and personal)

Recurrent balanitis with inability to retract the prepuce

Very long prepuce (rarely)

Except for the ritual operation, most circumcisions are unnecessary. It is normal for the prepuce to be long, adherent to the glans within, for these parts become satisfactorily separated and the prepuce mobile in the first few yrs of life.

Recurrent balanophosthitis and phimosis often follow attempts by the parents forcibly to retract the prepuce.

In adults:

Inability to retract for intercourse

A tight frenum

Ballanits

Prior to radiotherapy for carcinoma

A posterior slit may suffice especially if an emergency arises e.g. paraphimosis

Technique in an adult:

Applying a clamp or bone forceps across the redundant prepuce distal to the glans with blind division of the foreskin can no longer be condoned (forgiven). This can lead to partial or total amputation of the glans. It is better to perform a proper circumcision under direct vision as in an adult .

A new device (plastibell (holster)) is used. The ring separates spontaneously between 5 and 8 days post operatively. The device causes excessive oedema i.e too tight, it can be removed easily by fracturing the ring.

The foreskin is freed and retracted

After the plastibell device has been slipped in place over the glans penis, the foreskin is ligated over the groove of the plastibell, and redundant foreskin is cut away.

In adults and adolescents:

Preferred method:

The prepuce is retracted until it's tense orifice is apparent or until the tip of the glans penis comes to view.

On the edge of the prepuce are placed three haemostats , one in the midline ventrally and two either side of the midline dorsally.

The prepuce is then slit up in the midline dorsally to within 1.25 cm of the corona. The under surface of the prepuce having been completely separated from the glans penis and corona, the layers of each flap are excised , keeping 1.25 cm distal to the corona.

The superficial layer is retracted and bleeding points are secured and ligated.

The inner layer of the prepuce having been trimmed to 3mm from the corona, the two cut edges are approximated accurately with fine interrupted catgut stitches.

The cut edge at the immediate vicinity of the frenum can be drawn together neatly by a mattress suture, the four in one frenal stitch.

GANGRENE

Gangrene is death with putrefaction of macroscopic portion of tissue. It is commonly seen affecting the distal part of a limb, the appendix, a loop of small intestine and sometimes organs like the gall bladder, the pancreas, testes and the appendix.

VARIETIE OF GANGRENE ACCORDING TO CAUSE

Secondary to arterial obstruction from disease, for example;

Thrombosis of an atherosclerotic artery

Embolus from the heart in arterial fibrillation or after coronary thrombosis,

Arteritis with neuropathy in diabetes

Beurger's disease

Arterial shutdown in Raynaud's disease or ergotism

Effect of intra arterial injection of thiopenthone or cytotoxic substances.

Infective gangrene

Boils and carbuncles, gas gangrene, gangrene of the scrotum (fournier's gangrene)

Traumatic

Direct, such as crashes, pressure sores and the constriction groove of strangulated bowel, or indirect, due to injury of vessels at some distance from the site of gangrene, e.g. pressure on the popliteal artery by the lower end of a fractured femur.

Physical gangrene:

As in burns, scalds, frost bite, chemicals, irradiation and electricity.

Clinical features of gangrene:

The parts lack;

Arterial pulsation,

Venous return

Capillary response to pressure (color return)

Sensation

Warmth and function.

The color of the part changes through a variety of shades according to circumstances (pallor, dusky grey, mottled, purple) until finally taking on the characteristic dark brown, greenish black or black appearance, which is due to the disintegration of haemoglobin and the formation of iron sulphide.

Clinical types of gangrene:

Dry gangrene: - occurs when the tissues are dessicated by gradual slowing of the bloodstream. It is typically the result of atherosclerosis. The affected part becomes dry and wrinkled, discolored from disintegration and greasy to the touch.

Moist gangrene: - occurs when both venous and arterial obstruction are present, when the artery is suddenly occluded, as by a ligature or embolus, and in diabetes. Infection and putrefaction are always present, the affected part becomes swollen and discolored, and the epidermis may be raise in blebs. Crepitus may be palpable, owing to infection by gas forming organisms. Moist gangrene is manifest also in such conditions as acute appendicitis and strangulated bowels.

SEPARATION OF GANGRENE

Separation by demarcation:

A zone of demarcation between the truly viable and the dead or dying tissue appears first. It is indicated on the surface by a band of hyperaemia and hyperaesthesia. Separation is achieved by the development of a layer of granulation tissue which forms between the dead and the living parts. These granulations extend into the dead tissue until those which have penetrated farthest are unable to derive adequate nourishment. Ulceration follows and thus a final line of demarcation (separation) forms which separates the gangrenous mass from healthy tissue.

In dry gangrene – If the blood supply of the proximal tissue is adequate, the final line of demarcation appears in a matter of days and separation begins to take place neatly and with the minimum of infection (separation by aseptic ulceration). Where bone is involved, complete separation takes longer than when soft tissues are affected, and the stump tends to be conical as the bone has a better blood supply than its coverings.

In moist gangrene – There is more infection and suppuration extends into the neighboring living tissues, thereby causing the final line of demarcation to be more proximal than in dry gangrene (separation by

septic ulceration). This is why dry gangrene must be kept as dry and aseptic as possible, and why every effort should be made to convert moist gangrene into the dry type.

Vague demarcation spread of gangrene, skipping and die back

In many cases of gangrene from atherosclerosis and embolism, the final demarcation is very slow to form or does not develop. Unless the arterial supply to the living tissue can be improved, the gangrene will spread to adjacent tissues or toes, or will suddenly appear as 'skip' areas further upwards beyond the line of demarcation along the lymphatic vessels or cellular tissue into the healthy parts, extensive inflammation then results. Except in diabetic gangrene without concomitant atherosclerotic obstruction these forms of spread do not usually respond to efforts to save the limb and an above knee amputation becomes necessary. To attempt local amputation in the phase of vague demarcation is to court failure as gangrene reappears in the skin flaps (die back)

Treatment

General principles

A limb saving attitude is needed in most cases of asymptomatic gangrene affecting hands and feet. The surgeon is concerned with how much can be preserved or salvaged. With arterial disease all depends upon there being a good blood supply to the limb above the gangrene, or whether a poor blood supply can be improved by such a measures as percutaneous transluminal angioplasty or direct arterial surgery. A good or an improved blood supply indicates that a conservative excision is likely to be successful and a major amputation is required for a bad crushed limb, rapidly spreading symptomatic gangrene and gas gangrene

General treatment includes that of cardiac failure, arterial fibrillation and anaemia, to improve the tissue oxygenation. A nutritious diet, is essential in all forms of gangrene. Control diabetes if present. There is pain, especially at night. Pain may be difficult to relieve. Non addictive drugs should be used whenever possible.

Local treatment:

The affected part should be kept dry

Exposure and the use of a fan may assist in the dessication and may relieve pain.

The limb must not be heated

Protection of local pressure areas, e.g. the skin of the heel or the malleoli, is required otherwise fresh patches of gangrene are likely to occur in these places

Bed cradle, padded rings.

VARIETIES OF GANGRENE

DIABETIC GANGRENE

Diabetic gangrene is due to three factors, thus;

Trophic changes resulting from peripheral neuritis

Atheroma of the arteries resulting in ischaemia

Excess of sugar in the tissues which lowers their resistance to infection including fungal infection . The neuropathic factor impairs sensation and thus favors the neglect of minor injuries and infection so that inflammation and damage to tissues are ignored .Muscular involvement is frequently accompanied by loss of reflexes and deformities. In some cases, the feet are deformed (neuropathic joints). Thick callosities develop on the sole and are the means whereby infection gains entry.

Clinical examination and investigations

Urine and blood for diabetes

Palpfate dorsalis pedis and posterior tibial pulses

Absence of rest pain and intermittent claudication imply that there is no assocated major arterial disease

Bacteriological examination is made of any pus

A radiograph may help to reveal to reveal the extent of any osteomyelitis

Treatment:

Control diabetes by diet and appropriate drugs

Infection requires incision and drainage with removal of any obviously dead tissue

Treat as explained above.

DIRECT TRAUMATIC GANGRENE

This is due to local injury and may arise as a result of crushes, pressure (as in the case of splints or plasters) or bedsore. Gangrene following severe injury like when a vehicle passed over a limb, is of the moist variety and excision without delay is indicated. Amputation may be performed as close to the damaged part as will leave the most useful limb.

Bed sores: (decubitus ulcer) are predisposed by five factors

Pressure

Injury

Anaemia

Malnutrition and

Moisture

They can appear and extend at an alarming rapidity in patients with disease or injury of the spinal cord and pts with debilitating illness

Prophylactic measures should be taken to prevent bed sores and they include;

Avoid pressure over the bony eminencies

Regular turning of pts

Nursing on specially designed beds

A bed sore is expected if erythema appears which does not change color on pressure . The part must be kept dry. Actual bed sores may be treated by lotions or by exposure to keep them as dry as possible. Once pressure sores develop, they are difficult to heal. They should be kept clean and debrided. The HB of the pt should be maintained at a normal level by transfusion of packed cells if need be. If the pt is young and healthy, excision of the dead tissue and flap pedicle skin grafting is often successful.

INDIRECT TRAUMATIC GANGRENE

This is due to interference with blood vessels;

From pressure by a fractured bone in a limb or by strangulation (strangulated hernia)

Thrombosis of a large artery following injury

Ligation of the main artery of a limb, as after division by injury

Poor technique for local digital anaesthesia. The combination of a tourniquet and an adrenaline containing local anesthetic solution can lead to permanent occlusion of all arteries.

The likelihood of gangrene depends upon the sufficiency of the collateral circulation.

Treatment:

It is directed towards the cause for example; close or open reduction of a fracture together with direct arterial surgery for the damaged vessel will prevent onset of gangrene . The limb must be kept cool to reduce metabolism to the full.

Ergot: I s a cause of gangrene among the dwellers on the shores of the Mediterranean sea and the Russian steppes who eat rye bread infected with claviceps purpurae. It also occurs in migraine sufferers who, for prophylactic reasons, unwittingly take preparations over a long period. The fingers, nose, and ears may be affected.

Physical and chemical causes of gangrene

Frost bite:

-This is due to exposure to cold, especially if accompanied by wind or high altitude (like mountain climbers)

Also encountered in elderly, or the vagrant during cold spells.

Treatment

Frost bitten parts must be warmed very gradually. Any temperature higher than that of the body is detrimental.

Clothing should be provided

Powerful analgesics

Warm drinks

Paravertebral injection of the sympathetic chain may be helpful in relieving associated vasospasms

Hyperbaric oxygen may help (at greater than normal)

Trench foot:

Is due to cold, damp and muscular activity.-

It is predisposed by tight clothing like;

Garters (elastic bands)

Ill fitting shoes

Numbness is followed by excruciating pain when boots are removed. The skin is mottled like marble and, in severe cases, blisters containing blood stained serum develop. Moist gangrene follows. The pathology is similar to that of frost bite and the treatment is essentially the same.

Inadvertent intra-arterial injection thiopenthone:

The appreciation by palpation of pulsation of the vessels and of the withdrawal of bright red blood prior to injection should prevent this calamity. Injection causes immediate and severe burning pain, with blanching (becoming pale) of the hand.

Drug abuse:

In drug addicts, usually, the femoral artery in the groin is involved and is usually due to inadvertent intra-arterial injection of drugs.

Chemical gangrene:

Carbolic acid (phenol) is the most dangerous, as anaesthesia masks the pain which occurs before the onset of gangrene. The gangrene is due to local spasms. There is also danger of severe systemic effects from absorption of phenol. Local bicarbonate soaks should be applied. Later, excision of the slough and skin grafting are necessary.

Ainhum:

A disease of unknown aetiology, usually affects black males and some females who have run barefoot in childhood. A fissure appears at the level of interphalangeal joint of a toe, usually the fifth. This fissure becomes a fibrous band, which encircle the digit and causes necrosis.

CHEST NJURY

They are common after trauma and are frequently severe. Almost half of all accident deaths include some element of chest injury, and approximately a quarter of these deaths can be directly attributed to thoracic injuries. Blunt injuries to the chest, such as from R.T.A are more common than those from penetrating trauma, except in some urban areas where penetrating injuries predominate. Among penetrating injuries, stab wounds are in general more common than gunshot wounds. Most chest injuries can be treated with relatively simple methods, such as tube thoracostomy, appropriate analgesics and good pulmonary care. However, delay in diagnosis and treatment of severe chest injuries (e.g. tension pneumothorax, aortic transaction, rib fractures with pulmonary contusion) is a common cause of preventable death after trauma.

Anatomic consideration:

The thorax can be divided into anatomic zones:

The chest wall

The pleural space

The pulmonary parenchyma

Mediastinal structures

Injuries to the chest wall include injuries to the bony thorax and shoulder girdle, as well as the soft tissue injuries.

The pleural space injuries include the pneumothorax and hemothorax, in which the potential space between the visceral and parietal pleura is occupied by either blood or air.

Pulmonary parenchymal injuries include contusion, hematoma and pneumatocele.

Mediastinal injuries involve major vascular structures and the aerodigestive tract. Mediastinal vascular injuries include injuries to the heart, aorta, and great blood vessels. Aerodigestive injuries include tracheal and bronchial disruption, traumatic asphyxia, and esophageal injuries.

Diagnosis:

Initial evaluation- Initial evaluation and treatment of a patient with chest injuries is the same as for any trauma victim;

An effective airway is secured

Adequacy of breathing is ensured and circulation is assessed and supported with control of external hemorrhage.

Establishment of a large bore peripheral venous access.

Therapy for potentially life threatening problems should be initiated immediately.

A careful relevant history should be taken, including details of the mechanism of injury. The speed and direction of impact and the degree of frontal deceleration are important factors in motor vehicle crashes. Aorta transaction is associated with severe deceleration injury. Patient not using restraint systems are likely to contact the steering wheel or the dash board of the car, placing them at an increased risk for chest injuries, such as rib fractures, flail chest, and pulmonary contusion, as well as tracheal or laryngeal injuries.

In patients with penetrating trauma, the characteristics of the offending instrument are important. External wounds can be misleading. Pts with complaints of hoarse voice, dyspnoea, throat pain and dysphagia should be carefully evaluated for injuries to the larynx and the cervical portion of the esophagus. Complaints of dyspnoea or pressure in the chest with or without chest wall pain may be indicative of pneumothorax or hemothorax.

The pt's past medical history is also important. A history of pulmonary disease, heart disease, or prior thoracic surgery can alter interpretation of diagnostic studies and affect therapeutic decisions. A history of medications, allergies, smoking, and the recent ingestion of drugs and alcohol should be obtained. The physical examination begins with complete exposure of the chest and inspection for for signs of contusion, lacerations, or penetrating wounds. These visible signs may give clues to the mechanism of injury. The breathing pattern, effectiveness in ventilation, and any abnormal motion of the chest wall should be observed. Chest wall splinting and shallow respiration may be noted in pts with rib fractures. Asymmetrical chest wall expansion with hyperinflation of one hemithorax is suggestive of tension pneumothrax. Paradoxical motion of a segment of chest wall is diagnostic of flail chest. Pressure of penetrating wounds should be noted, both anteriorly and posteriorly. The wounds should not be probed because it can turn a minor laceration injury into a pneumothorax, requiring a chest tube and longer hospitalization. Penetrating injuries below the nipple line must be presumed to involve the abdominal cavity as well.

On auscultation, the breath sounds should be compared bilaterally for quality and symmetry. Absence of breath sounds on the side is highly suggestive of hemothorax or pneumothorax.

On palpation of the chest wall may demonstrate areas of tenderness that may be associated with fractures of the ribs, sternum, or clavicle. Areas of referred pain should be noted, such as sternal compression causing lateral rib pain in the case of lateral rib fractures. Shoulder pain may be associated with diaphragmatic irritation of splenic injury (kehr's sign).Crepitus over the chest wall may be due to fractures or by air in the subcutaneous tissues from a pneumothorax. On percussion hyperesonance should raise suspicion of pneumothorax. Dullness to percussion is suggestive of hemothorax.

Other investigations

CXR

It may show fractures of ribs, clavicle, or sternum.

Widening of the mediastinum (superior)

Presence of air in the mediastinum indicating injury to the esophagus or tracheobronchial tree.

Haziness over one hemithorax can indicate a hemothorax (supine).

Treatment

Most chest injuries can be successfully managed without surgical intervention. The routine use of chest tube for the treatment of hemothorax and pneumothorax is a cornerstone of therapy.

Thoracotomy is often needed for the control of massive bleeding or bleeding that persists despite chest tube.

TYPES OF CHEST INJURIES

They are usually classified according to the type of insult that caused the damage. The injury is often influenced by the setting in which it occurred; military or civilian, urban or rural.

Most military injuries are often high velocity penetrating wounds. Low velocity gunshot wounds are replacing knife wounds as the most common in urban civilian population. Blunt injuries from motor vehicle or occupational (e.g. logging) accidents make up the majority of non-urban injuries. Penetrating wounds are becoming more frequent in sub-urban and rural areas as violent crimes increase, and blunt trauma also occurs commonly in urban areas. Complications and death are often associated with;

Pulmonary contusion

Post traumatic pulmonary insufficiency, and

Trauma to the heart and great blood vessels, are significant.

Penetrating wounds of the lower thoracic region are treacherous. The diaphragm usually rises to the level of the nipples during expiration and penetrating trauma to this area can injure the subdiaphramatic viscera. Some surgeons belief that a stab wound of the left lower part of the chest mandates early abdominal exploration, because the knife may have injured the spleen, stomach or colon. The abdominal findings in these pts may be overshadowed by initial peritoneal lavage. Whatever the cause, the principle of management should remain focused on the mechanical systems involved;

The pump

The hydraulics

And the bellows (the suction-blow system that draws atmospheric air into the alveoli and expels it). The heart must be working, and the vessels must have the integrity and suitable contents to transport the gases to and from the tissues.

Conditions requiring urgent correction:

Airway obstruction:

Most pts with major disruption of the airway leading to obstruction will not the initial accident. The leading cause of death at the accident site is airway obstruction. During the early stages of resuscitation and transportation, correctable airway obstruction may occur. The oropharynx should be cleared of any mechanical debris and the chin and neck positioned to facilitate opening the posterior pharynx (chin thrust). Tracheal intubation may be required. If the upper airway injury prevents safe access to the vocal cords from above, cricothyroidectomy should be performed.

Tension pneumothorax:

It occurs when there is injury to the lung parechyma allowing air to enter the pleural cavity (space) with each respiratory effort. It occurs when there is a flab- valve effect of the injury preventing the air re-entering the bronchial tree for regress through the trachea during expiration. Tension develops within the pleural space until equilibrium with the negative pressure the pt is able to generate is reached. At that time effective ventilation and venous blood can no longer enter the chest. Pain may be the only complaint, with no evidence of respiratory distress. However, if the lung wound is behaving like a check valve, some air escapes into the pleural cavity with each inspiration or with each cough. Gradually intrapleural pressure builds up, the lung collapses, and tension pneumothorax can develop. A shift of the mediastinum and compression of the large veins result in a decreased cardiac output that may lead to sudden death. The diagnosis should be made instantly by observation of a pt with dilated neck veins making respiratory effort but not respiratory motions and unable to move air. It is immediately confirmed by the hyperesonant percussion note over the injured hemithorax and absent or distant breath sounds. The immediate release of the tension by placement of a large bore needle followed immediately by insertion of a thoracostomy tube is life saving.

OPEN PNEUMOTHORAX

It has a sucking chest wound in which a full segment of the chest wall has been destroyed and the negative intrapleural pressure sucks air directly through the chest wall. Occurs most commonly after shot gun blasts, explosions with flying debris or piercing (implement) injuries.It may or not be associated with underlying parenchymal damage wound.

Patient may present with normal or collapsed neck veins. Patient makes respiratory motions but no air movement.

On inspection (immediate) there is a chest wound.

The patient is stabilized by any mechanical covering over the wound. As soon as possible a water tight dressing should be placed in place and an intercostals catheter inserted into the pleural cavity. Early debridement and formal closure of the wound should then be performed.

MASSIVE FLAIL CHEST

When severe blunt injury results in two point fractures of four or more ribs, a segment of the chest wall becomes flail. On inspiration, the negative pressure in the chest pulls the unstable segment of the wall inwards in a paradoxical motion. The pt may be unable to develop sufficient intra tracheal negative pressure to maintain adequate ventilation, and

Atelectasis

Hypoxia and

Hypercapnia occur.

A pt who is conscious may splint the segment sufficiently to make it inapparent to cursory examination, but the continuing extra effort In the attempt to move air soon leads to tiring and may result in sudden respiratory decompensation. The progressing failure may be aggravated by the developing pulmonary contusion that accompanies blunt trauma sufficient to break multiple ribs. In the unconscious pt, the lesion may be less dangerous because it is more readily recognized and more apt to be treated early.

In massive flail chest, the diagnosis may be difficult unless the chest wall is visualized during the respiratory effort. If unconscious, the pt ordinarily is making vigorous respiratory motions but moving little air. The paradoxical segment should be obvious. The pt who is awake may exhibit a very rapid shallow breathing pattern at or above 40 breaths per minute. Other aspects of management include endotracheal intubation and positive pressure ventilations are mandatory.

THE SKULL

MICROCEPHALLY:

An abnormally small head.

May be associated with agenesis of the brain and imbecility

May result later from premature synostosis in normal child

OXYCEPHALLY

The skull is egg shaped following premature fusion of the sutures

Most patients develop increased intracranial pressure.

Treatment: cranioplasty allows normal skull expansion and cerebral development.

MENINGOCELE:

Is protrusion of portion of pouch of dura matter through congenital bone defect forming a cyst filled with C.S.F.

Signs and symptoms:

Protrusion of a part of dura matter through a defect in the skull at the root of the nose or over the occipital bone.

Transphenoidal projections protrude through the base of the skull into the nasopharynx mimicking the nasal polyps. Attempted removal of such a projections has resulted In development of meningitis.

Meningocele presents at birth and forms a tense rounded swelling which is translucent. It yields an impulse when the child coughs or cries. Growth of the skull may occlude the neck of small sac and a cyst remains which does not pulsate and is not affected by coughing.

ENCEPHALOCELE:

It is similar to meningpocele but some part of rain is also involved. If this cerebral extension contains part of a ventricle the encephalocele is called hydroencephalocele. In encephalocele and hydroencephalocele vascular pulsations are present. The child may be still born or may have an associated degree of idiocy or both.

Treatment for meningocele and encephalocele:

Skin surface should be protected by tulle grass to prevent ulceration and infection.

If at the age of 1/12 the child shows normal development operation is done under L.A as the child sucks a feeding bottle. A curve incision is made in one margin of the sac so that when the wound is sutured the incision will not overly the bone. The neck of the sac is identified, ligated and the sac is removed together with the excess skin. Muscle fascia are brought together over the bone defect.

Many cases of meningocele have a small encephalocele at the base and if this is so it is removed with the sac without any harmful effect as the tissue will already be functionless.

TUMORS OF THE SKULL

Benign tumors:

They are rare

Occasionally an “ivory” osteoma (compact osteoma) arises in the region of an air sinus.

The lesion constitutes of a small knot of extremely hard and dense but otherwise normal often arising in the inner or outer table of the skull

Malignant tumors:

They are the same as those of the other bones.

Pericranial sarcoma:

Its consistence depends on its vascularity and rate of growth. It may be pulsatile or of an almost bone hardness. It is not common. The commonest is secondary from the breast, prostate, and thyroid glands.

Hypernephroma (Grawitz tumor):

It is a malignant tumor of the kidneys. It grows to produce more rapidly growing vascular tumors which pulsate when the outer table is eroded. Cellular deposits from hypernephroma produces a single clear area with irregular margin on X-ray of the skull. Deposits from ca breast are usually multiple.

INTRACRANIAL ABSCESS:

There are three types;

Extradural abscess: it is produced by osteomyelitis of the skullwhich may in turn occur as a result of infection from the following sites;

Direct infection such as a compound fracture of the skull.

Local extension of infection from the frontal sinus or mastoid anthrum or cellulitis of the skull

Blood borne infection by circulating organisms practically unknown but rarely follows boozing of bone in young children. Extradural abscess is usually secondary to spread of infection from middle ear or frontal sinus.

Clinical features:

They are those of osteomyelitis;

Acute localized head ache

Tenderness on local percussion

Localized pitting edema of the skull over the affected area “pott’s puffy tumor”

Constitutional signs and symptoms i.e. nausea, general malaise, rigors and fever

Rarely if the abscess is large there is evidence of pressure of neurological signs such as convulsions, paralysis, and paresis.

Treatment:

Drain the abscess and the process depends on the cause of the abscess. Many cases are dealt with by removal of the posterior wall of the frontal sinus incase the abscess is secondary to frontal sinusitis.

Burr hole at the site of edema, the dura is pressed slightly inwards to allow the pus to escape spontaneously. The pus can be removed by suction. Penicillin powder is then insufflated (blown) into the wound and then wound drained for 24hrs. Systemic antibiotics in adequate doses should be given.

Subdural abscess:

It used to be fatal but now can be treated with 30% mortality. It is produced by thrombophlebitis of superior longitudinal sinus (one of the channels containing venous blood). It usually spreads from infections of the frontal sinus or accessory air cells in the mastoid process. Infection extends from the superior sinus to the superior cerebral veins and thus infects the subdural space. The abscess extends in the subdural space over the cerebral hemispheres often bilaterally and must be treated before the spread to the inner or under aspects of the cerebral hemispheres. Successful treatment depends on; early recognition and early intervention.

Clinical features:

It follows a heavy cold or influenza

The pt runs a high temperature becoming dehydrated with wrinkled skin.

Blockage of the superior compartment of the superior sinus of the frontal sinus into which Csf is absorbed and produces a raised intracranial pressure with head ache and later on papilloedema (edema of the optic disc).

Blockage of the lower compartment receiving the superior cerebral veins may cause epilepsy and paralysis of sudden onset and associated features.

All these appear in a matter of days (sudden).

Treatment:

Bilateral frontal burr holes are made just between the hairlines above temporal crest.

On opening dura thin pus is found in the subdural space and is allowed to escape.

A fine catheter is then introduced to instill an appropriate antibiotic into the space about 2-3 ml

Intravenous systemic antibiotics in full dose.a

Intracerebral abscess:

Are produced by

- Implantation of infection

- Blood metastasis

- Local extension of an adjacent infection (more than 50% result from extension from the middle ear)

NB: in diagnosis general features are more important than focal features which are few in number and come late.

Clinical features:

Acute stage

Persistent pyrexia and headache should lead to suspicion of an abscess in association with an ear or sinus infection.

Raised pulse rate at the start of the acute stage but as the abscess enlarges and the intracranial pressure is raised there is slowing of the pulse.

Irritability

Drowsiness

Vomiting

Leucocytosis but may not be significant especially if some other condition is present.

Focal signs are often absent in the acute stage

Lumbar puncture will show increased cells and proteins at about 80mg%. as a result of administration of antibiotics in the treatment of the primary focus of infection. Many cases are arrested and many abscesses are aborted in the acute stage without frank pus

Sub acute stage:

Low temperature to subnormal

Low pulse rate

If the abscess is located on the frontal lobe there is a contralateral facial weakness (opposite side of the face)

If the abscess is on the temporal lobe there will be contralateral hemiparesis with absent abdominal reflexes and an extensor plantar response (Babinski +ve)

If on the cerebellum there will be nystigmus, hypotonus and incorodination on same side of the lesion

Lp shows reduced number of cells but an increase in protein s to 120 mg %

Chronic stage:

Intermittent headache

General ill health

Pallor

Catchexia

C.s.f may return to normal but the abscess continues to enlarge

Skull adopts the enlargement of the abscess and can accommodate the abscess but when no more space is available the pt develops features of increased intracranial pressure

NB: There may be no physical sign in the chronic stage. In all cases of suspected cerebral abscess it is important to examine all sources of infection or foci. For example, o.m, sinusitis, wound on scalp and tonsillitis. In many cases probable cause is usually clear but discharge from an ear may cease by the time intracranial abscess develops.

Treatment:

Formerly, drainage of the abscess was the method of treatment.

Aspiration and occasional excision have now replaced drainage. Drainage is only indicated if the abscess is superficial as in the abscess following penetrating wound.

In the early stage of cerebral inflammation antibiotics are indicated.

Ventriculography may be required to determine whether there is an abscess. When the abscess has localized or when signs show danger a diagnostic burr hole is made and instillation of antibiotic. The burr holes are made depending on where the abscess is situated.

For the frontal abscess the burr hole is made immediately within the hairline

F or cerebellar abscess the burr hole is made over the occipital plate.

CEREBRAL TUMORS

They arise in connection from with the meninges, nerve sheaths or from the cerebral substance itself. Tumors of the pituitary gland, gummas, tuberculomas, vascular malformations, blood clots, chronic

abscesses can also contribute to cerebral tumors. Secondary Ca is by far more common than primary intracranial tumors. Secondary deposits commonly originate from the lung but may originate from any organ of the body and the naso-pharynx. When secondary deposits have been excluded the following primary tumors should be suspected;

meningioma 18%

Neurinoma 8%

glioma 42%

pituitary adenoma 12%

hemiopharyngioma 5%

blood vessel tumors 2%

glanulomas and unclassified rare tumors 6%

Meningioma:

- they vary in structure and vascularity
- are globular
- tumor arises from the arachnoid and gets secondary attachment to the dura
- the arteries and veins of the dura provide nourishment to the tumor

Neurinoma:

- Found from the sheath of the 8th cranial nerve (auditory nerve)
- May be multiple in association with multiple neurofibromatosis of the skin
- May occur in association with cerebrospinal meningiomas
  Rarely occurs on its own.

Clinical features

Initial period of silent growth- All cerebral tumors have a silent growth period. The tumors vary depending on the site and rate of growth. If the tumor is not near any area which will produce signs and symptoms it will occupy space in the sub arachnoid cisterns (space) and they serve as reservoirs foe the CSF. The tumor will then flatten and displace the ventricle and the brain until it cannot gain any more width. Once there is no more space the tumor will produce symptoms of increased intracranial pressure

like, effortless vomiting, papilloedema, morning headache etc. in the case of haemangioma the period may take several yrs. I f the tumor is situated in a vital area it may as a result of its local effects produce symptoms of epilepsy or progressive neurological syndromes before any evidence of increased intracranial pressure is produced. The mere absence of headache, vomiting and papilloedema does not exclude the tumor.

Focal syndromes and epilepsy –

Epilepsy arising for the first time in adult life should always be suspected to be due to a tumor until proved otherwise. Idiopathic epilepsy does not occur before the age 6yrs. 99% of cases of idiopathic epilepsy have their first seizure before the age of 30yrs. After 30yrs epilepsy is usually symptomatic. In patients of between 30-50 yrs cerebral tumor is usually a common cause of epilepsy.

Progressive focal syndromes should also be regarded as indicating cerebral tumors until proved otherwise. Only cerebral tumor produces a steadily progressive syndrome

Raised intracranial pressure : May develop in association with focal symptoms or may be the sole evidence of the presence of a tumor. Signs of raised intracranial pressure occur. Late in the tumors of the frontal lobes, signs of raised intracranial pressure occur. Early in the tumors of temporal and parietal lobes which obstruct the outflow of the CSF from the adjacent ventricle. They occur earliest in tumors located in the midline and posterior fossa which obstruct the flow of CSF from both ventricles and produce an internal hydrocephalous.

Signs and symptoms:

Headache in the early morning aggravated by coughing and straining.

Vomiting occurs without warning and not preceded by nausea not related to food

Bradycardia

Retarded mental activity

Blindness from papilloedema

Stage of cone formation

When the intracranial pressure becomes high, the inner border of one hemisphere may be forced under the falx cerebri. Eventually the hemisphere blocks the pathway for the absorption of the CSF. The temporal lobe may be forced down from above into the tentorial opening. A part of the cerebellum may also be pushed into the tentorial opening. These are the ominous signs of a threatened cone formation;

violent paroxysmal norcturnal headaches

Drowsiness

Slow pulse

Slow cerebration

Neck stiffness

Unilateral pupillary dilatation (an urgent sign)

NB: lumbar puncture must be avoided at the stage of cone formation

Frontal lobe tumors

If situated deeply they present with the following;

Progressive change in personality

Lack of insight

Neglect of normal hobbies, occupation and duties

Alteration in emotional reaction particularly noticeable to relatives e.g euphoria, irritability

Epilepsy is generalized in type and localizing signs are limited to contralateral facial weakness.

Parietal lobe tumors

They produce;

Jacksonian epilepsy

Progressive hemiparesis

Examination reveals loss of touch resulting in inability to recognize the size and shape of objects

Deeply situated tumors may show defects of a special relationship and loss of power of calculation.

Occipital lobe tumors:

Present with generalized epilepsy which;

Is preceded by an aura or flashing lights in the contralateral visual field

Homonymous hemianopia (loss of vision on the same side)

Temporal lobe tumors

Those on the left side produce;

Progressive apasia

Visual and auditory hallucination

Generalized convulsions

Hallucinations of smell and taste

Dreamy states of unreality

Localizing signs e.g. hemiparesis

Investigations of a cerebral tumor

History: may sometimes locate the site of the tumor and also give a hint of it's pathological type. If the complaints are of long standing it shows it is a slow growing tumor. A short history may be due to final breakdown of adaptation in a slowly growing tumor or may indicate a rapid malignant growth. History may suggest a primary disease to which a cerebral condition is only secondary. Since metastasis cancer is far more common than primary cerebral tumors. Attention must be paid to know about;

Wt loss

Recent cough or

Hemoptysis

Secondary brain abscess is suggested by a history of lung abscess and bronchiectasis. Symptoms like catchexia and ear discharge suggest otitis brain abscess. Wt lossis always suspicious as there is no wasting with primary cerebral tumor.

Clinical examination: it must be include general examination in search of primary diseases.

Neurological examination of cranial nerves and nerve tracts. This may help to localize where the tumor is but does not indicate what kind of tumor it is.

Others (accessory) investigations. This are essential and must be done;

Skull x-ray

Cxr

Blood for ESR

CXR may reveal an unsuspected bronchial ca

30% of bronchial ca present with signs of cerebral symptomsbefore any chest symptoms

A skull X-ray may show presence of a tumor in the following ways;

By pressure changes which include (a) a beaten silver appearance of the vault as a result of the pressure of a tight involusions (b) separation of sutures especially in young patients (c) erosions of the crinoids process is a valuable sign because it indicates a long standing pressure and therefore a possibly recoverable tumor

By lateral displacement of calcified pineal shadow indicating the site of the tumor. The pineal body is a small redish grey conical structure situated in dorsal surface of the midbrain. Its function is not known but thought to secrete a hormone related to growth.

By characteristic calcification produced by tumors such as astrocytoma (slow growing tumor of the glial tissue of the brain), angioma, meningioma, tuberculoma and 50% of craniopharyngi omas don't show calcification

By alteration in skull vascular markings especially in meningiomas

By changes in the skullbones including local expansion at the site of a cyst and evidence of bone destruction

E.E.G: Certain characteristic wave forms indicates the site or presence of deep seated tumor and also distinguish between epileptic seizure produced by focal lesions and seizure produced by idiopathic causes

Lumbar puncture in early cases where it is necessary to exclude non-tumerous conditions like meningitis and tuberculosis. The pressure and content of CSF are recorded and increase in pressure and protein suggests a tumor. Lumbar puncture should be avoided in the presence of increased intracranial pressure is absolutely contraindicated at the stage of cone formation

Surgical investigation by arteriography. Involves ingestion of dye into common carotid artery and films taken at 1 second intervals. This helps to localize a silent tumor producing pressure but no physical signs or may indicate extent of a known tumor and also provide evidence of its type

THE NECK

The brachial apparatus and its abnormalities:

Brachial cyst (already discussed)

Cystic hygroma (already discussed)

Brachial fistula

Cervical lymphadenitis

Brachial fistula:

It may be unilateral or bilateral. It presents a persistent second brachial cleft that did not disappear whose occluding membrane has been interrupted. The external orifice of the fistula is nearly always situated on the lower 1/3 of the neck near the anterior border of sternomastoid. The anterior orifice is situated on the anterior aspect. The inner orifice is located on the anterior aspect of the posterior pillar of the faucies just behind the tonsils. But more often the tract ends blinds at the lateral pharyngeal wall and becomes a brachial sinus other than a fistula. The pillars of the faucies lie lateral to the tonsils. The track closed with muscles and lined by ciliated columnar epithelium remains until the lining has been destroyed by repeated infection. The discharge is purulent. The fistula can be secondary to an incision of an infected brachial cyst.

Treatment:

It should be excised when causing troublesome symptoms like producing mucous.

CERVICAL LYMPHADENITIS

There are approximately 800 lymphnodes in the body. About 300 lie in the neck. Inflammation of lymph nodes in the neck is common. Infection occurs in the oral and nasal cavities,ear, scalp, and the face.

Acute cervical lymphadenitis: the affected lymph nodes are enlarged, tender and a varying degree of pyrexia. Treatment is directed to the general condition and focus of infection. The neck should be protected by a bandage or cotton wool. If inspite of antibiotic therapy pain continues or certain lymph nodes become large formentation is done. Abscess should be drained if formed.

Chronic cervical lymphadenitis: in an early stage it is extremely difficulty to differentiate tuberculous adenitis but clinical experience shows that chronically inflamed cervical lymphnodes within 3-4 weeks is nearly always Tb adenitis.

Tuberculous adenitis: Majority of the patients affected are children but can occur for the first time at any age. Usually one group of nodes is affected first most frequently are those of the upper jugular chain. More rarely you have all groups affected and there is matting or periadenitis becoming evident. When there is widespread infection.

Cervical source of infection: majority Tb bacilli gain entrance through the tonsil on the corresponding side. The nodes of the posterior triangle are infected in 22% of cases. That probably stems from adenoidal infection. Contrary to what is believed it is the human but not the bovine strain that is responsible for Tb adenitis in about 90% of the cases. In black Africans it accounts for 100% cases (human strains). In 80% of the cases the tuberculous process is virtually limited to the clinically affected lymph nodes. Nevertheless a primary focus in the lungs should be suspected. Tb adenitis can coexist with renal Tb and therefore urine should be examined for Tb bacilli. If the patient develops resistance, or as a result of inappropriate treatment, it becomes chronic. In other circumstances caseating material liquefies, breaks through the capsules of nodes and a cold abscess results. The pus is at first confined to the deep cervical fascia. In a few week's time these dense sheath becomes eroded at one point and pus

flows through the small opening into the much larger space beneath the superficial fascia. The process is now said to have reached a stage of collar stud abscess. The superficial abscess enlarges and unless suitable treatment is adopted the skin soon becomes red over the centre of a fluctuating swelling and before long a distinguishing sinus forms.

DDX: Depends on the nature of swelling;

When the swelling is solid > chronic non tuberculous lymphadenitis

>Hodgkins lymphoma

>nonHodgkins lymphoma

>secondary malignant disease.

- cystic swelling * brachial cyst

* extension of an abscess connected with a tuberculous cervical vertebrae.

- sinus -actinomycosis

- acquired brachial fistula

Treatment

General measures and appropriate chemotherapy

Aspirate abscess if present and culture the Tb bacilli and sensitivity to anti Tb drugs.

NB: Repeated aspiration of the collar stud abscess is not recommended because it can lead to sinus formation or secondary infection.

Operation for collar stud abscess. An incision is made in line with the skin creases and the pusIn the superficial compartment is mopped away. The whole space in the big fascia is opened so that caseating lymph node is scrapped with a curate and the cavity packed with iodoform gauze. The gauze is removed after 24hrs but the sutures removed after 10 days.

Excision of lymph nodes when there is no local response to chemotherapyand when a sinus forms. If there is coexistent PTB it is illogical to remove the lymph nodes.

PRIMARY MALIGNANT TUMORS OF THE NECK

CAROTID BODY TUMOR: (PITATO TUMOR):

The carotid body is situated at the bifurcation of the carotid artery. It is the most important part of the chemoreceptor system. Carotid body tumors remain localized for yrs but regional metastasis occur in the about 20-30% of the cases while distant metastasis occur less frequently.

Clinical features:

Unilateral

Middle age but may come earlier

Diagnosis is suggested by a long history of a lump at the bifurcation of the carotid artery which moves from side to side, left to right but not vertically.

Usually a pulsating vessel overlies the outer surface of the tumor.

Investigations.

Arteriography is valuable and shows the carotid fork to be broader and bluish or a red colouration outlining the tumor. The special danger of excising the tumor is due to its vascularity and this can cause;

Torrential hemorrhage occurring if biopsy is attempted in the wrong belief that the lump is an infected neoplastic lymph node.

Puncture of one of the carotid arteries and to control of bleeding by artery forceps results i n occlusion of carotid artery with hemiplegia or death occurring in at least 33% of the cases.

If extirpation is to be attempted it is essential to have a length of silicon bypass tubing in case carotid occlusion becomes necessary.

In some cases it is possible to dissect the tumor away from the carotid York. But when the tumor is inseparable resection is necessary and a bypass is necessary while a veinautografty is being inserted to restore arterial continuity. Recurrence is unusual. The tumor is not sensitive to radiotherapy.

THE THYROID GLAND AND THE THYROGLOSSAL TRACT

Embryology: thyroid gland develops from the median duct of the pharynx.

Surgical anatomy: -

it weighs gms

lies deep to infrahyoid muscle and the sternomastoid

medially related to thyroid cartilage and upper portion of tracheal and the recurrent laryngeal nerves which lies in the groove between trachea and the esophagus

posteriorly lies the pharynx, esophagus, parotid glands which may be imbedded into the thyroid or lies close to it, and the common carotid artery.

The gland consists of left and right lobe joined by an isthmus. The pyramidal lobe is situated on the isthmus and it marks the junction of the thyroglossal tract. The pretracheal fascia surrounds the gland and is attached to the thyroid cartilage and the hyoid bone. This explains why the gland moves down

with swallowing. The functioning unit of the thyroid is a lobule supplied by a single arteriole consisting of 20-40 follicles lined by cuboidal epithelium. The resting follicles contain cholloid in which fibroglobulin is stored.

Blood supply:

Arterial supply is very rich, 2 superior and 2 inferior thyroid arteries and branches from tracheal and esophageal arteries. Occasionally there is an additional supply from the thyroid long artery, a branch of inominate artery.

Venous drainage:

Superior and middle thyroid veins which drain into the internal jugular veins while inferior thyroid veins drain to the inferior thyroid vein.

Lymphatic drainage:

Some lymph channels pass directly to deep cervical lymph nodes but the subcapsular plexus drains mainly to the pretracheal and paratracheal nodes and the nodes of superior and inferior thyroid veins and then finally these drains to the deep and mediasternal lymph nodes.

HYPOTHYROIDISM

Classification:

Failure of thyroid development;

Complete

Partial

Endemic cretinism (often goitorous)

Iatrogenic hypothyroidism

After thyroidectomy

After radio-iodine therapy

After pituitary removal

Drug induced by anti-thyroid drugs, para-aminosalicylic acid (P.A.S) and iodides in excess.

Autoimmune thyroiditis which can be;

Non-goitorous (primary myxoedema)

Goitorou (Hashimotos disease or goiter)

Goitorogens;

Vegetables

Peanuts

Cabbage

Tunip

Sprout

Drugs

Propylthiurocil

Carbimazolepotassium percherates

Potassium thiocyanate

Iodides

Dyshormogenesis

Vascular damage to the anterior pituitary.

CRETINISM (INFANTILE OR FETAL HYPOTHYROIDISM)

Sporadic cretinism is due to complete or partial failure of thyroid development. The parents and the other children may be normal. Partial failure causes juvenile myxoedema. In endemic areas goitorous cretinism is common due to maternal and fetal iodine deficiency. Immediate diagnosis and treatment with thyroxine within a few days of birth are vital if physical and psychological development is to occur or further deterioration is to be prevented. Women under treatment with anti-thyroidbdrugs may give birth to hypothyroidic infants.

ADULT HYPOTHYROIDISM

Myxoedema is a very advanced form of adult hypothyroidism. Myxoedema should never be applied to the mild degree of hypothyroidism commonly seen more frequently after thyroidectomy and with auto-immune thyroiditis.

Symptoms;

Significant early symptoms include

Tiredness

Mental lethargy

Cold intolerance

Increase in wt

Menstrual disturbance

Carpal tunnel syndrome due to increase in tissue fluid which causes pressure to the median nerve characterized by numbness, tingling, and constipation.

Signs;

Slow pulse rate

Dry skin and hair

Cold extremities

Peri-orbital puffiness

Coarse voice

Slow movement and slow action of the ankle jerk.

Investigations;

Serum T4 is below 55 nmol/l

Radio-iodine studies show reduced thyroid uptake and increased renal excretion. Iodine uptake of less than 12%/24hrs is diagnostic.

Serum T.S.H are raised

T.R.H test is helpful in doubtful cases.

Treatment:

The hormone, Levo-thyroxine is curative. A full replacement dose of 0.15- 0.2 mg/day can be given as a single dose. In the elderly or those with myocardial insufficiency the initial dose must be as low as 0.05mg/day and increased carefully.

If a rapid short lived response is required tri-iodothyronine is used.

MYXOEDEMA

The s/s of hypothyroidism are exaggerated. There is a typical appearance;

Pt has a bloated look (face looks swollen)

Lips are pushed out

A dull expression

Supra-clavicular puffiness

Malar flush (flushing of the cheeks and a yellow tinge of the skin)

Myxoedema comma occurs in neglected cases. In myxoedema comma carries a high risk of mortality. Temperature is low and the pt must be warmed slowly. 1 gm of hydrocortisone i.v should be given and i.v tri-iodothyronine slowly increasing doses.

PRIMARY OR ATROPHIC MYXOEDEMA

It is considered to be an auto-immune condition which is similar to Hashimotos disease but without goiter formation from T.S.H stimulation. It is more severe than goitorous myxoedema.

DYSHORMOGENEISI AND GOITOROGENS

Genetically determined deficiencies in enzyme thatcontrolthe synthesis of hormone thyroxine and it leads to formation of goiter. If the enzyme deficiency is of mod erate degree, a simple uethyroidgoiter occurs. Similarly goitorogens may produce goiter with or without hypothyroidism.

GOITER

Definition: An enlarged thyroid gland.

Classification of goiter:

Simple goiter (may be endemic or sporadic) + euthyroid which is divided into diffuse hyperplastic and nodular goiter. There is no hypothyroidism.

Toxic goiter: It is divided into;

Diffuse toxic goiter (Grave's disease) where the whole gland is enlarged

Toxic nodular goiter

Toxic nodule (solitary)

Neoplastic goiter: which can be benign or malignant.

Thyroiditis which can be granulomatous thyroid (DeGuervan's disease), auto-immune thyroiditis or Riedel's thyroiditis.

Other rare goiters:

Acute bacterial thyroiditis

Chronic bacterial thyroiditis (Tb or syphilis)

Amyloid goiter

SIMPLE GOITER:

It is due to stimulation of the thyroid gland by anterior pituitary, i.e. increased level of T.S.H. T.SH secretion is increased by low levels of circulating thyroid hormones. Any factor that maintains a persistently low level of circulating thyroid hormone can be responsible for simple goiter. The most important is iodine deficiency but defects in hormone synthesis may also be responsible.

Aetiological factor:

Iodine deficiency:- The daily requirement is about 100-105 ug. In nearly all cases where simple goiter is endemic there is a very low iodine content in water and food. In Kenya, Eburu nearGilgil, lowland where soil lacks iodine or the water supply comes from far away in the highlands. Calicium is also goitorogenic and goiter is common in low iodine areas of chalk or limestone. Although iodides in food and water may be adequate there may be failure of intestinal absorption.

Defects in the synthesis of thyroid hormones

Enzyme deficiency within the gland which are responsible for many sporadic goiters in non-endemic areas. And there is often family history in this cases suggesting genetic defect. If the iodine intake defect is very high and enzyme deficiency may be overcome. Iceland has never reported any goiter case because they encourage people to a lot of iodine. Enzyme deficiency is often associated with a low iodine intake e.g. a dislike for seafood.

Well known goitrogens are in the vegetables of the brassica family e.g kale, cabbage and tunips. They contain thiocyanate. Others are drugs such as P.A.S and anti-thyroid drugswhich interfere with iodine trapping. Carbimazole and Flouracil compound interfere with oxidation of iodine to thyrocin to form triiodothyrosines. Iodides in large amounts are goitrogenic because they inhibit the organic binding of iodine and give rise to an iodide goiter which is usually seen in asthmatics who have taken preparations containing iodides for a long period e.g. Felsol.

Clinical types of simple goiter:

Simple hyperplastic simple goiter—It corresponds to the first stages of the natural history of a simple goiter due to persistent T.S.H stimulation causing hyperplasia. The goiter appears in childhood in endemic areas but in sporadic cases it appears in puberty when metabolic demands are very high. If T.SH stimulation ceases the goiter may regress but tends to recur later at times of stress such as pregnancy. The gland is; soft, diffuse, may be large enough to cause discomfort. A choroid goiter is a late stage of a diffused hyperplasia when T.S.H stimulation has fallen off.

Nodular goiter:- Persistent fluctuating T.S.H stimulation results inevitably in progressive nodular formation. Nodules are usually multiple forming a multinodular goiter. Occasionally only one macroscopic nodule is found but microscopic changes will be present throughout the gland. This is one form of clinically solitary nodule. Nodules appear early in endemic goiter and appear later in sporadic goiter 20-30 yrs. Although the pt herself will not be aware of the goiter until late 40s or 40s. all types of simple goiter are far more common in females than males. Recently oestrogen receptors have been identified in normal thyroid tissue.

Diagnosis:

Usually straight forward. Nodules are palpable and often visible, smooth, firm and not hard. It is not painful and moves freely with swallowing. Hardness or irregularity due to calcification may mask carcinoma. A painful nodule with sudden appearance or rapid enlargement raises suspicion of ca but is usually due to hemorrhage to a single nodule.

Investigations:

Test for thyroid function to exclude hyperthyroidism

Titers of thyroid antibodies estimation to differentiate nodular goiter from Hashimoto's disease.

Plain X-ray of the neck may show calcification and tracheal deviation or compression.

Complications:

Tracheal obstruction due to gross lateral replacement or decompression of the trachea in lateral or antero-lateral.

Acute respiratory obstruction

Secondary thyroid toxicosis

Carcinoma usually of follicular thyroid ca. it is uncommon but an increased incidence has been reported from endemic areas.

Prevention and treatment of simple goiter:

All cooking and table salt should be iodized by adding potassium iodide 1:1000. In endemic areas this prophylactic measures have reduced simple goiter.

In the early stage hyperplastic goiter is reversible when or if levothyroxine is given in maximum doses 0.2 mg/day for several months and then very slowly tail off to 0.1 mg/day which should then continue for several yrs.

If regression does not occur thyroidectomy may be done for cosmetic measures or pressure symptoms.

Nodular stage of simple goiter multinodular goiter is often uncomfortable and unsightly and view of the possible complications subtotal thyroidectomy is advisable unless the expectation of life is short. Resection aims at removing the nodules and having up to 8gms of relatively normal tissue in each remnant.

Occasionally the multinodular change is asymmetrical with one lobe. One lobe significantly involved with only a minimal amount and even micro-nodules. Under this circumstances unilateral total lobectomy of the affected side is the appropriate management. In many cases the causative factors persist and recurrence is likely particularly in the younger patient unless further T.S.H stimul ation is prevented. 0.1 mg of levothyroxine/day should be given post operatively to all pts until after menopause. If one thyroid lobe appears normal in size and inconsistent it is not justified to resect that lobe but post operative levothyroxine is essential.

Clinically solitary nodule

Def. A goiter which on clinical examination appears to be a simple nodule in otherwise normal gland. There are two categories;

Nodules in which there is a certainty or suspicion of malignancy. In these nodules exploration is essential.

2nd far larger category in which there is a smooth, firm, mobile nodule which is probably benign and small but carries a significant risk of being a carcinoma. In this category about 50% proof to be simple multilobular goiter. The thyroid status of this pt must be established by clinical examination and by laboratory tests. Isotope scanning is essential to find if there is hyperthyroidism. Isotope scanning is only essential if there is hyperthyroidism but of limited value if there is malignancy. This test should divide the pt into three categories;

Those who are hyperthyroid with a “hot” (hyperactive) nodules i.e. one which takes up isotope while the surrounding thyroid is inactive because the nodule is producing such a high levels of the hormo ne that T.S.H secretion is suppressed.

Those who are euthyroid with a “warm” (inactive) nodule. A warm nodule takes up isotope and so does the normal tissue around it.

Those who are euthyroid with a “cold” (inactive) nodule. A cold nodule does not take up isotope.

Solitary toxic nodule:

It is never malignant.

It is a toxic adenoma.

It is treated by excision or radio-iodine.

It is a functioning adenoma or a simple nodule with some active thyroid tissue in it. Very rarely a very differentiated ca may take up isotope. Because a functional adenoma may develop an overactive adenoma it should be excised.

Euthyroid cold nodule: it is suspect because a carcinoma so rarely takes up isotope, if normal thyroid tissue is present. It should be excised. Resection entails taking a wide marginal healthy thyroid tissue. In order to achieve this total lobectomy should be done on the side of the lesion including the thyroid isthmus. Subtotal lobectomy is rarely appropriated and should only be performed when the; nodule is small, anteriorly situated, and has been shown on cytology to be benign. Incisional biopsy is totally contraindicated as it could result in seedling of malignant cells.

Other investigations of solitary nodule include;

Needle biopsy—It is an available technique which can aid diagnosis and influence its subsequent management. Two distinct types of needle biopsy are

(i). Trucut large needle biopsy. It produces a core tissue for cytology. It has a high diagnostic accuracy but has poor pt compliance. They are also associated with complications like pain, bleeding, tracheal damage and recurrent laryngeal nerve damage.

(ii). Thin needle aspiration biopsy (ABC) –It produces a thin smear for cytology. It has an excellent pt compliance. It is simple and quick to perform even in OPD. It can be performed repeatedly and if well performed it has a high diagnostic accuracy many aspirations have been done without complications. Tumor implantation with seeding does not occur. Thyroid condition which tend to be diagnosed with thin needle ABC are (i) colloid nodules (ii) thyroiditis (iii) papillary ca (iv) anaplastic ca(v) lymphoma

Thyroid cysts can also be aspirated and the aspirate examined cytologically. However there is need for caution as many cysts occur in malignancies. After aspirating a cyst a check must be made for any residual mass. Ideally a frther sample should be taken from a cyst wall and take it for cytology. Any cyst which reaccumulates after initial aspiration must be subjected to surgery.

Ultra sound- this is of limited value in the diagnosis of malignancy but should help to differentiate between solid and cystic nodules and other nodules present but can't be palpable nodule.

Computerized axial tomography (CAT SCAN) and magnetic resonance imaging (MRI) are sophisticated. They have only small role to play in the day to –day management of thyroid disorders

Fluorescent scanning- It permits an in vivo demonstration of thyroid gland iodine content. In an old solitary nodule the ratio of iodine content in the nodule to that of the corresponding of the contralateral lobe may be used to distinguish between benign from malignant lesions.

NB: Clinically solitary nodule presents a diagnostic problem when the nodule is smooth, firm, and mobile in a euthyroid pt. scanning, needle bx and ultra sound may increase or decrease the suspicion of malignancy but excisional biopsy is the only certain diagnostic procedure.

Retrosternal goiter: a very small number of retrosternal goiters arise from ectopic thyroid tissue but most arise from the lower pole of a nodular goiter. If the neck is short and the tracheal muscles are strong as in men the negative intra- thoracic pressure tends to draw these nodules to the superior mediastinum. The degree of descent varies and this accounts for 3 types of retrosternal goiter.

Substernal type – this is when the nodule is palpable clinically

Plunging type — occurs when intra thoracic goiter is occasionally forced into the neck due to increased intra-thoracic pressure.

Intra-thoracic goiter.

Clinical features of retrosternal goiter

h/o a previously cervical goiter which has disappeared is common.

A retrosternal ,goiter may be symptomless or produce severe symptoms such as; dyspnoea especially at night and cough or stridor. The pt may attend a chest clinic with a diagnosis of Asthma before the discovery of the true diagnosis.

Dysphagia

Engorgement of neck veins and superficial veins on the chest wall. In severe cases there may be obstruction of superior vena cava

Recurrent laryngeal nerve paralysis resulting in dysphonia (hoarseness) rare.

Malignant or toxic.

Investigations:

X-rays show a soft tissue shadow in the superior mediastinum and sometimes with calcification often causing deviation and compression of the trachea.

Iodine 1, 2, 3 scan may help to distinguish a retrosternal goiter from a retrosternal tumor.

If there are obstructive symptoms it not wise to treat with anti-thyroid drugs or radio-iodine as they enlarge the goiter further. Resection can almost always be carried out from the neck and a midline

sternoctomy is often unnecessary. Hemorrhage is rarely a peoblem because the goiter takes its blood supply from the neck.

The recurrent laryngeal nerve should be identified before delivery of the retrosternal goiter by traction and finger mobilization. This is because the recurrent laryngeal nerve is vulnerable to damage by traction and finger mobilization. If a large multinodular retrosternal goiter cant be delivered intact it can be broken by finger and removed peacemeal but this should never be done if the lesion is solitary.

TOXIC GOITER (thyroitoxicosis/ hyperthyroidism)

The term thyroitoxicosis is retained as much as possible because hyperthyroidism is not responsible for the manifestation of the disease.

Clinical types of thyroitoxicosis;

Diffuse toxic goiter (Grave's disease)

Toxic nodular goiter

Toxic nodule

Hyperthyroidism due to rarer causes

Diffuse toxic goiter:

This is a diffuse vascular goiter appearing at the same time as the hyperthyroidism usually in the younger women and frequently associated with eye signs. The syndrome is that od primary thyroitoxucosis. The whole of the functioning thyroid tissue is involved and the hypertrophy and hyperplasia are due to abnormal stimulatingantibodies.

Toxic nodular goiter

A simple nodular goiter is present for a long time before the hyperthyroidism usually in the middle aged or elderly and very infrequently associated with severe eye signs. The syndrome is that of secondary hyperthyroitoxicosis. In many cases the nodules are inactive and it is the internodular tissues that are super active. Basically toxic nodular goiter is graves' disease in a background nodular goiter. In some cases we may haveone or all nodules being hyperactive. In this case hyperthyroidism is due to autonomous thyroid tissue.

Toxic nodule

It is a solitary overactive nodule. It is autonomous and its hypertrophy and hyperplasia are not due to T.S.Ab. because T.S.Ab secretion is suppressed by the increased level of the circulating thyroid hormones the normal thyroid tissue surrounding that nodule is itself suppressed and is inactive.

Histology: the normal thyroid gland institute of acini lined by flattened cuboidal epithelium and looks uniform all through. In hyperthyroidism there is hyperplasia of the acini which are lined by high columnar epithelium. Many of the acini are empty and others contain vacuolated colloid.

Symptomatology of thyroitoxicosis:

Age incidence: it may occur at any age

Sex incidence: thyroitoxicosis is 8 times common in females than males, i.e. F:M =8:1

WAYNE'S CLINICAL DIAGNOSTIC INDEX- It gives all the important symptoms and signs of thyroitoxicosis and indicates by their score the relative importance of each symptom and sign.

Most important significant symptoms,

Wt loss inspite of good appetite

A recent preference for cold or heat intolerance

Palpitations.

Important significant signs,

Excitability of the pt – pt appears agitated and is unable to sit still

The presence of goiter

Exophthalmos

Tarchycardia or cardiac arrhythmias the resting or sleeping pulse is elevated above 80 beats per a minute and this is diagnostic. Water hammer pulse is felt. There is an initial high upthrust and then quickly falls away. There may be a rise in systolic pressure and extra systole may occur.

Wayne's clinical diagnostic index

Symptoms of recent	present		signs		
Onset and /or increased					
Severity	score	score		present	absent
Dyspnea on exertion	+1	-	1. palpable gland	+3	-3
Palpitations	+2	-	2. Bruit of gland	+2	-2
Tiredness	+2	-	3. Exophthalmos	+2	
Preference for heat	-	-5	4. Lid retraction	+2	
Regarless of duration					
Preference for cold	+5	-	5. Hyperkinetic movement	+4	-2
Indifferent to temp.	0	-	6. Lid lag	+1	
Excessive sweating	+3	-	7. Fine finger tremors	+1	

Nervousness	+2		8. Hands hot	+2	-2
Increased appetite	+3		9. Hands moist	+1	-1
Decreased appetite		-3	10. Casual pulse rate		
			Atrial fibrillation		+4
Increased wt.		-3	11. Regular rates		
80/min.	-3				
80-90/min.	0				
90/min.	+3				
Decreased wt		-3.			

key

key: symptoms score + sign score =diagnostic index

if the diagnostic index is under 11 then it is non toxic goiter

11-19 it is suspicious

Over 19 it is toxic

Goiter in 1 degree thyroitoxicoxis is diffused and vasculem may be large or small.

Firm or soft

Athrill of bruit may be present usually at the upper pole of the gland over the superior thyroid arteries.

The oriset is aborpt but remission and exacerbation are common

Hyperthyroidism is severe than 2 degrees thyroitoxicesis but in 1 degree thyroidoxicosis cardiac failure is rare.

OTHER MANIFESTATIONS NOT DUE TO HYPERTHYROIDMS PER SE

Orbital proptosis

Opllithslmophgica

Pre-tibial myxoedema inslightly skin over the chins and feet

In 2 degree thyrotoxicosis the goiter:

Nodular

Onset is insidious

Pt may present with cardiac failure or it is characteristic that hyperthyroidism is not seen severe arterial fibrillation

Eye signs apart from lid lag and lid spasms are very rare.

CARDIAC RHYTHM

A fast heart rate, which persist during sleep is characteristic of thyroitoxicosis. As the disease progresses cardiac arrhythmias are super imposed to the tarchycardia and this arrhythmias are common in older patients' with thyroitoxicosis because of preference of incidental heart disease.

There are 4 stages of development of thyroitoxicosis arrhythmias:

First multiple extrasystole

Paroxysmal arterial tarchycardia

Paroxysmal arterial fibrillation.

Persistence arterial fibrillation not responding to digoxin

MYOPATHY

Weaknesses of proximal limb muscles are commonly found to be weak if looked for. Occasionally severe weaken resembling myasthenia gravis a disorder characterized by muscle weakness. When this occurs in the thyroitoxicosis it is called thyroitoxicostic myopathy. The patient recovers from myopathy when hyperthyroidism is controlled.

EXOPHTHALMOS

Some degree of exophthalmos is common. It may be unilateral. True exophthalmos is a proptosis of the eye caused by infiltration of the retrobulbar tissues filled with fat and round cells which a varying degree of retraction spasms of the upper eye lid. These result in widening of the eye palpetral fissure so that the sclera can be seen clearly above the main of the eye lids and cornea.Spasm are retraction of the eye lid usually diapers when the hyperthyroidism is controlled but that may be improved by beta adrenergic blocking agents like cuanethidine eye drops.Weakness of the extra occular muscles particular the elevators results in diplopia. In severe cases of exophthalmos, papilloedeme and ulceration of the cornea occurs. When severe and progressive it is known as malignant exophthalmos and the eye may be destroyed. Exophthalmos is usually self limiting and may even regress a littleSleeping in a propped up position and lateral tarsorrhaphy (suturing eyelid together) help protect the eye ball but will not prevent development of exophthalmos. Hypothyroidism increase the exophthalmos by a few millimeters and

this should be avoided. Improvement has been reported with massive doses of predinson. Intraorbital injection of steroids is dangerous because of the venous congestion. When the eyes are in danger orbital decompression may be required.

Pre-tibial myxoedema:

This is the thickening of the skin over the shin and feet by amucin like deposit nearly always associated with true exophthalmos. Past or present hyperthyroidism and high levels of T.S.Abs, is usually symmetrical and minor degrees are common but they are easily missed. The earliest stage is a shiny red dark skin with coarse have. The skin may be cyanotic when exposed to cold. In severe cases the skin of the whole leg below the knee involved together with the skin of the foot and ankle and there may be clubbing of fingers and toes.

Diagnosis of thyroitoxicosis:

Most cases are diagnosed clinically and the wayne diagnostic index is necessary. Difficulty is likely to arise in the differentiation of mild thyroitoxicosis from anxiety when a goiter is present. In this cases where differentiation must be done, diagnostic tests must be done. If there is still doubt after aroutine thyroid profile and an iodine 1, 2, 3 thyroid test then a T.R.H test should be done. Thyroitoxicosis diagnosis is established by estimating the T3 levels. It should be suspected if the clinical picture is suggestive but routine test for thyroid function are within the normal range and this is called T3 thyroitoxicosis. A thyroid scan is essential for the diagnosis of an autonomous toxic nodule. There are four general clinical features thyroitoxicosis;

In children with a growth spurt, behavior problem and myopathy

Tarchycardia or arrhythmias in the elderly

Unexplained diarrhea

Loss of wt.

Principles of treatment of thyroitoxicosis:

Non specific measures

Bed rest

Sedation

In established thyroitoxicosis this measures should be used in conjunction with specific measures.

Specific measures:

Anti-thyroid drugs

Surgery

Radio-iodine

Antithyroid drugs

Those in common use are

Carbimazole (neomarcazole) and

Propylthiouracil

Others include;

Beta adrenergic blockers e.g propranolol

Iodides were once thought to reduce vascularity of the thyroid gland. They should only be used as immediate pre-operative treatment in some days before surgery.

Anti thyroid drugs are to restore the pt to euthyroid state and these are used for a long period in hope that a permanent solution may occur. It should be noted that anti-thyroid drugs cannot cure a toxic nodule. The overactive thyroid tissue is autonomous and even if you bring the pt to euthyroid state recurrence occur when you discontinue the drug.

Advantages of anti-thyroid drugs

No surgery

No use of radio-active material

Disadvantages

Rx is prolonged and failure rate after a course of 11/2 -2yrs is 50%. Long term Rx is unacceptable to many pts. Recently there has been a trend towards the use of short term drugs of about 6/52.

It is impossible to predict which pt is likely to go into remission

Some goiters enlarge and become very vascular during treatment even when thyroxine is given together with drugs. This is probably due to T.S.Abs stimulation during the prolonged course of Rx but is not a direct effect into the blood. Very rarely there is a dangerous a drug reaction which is characterized by; agrnulocytosis, urticaria, anaemia

In agranulocytosis, if the pt develops sore throat, discontinue treatment until the level of wbc count decreases. Initially start with 10mg of carbimazole tds and there is a latent interval of 7-14 days before any clinical improvement is apparent. It is most important to maintain high concentration of the drug through out the 24hrs by spacing the doses by 6-8 hourly administration. When the pt becomes euthyroid use a maintainance dose of 5mg bd or tid for another 12- 28 months. Carbimazol acts by imparing the binding of iodine. It laso has hormone suppressive action on T.S.Abs. it is effective in almost all pts though it takes as long as 4-6 wks to give the effects that have been mentioned.

Give T3 20ug bd- tid or thyroxine 0.1mg od in conjunction with ant-thyroid drugs, there is less danger of producing iatrogenic thyroid insufficiency or an increase in size of the body. Potassium perchlorate 200-400mg qds is sometimes used. It has ability to block iodine transport. It's action is slower than carbimazole. It also produces side effects. Propranolol has recently been used when ordinary anti-thyroid therapy has failed and also in cases of thyroid crisis.

SURGERY

In diffuse toxic goiter and toxic nodular goiter with overactive interlobular tissue surgery cures by reducing the mass of the overactive tissue. A cure is probable if the thyroid tissue is reduced a critical mass. This may result in reductionof T.S.Abs or the circulating T.S.Abs however high it's level it can only produce limited hypertrophy and hyperplasia.

In the autonomous toxic nodule surgery cures by removing the tissue.

Advantages of surgery:

Goiter is removed

Long term drug maintenance is not necessary.

Cure is rapid. Euthyroid atste is achieved quickly.

Cure rate is high if surgery has been accurate.

Disadvantages:

A recurrence of thyroitoxicosis in less than 5% of the cases.

Every operation carries morbidity but with suitable preparation with an experienced surgeon, the morbidity is negligible.

Although postoperative thyroid insufficiency occurs in some 20-30%, this is rarely due to operation itself.

Parathyroid insufficiency occurs in < 0.5% cases.

RADIO-IODINE:

It was first used in treatment in 1922. Iodine destroys the thyroid cells. As in thyroitectomy it reduces the thyroid mass to below the critical level. It inhibits iodine binding. It is of considerable value since it is a faster mode of treatment. The main indication is repeated toxicity after previous surgery. It is more effective in diffuse goiter than nodular goiter.

Advantages:

No surgery is necessary

No prolonged drug therapy

Disadvantages

Isotope facilities must be available

There is a high and progressive incidence of thyroid insufficiency which may reach 75-80% after 10yrs. Incidence increases with live. 75-80% develops hypothyroidism after 10yrs due to sublethal damage to those cells not actually destroyed by the initial treatment and this eventually causes failure of cellular production.

An indefinite follow up is essential because of the real and potential risks such as production of carcinomatous changes most clinicians don't give radioiodine in pt less than 45yrs of age. The dose of radio iodine varies in the size of goiter a suggested dose 160 micro-curies/gm of thyroid tissue. Response is slow but a substantial improvement can be expected after about 8-12 wks. Accurate dosage is difficulty and if after 2 wks there is no clinical improvement further dose is necessary. Two or more doses is necessary in about 20-30%.

The choice of therapeutic agent:

Each case must be considered individually. They must be modified according to facilities availableand personality, intelligency and wishes of the individual pt, her business or family commitment and any other co-existing surgical or medical condition.

How to choose therapeutic treatment in diffuse toxic goiter:

Over 45yrs radio iodine

Under 45yrs (i) for large goiter ,surgery (ii) for small goiter, anti-thyroid drugs. Large goiters are uncomfortable and remission with anti-thyroid drugs is less likely as small goiter.

Toxic nodular goiter

Surgery is the treatment of choice. Toxic nodular goiter does not respond as rapidly as a diffuse toxic goiter does to radio-iodine or anti-thyroid drugs. The goiter itself is often large and uncomfortable and enlarges further if anti-thyroids are given.

GASTROINTESTINAL TRACT (G.I.T)

THE ESOPHAGUS

Surgical anatomy:

This is a fibromuscular tube measuring 25cm long. It occupies the posterior mediastinum and extends from the cricopharyngeal sphincter to the cardia of the stomach. 2cm of this tube lies below the diaphragm. The musculature of the upper one third is mainly striated, giving way to smooth muscle below. It is lined by squamous epithelium and replaced by specialized epithelium at the level of the hiatus similar to gastric mucosa but without oxyntic and peptic cells. The specialized mucosa lines the lower 3cm. Nerve supply (parasympathetic) is mediated by the vagus through an extrinsic and intrinsic plexus. The intrinsic plexus has no

meissner's which is elsewhere throughout the alimentary canal. There are thre physiological constrictions in the tube with distinct lesions at each level as shown below.

Diagram:

Physiology:

The main function is to form part of coordinated mechanism transferring food from the mouth to the stomach. The initial mov ement of food through the oropharynx is induced voluntarily and involves sequential contraction of the respiratory passages and opening of the upper esophageal or cricopharyngeal sphincter. The body of the esophagus then sweeps the food bolus by an involuntary peristaltic wave through a relaxed gastro-esophageal sphincter into the stomach. The cricopharyngeal sphincter is normally closed at rest and serves as a protective mechanism against regurgitation of stomach contents into the respiratory passage. Fail ure of it to relax on swallowing may predispose to development of a pharyngeal pouch (pulsion divericulum). At the lower of the esophagus there is a physiological sphincter, which together with other anatomical mechanisms, prevent gastric contents refluxing. The esophagus is a peristaltic organ, and the sphincter relaxes in advance of the peristaltic wave. Abnormal conditions e.g achalasia or scleroderma show changes in both esophageal peristalsis and sphincter tonus and function.

Dysphagia: difficulty (not pain) in swallowing. There are two types, i.e. oropharyngeal and esophageal. The type of dysphagia is vital. It may be dysphagia for solids or liquids, intermittent or progressive, precise or vague in its appreciation. Pain may be present. Painful dysphagia is usually due to esophagitis.

Regurgitation:

It is important to report the volume, contents, presence of blood or bile and the reaction to litmus. Loss of wt, cachexia and change of voice are also important symptoms.

Investigations of the esophagus:

Radiography- it is the most valuable investigation. A plain film will show an opaque foreign body and the site of its arrest. A barium swallow is vital and will show motility, size, distortion, or presence of S.O.L.

Eso[phagoscopy- it is required to view the inside of the esophagus and the esophagogastric junction, to obtain biopsy, for removal of foreign bodies and to dilate strictures. There are two instruments available;

A rigid esophagoscope

Flexible fibreoptic esophagoscope

Esophagoscopy with a rigid esophagoscope should never be carried out without a preliminary barium swallow. If this rule is broken then sooner or later the esophagus will be perforated during the examination.

PH measurement-these are carried out to measure the presence or absence of re flux with change in posture and also decide whether the pain of which the pt complains is indeed due to acid reflux into the esophagus.

Therapeutic procedures:

Removal of foreign bodies- some objects are removed fairly easily and these cases it is usually necessary to withdraw the esophagoscope at the same as the retrieving forceps which are holding the foreign body. If the foreign body is sharp or jagged and is likely to damage the esophagus during its removal, it is preferableto open the esophagus above the fb through a high thoracotomy.

Dilatation of stricture- benign and malignant strictures may require dilatation with bougies which must be well introduced and without too much force.

DDX OF DYSPHAGIA:

Esophagitis

Pulsion diverticulum

CONGENITALESOPHAGEAL ANOMALIES

Atresia with or without trachea-esophageal fistula

Stenosis- rare

Short esophagus with hiatus hernia- rare. Most cases of short esophagus are a result of a hiatus hernia.

Dysphagia lusoria(compression by an aberrant subclavian artery) i.e. esophageal compression by an abnormal artery usually the subclavian artery.

Congenital atresia of the esophagus:

It is usually associated with a trachea-esophageal fistula. It is seen that 85% of cases it is the lower segment that communicates with the trachea.

Diagram

Clinical features:

The new born baby regurgitates all its first and subsequent feeds.

Saliva pours from the mouth almost continuously. This is a sign of esophageal atresia because it does not occur in any other condition.

Attacks of cough and cyanosis on feeding

It should be suspected in all cases of polyhydramnious(50% of cases of atresia – hydramnious was present)

Clinical confirmation:

Size 10 rubber catheter is introduced into the esophagus through the mouth. If an obstruction is encountered at about 10cm from the lips, the diagnosis is practically atresia.

Radiological confirmation:

Never give barium emulsions in this cases.

Injection of not more than 1ml of dianosil down the catheter will demonstrate the catheter. During the examination the supine position is advised, because in the rare cases of categoriesthe medium is likely to enter the trachea. In all cases the dianosil should be aspirated after the radiograph has been taken.

Gas in the stomach will confirm that the lower end of the esophagus reaches the trachea and that an anastomosis can be carried out.

Treatment:

Urgent surgery

Preoperatively N.P.O., rehydration, antibiotics.

Aspiration pneumonia is nearly always present and antibiotics should be given.

Operation- urgent right sided thoracotomy at the 5th intercostals space.

Complications –pneumonia, leakage from the anastomosis.

FOREIGN BODIES IN THE ESOPHAGUS:

All sorts of swallowed fbs have become arrested in the esophagus. They range from coins, pins, and dentures head the list. All cases should have an urgent-rayexamination including dilute barium or water soluble contrast medium(gastrographine)swallow.

Rigid esophagoscopy is necessary in almost all cases. Position so that it may be grasped by suitable forceps introduced through the esophagoscope. The esophagoscope together with the forceps still grasping the Fb is then gently withdrawn.

ESOPHAGITIS:

It may be acute or chronic.

Acute – may follow burns or scalds. Infection(spreading from the pharynx), or peptic, sometimes from trauma of an indwelling stomach tube. Due to a sliding

Hiatus hernia. Reflux is common in pregnancy but usually resolve after delivery.

Reflux esophagitis:

Reflux of esophageal contents the lower esophagus is the 1st and foremost cause of esophagitis. Other causes include;

Decreased efficacy of esophageal anti-reflux mechanism particularly;

Central nervous depressants

Hypothyroidism

Pregnancy

Systemic sclerosing disorders

Alcohol

Tobacco

Presence of Ng tube

Presence of hiatus hernia

Inadequate or slowed clearance of refluxed material

Delayed gastric emptying and increased gastric volume, contributing to the volume of refluxed material.

Reduction in the reparative capacity of the esophageal mucosa by protracted exposure to gastric juice.

The acid- peptic action of gastric juice is critical to the development of esophageal mucosa injury. Anatomic changes depend on the causative agent and on the duration and severity of the expo sure. Simple hyperemia (redness) may be the only alteration.in uncomplicated reflux esophagitis three histologic features are characteristic;

Presence of inflammatory cells including eosinophils, neutrophils, and excessive numbers of lymphocytes in the epithelial layer.

Basal zone hyperplasia exceeding 20% of the epithelial thickness

Elongation of lamina propria papillae with congestion, extending into the top 3rd of the epithelial layer

Clinical features:

Although largely limited to adults > 40yrs, reflux esophagitis is occasionally seen infants and children. Clinical features include;

Dysphagia

Heartburn

Sometimes regurgitation of sour brush

Hematemesis or

Malaena

The potential consequences of severe reflux esophagitis are bleeding, development of stricture, development of Barret's esophagus.

BARRETT ESOPHAGUS:

This is a complication of longstanding with reflux and not in others.ing gastroesophageal reflux. It occurs over time in up to 10% of pts with symptomatic reflux disease. The distal squamous mucosa is replaced by metaplastic columnar epithelium as a result of prolonged injury. The pts tend to have a history of heartburn and other reflux symptoms. They appear to have more massive reflux with more and longer reflux episodes than most reflux pts. It is not known why the columnar epithelium develops in some pts with reflux and not in others.

The lesion on endoscopy, is seen as a red, velvety mucosa located between the smooth, pale esophageal squamous mucosa and the more lush light brown- pink gastric mucosa. It may exist as tongues or patches (islands) extending up from the gastroesophageal junction or as a broad circumferential band displacing the squamocolumnar junctionsegment. A small zone of metaplastic mucosa may be present only at the esophageal junction (short segment Barrett mucosa)

Microscopically the esophageal squamous epithelium is replaced by metaplastic columnar epithelium complete with mucosal glands. The metaplastic mucosa may contain only gastric surface and glandular mucus secreting cells, making clinical distinction from hiatal hernia difficult. Diagnosis is more readily made when the columnar mucosa contains intestinal goblet cells.

Clinical features

Symptoms of reflux esophagitis

Local bleeding due to ulceration

Stricture formation

Development of adenocarcinoma

INFECTIOUS AND CHEMICAL ESOPHAGITIS:

Other thangstroesophageal reflux (which is, a chemical injury), esophageal inflammation may have many origins as follows;

Ingestion of mucosal irritants e.g. alcohol, corrosive acids or alkalis(in suicide attempts) and excessively hot fluids (e.g. hot tea in Iran) as well as heavy smoking

Cytotoxic anticancer therapy, with or without superimposed infecton.

Infection after bacteriamia or viraemia. Herpes simplex virus and cytomegalovirus are the more common offenders in the immunosuppressed.

Fungal infection in the immunosuppressed or debilitated pts or during broad spectrum antimicrobial therapy. Candidiasis is by far the most common.

Uremia in renal failure

It may also occur after radiotherapy.

Morphology:

Infectious and chemical causesof esophagitis exhibit their own characteristic features but finally there is severe acute inflammation, superficial necrosis and ulceration with the formation of granulation tissue and eventual fibrosis.

Candidiasis: Patches or all of esophagus become covered by adeherent, gray white pseudomembranes teeming with densely matted fungal hyphae.

Herpes and cytomegalovirus:

Cause punched out ulcers of the esopheal mucosa.

Pathogenic bacteria: account for 10-15% of cases of infective esophagitis. They invade lamina propriawith necrosis of the squamous epithelium.

Chemically induced injury: (lye, acids, detergents). May produce only mild erythema and edema, sloughing of the mucosa, or outright necrosis of the esophageal ulceration may result from pharmaceutical tablets or capsules sticking in the esophagus

After irradiation: submucosal and mural blood vessels exhibit marked intimal proliferation with luminal narrowing. The submucosa becomes severely fibrotic and the mucosa atrophies.

ESOPHAGEAL VARICES: regardless of cause, portal hypertension, when sufficiently prolonged or severe, induces the formation of collateral bypass channels wherever the portal and caval systems communicate. The collaterals develop in the region of lower esophagus when portal blood is diverted through the coronary veins, then into the azygous veins, and eventually into the systemic circulation. The increased pressure in the esophageal plexus produce dilated tortuous vessels called varices. Varices develop in 90% of cirrhotic pts and are most often associated with alcoholic cirrhosis. Worldwide, hepatic schistosomiasis is the second most common cause of variceal bleeding.

Morphology: varices appear as tortuous dilated veins lying primarily within the submucos of the distal

CARCINOMA OF THE ESOPHAGUS

Accounts for 5% of all carcinomas.

Mostly occurs over the age of 45 yrs

More common in males than females

Predisposing factors(suggested)

Barrett esophagus

The lower esophagus is lined with columnar epithelium

It is secondary to esophago-gastric reflux (as in hiatus hernia) and ectopic gastric mucosa.

Tobacco – smoking/ chewing.

Heavy alcohol intake

Achalasia

Dietary carcinogens/ ? geographical e.g. molibdinum in mt Kenya region, Zimbambwe, nitrites (nitrosomines by fungi).

Plummer- vinson syndrome (Peterson Kelly)

Hot beverages

Poor dental and oral hygiene

Pathology:

The lesion is usually squamous cell carcinoma

True adenocarcinoma occurs in only 3-5% and arises from the columnar cell lined lower esophagus

Other carcinomas are usually of gastric origin spreading upwards

Macroscopically:

Three types are recognized;

A annular stenosing lesion usually at the cardia

An epitheliomatous ulcer with raised and everted edge

A fungating cauli flower like friable mass

Incidence:

Upper 1/3 = 17%

Mid 1/3 = 50%

Lower i/3 = 33%

Spread:

Direct: this is the main method of spread and most important to the surgeon. It occurs both transversely and longitudinall and erodes the muscular walls to invade the most important structures of the neck and posterior mediastinum. Also affects the the left main bronchus and trachea. It may perforate and cause mediastinitis and rarely causes massive bleeding from the aorta. May also affect the ruccurent laryngeal nerve causing hoarseness of voice.

Lymphatic: submucosal lymphatic permeation may lead to satellite nodules away from the main tumor. Also embolic spread to surrounding lymphnodes occurs. From the cervical esophagus the spread is to the lymphnodes of the supraclavicular triangle. From the thoracic esophagus spread is to the paraesophageal and tracheobronchal lymphnodes up to the subdiaphragmatic lymphnodes. From the abdominal esophagus spread is to the lymphnodes of the lesser curvature of the stomach.

Blood stream: metastasis is to the liver which is fairly common.

TNM STAGING:tumour nodule metastatic staging

T1

tumor < 5cm length

No obstruction

No circumfrential involvement

No extra esophageal spread

T2

Tumor > 5cm

No extra esophageal spread or

Tenis size + obstruction

Tenis size + circumfrential involvement but

No extra esophageal spread

T3

Any tumor + extra esophageal spread.

N0 – regionalnodes not involved

N1 – unilateral regional l'nodes involved

N2 – bilateral regional l'nodes involved

N3 –extensive multiple regional nodes involved

M0 – no distant metastasis

M1 – dista

nt metastasis

Stage I = T1, N0, M0

II = T1, N1, N2, M0

= T2, N0-2, M0

III = T3,any N, M0

= any T, N3, M0

IV = any T, any N, M1

Clinical features:

Usually(not always) occurs over the age of 45yrs

More common in men than females

Dysphagia – often the only symptom. The difficulty is steadily progressive. 40% of the pts report within 3/12. Otherwise the pt often delays and seeks advise when he can only swallow liquids by which time considerable,

Wt loss has occurred

Regurgitation(pseudo-vomiting) the regurgitated material is alkaline mixed with saliva and maybe with streaks of blood.

Anorexia I those tumors involvingthe lower esophagus

Pain is a late manifestation.

Investigations:

Cxr and cervical x- ray after barium swallow

Barium swallow

Esophagoscopy

Bronchoscopy

Ultra sound

Exfoliative cytology

C.t. scanning

Asseses;

Tumor length and width

Lung fields

Liver

Celiac glands

Mediastinal glands

Ct scan of the chest and abdomen evaluates local l'nodes spread and distant spread.

Routine investigations

Full blood count

LFTs liver functional tests

Total proteins

Electrolytes

Renal function test

Many of these pts have a longstanding nutritional deficiency and therefore hemoglobin, plasma proteins and blood chemistry must all be checked and corrected if necessary, especially before surgical treatment.

Treatment:

A gastrostomy should never be carried out in pts with ca. esophagus. Unfortunately about 25% of pts who present late are so ill that no treatment is possible other than short term measures to reduce suffering. The operative problem is to remove the tumor and restore continuity by interposition of stomach jejunum, or colon. Curative treatment should be attempted provided the assessment allows.

Asses operability

Clinical;

Pt fitness- weight, pulmonary reserves, to withstand a major operation.

B .CT evaluation: to rule out evidence of spread of the growth to the supraclavicular gland, trachea-bronchial tree or liver.

Palliative:

Intubation

Bypass

Radiotherapy

Laser treatment

Intravenous feeding

Curative treatment by surgery in 25%

Preparation

Correct anaemia

Fluid and electrolyte imbalance

Nutrition

Resection – provides hope for cure especially indicated in ca. of the lower 1/3(adenocarcinoma).

Postcricoid carcinoma:

It should be treated by R.T. The alternative surgical Rx of pharyngolaryngectomy with gastric transposition (Oug), colon transposition(Besley), or plastic tube insertion(Stuart) is a very major undertaking associated with a high complication rate.

Carcinoma of the upper 1/3 of the esophagus:

Early diagnosis is very rare and when dysphagia occurs there are often malign ant glands in the neck or a recurrent laryngeal nerve paralysis, indicating inoperability. The only treatment is then possible is R.T.

Carcinoma of the middle 1/3:

A growth of the area may become adherent to the aorta, venous azygous or left main bronchus. Surgical treatment usually has bad results (complication rate is very high).

Carcinoma of the lower 1/3:

It may be resected by a partial esophago-gastrectomy through a thoraco-abdominal incision through the 8^{th} rib.

Indication for radiotherapy:

Squamous cell carcinoma- radiosensitive

When the diagnosis is made late with spread

When surgical results are bad

When surgical complication rate is high

Palliative treatment:

The tumor may be inoperable due to;

General condition of the pt.

Presence of metastasis.

Tumor may be unresectable at thoracotomy or laparatomy.

Palliative procedure should be carried out to enable the pt to swallow.

This may be either by;

Surgical bypass

Intubation

Radiotherapy

Laser therapy.

Internal tube through tumor. To allow swallowing saliva and soft food. The three types of tubes used include;

Soultar tube (coiled Germany wire)

Celestine tube (armoured rubber tube with a long tail)

Nottingham (Atkinson tube)- funnel shaped proximally.

Palliative short circuit operation. Done when the tumor is unresectable. Esophagogastrostomy or esophago- jujenostomy is done.

Palliative radiotherapy. Patients are too ill to undergo radical radiotherapy for 4-6 wks but may gain some relief from a shorter course.

Laser treatment. The intra luminal bulk of the tumor can be destroyed by laser therapy to enable pts to swallow liquids and avoid choking on their saliva .

Terminal complications:

Unresected, the growth causes death in one of the following ways;

Progressive catchexia and dehydration

Pneumonia from perforation into some part of the bronchial tree.

Erosion of the aorta

PEPTIC ULCER DISEASE:

The term “peptic ulcer” embraces five types of conditions;

Gastric

Duodenal

Stomal following gastro-jejunostomy

Lower end esophagus following reflux esophagitis

Ectopic gastric mucosa (meckel’s diverticulum).

These occur in the presence of acid and pepsin.

Acute peptic ulcers:

Aetiology;

They are thought to be due to disruption of the gastric mucosal barrier. They occur as multiple erosions and at least half of the pts give a history of ingestion of drugs like A.S.A or tone of the other nonsteroidal anti-inflammatory groups. They classically present with hemorrhage and this is common in older pts with arthritis receiving the above mentioned drugs

Drugs

A.S.A

Steroids

NSAID

Stress

Shock

Curling’s ulcers

Cushing ulcers.

Pathology:

Acute peptic ulcers are usually multiple (in 75% of the cases > 3 of those lesions are present.

They can occur in any part of the stomach.

In the duodenum they are almost confined to the first part

The ulcers are oval/circular in shape and vary in size from 1-2 mm or more in diameter.

They are;

Shallow

Punched out

They don't invade the muscular coat.when healing occurs peptic ulcers are unlikely to leave scars.

Clinical features:

They occur frequently

Short lived attacks of dyspepsia which are not diagnosed and ulcer heals

They are recognized when they cause hematemesis

If on the anterior wall of duodenum perforation occurs.

Treatment:

If possible remove the cause.

Acute peptic ulcers tend to heal rapidly with medical treatment (antacids)

Blood transfusion may be required for hematemesis

Correct dietetic irregularities to prevent recurrence or chronicity.

Complications:

Recurrence

Chronicity

Anemia

Perforation

Chronic gastric ulcers:

CHRONIC GASTRIC ULCERS

Etiology:

It is associated with the normal acidity or hyposecretion

Atrophic gastritis

Usually occurs in a later age group

Constantly associated with smoking especially of cigarettes.

Incidence:

Affects 1-2% of the population

Affects both sexes equally.

Pathology:

Usually larger than aduodenal ulcer

Varies in size but in well established cases it will admit the tip of a finger

The floor of the ulcer is situated in the muscular coat of the stomach.

As it advances the ulcer occupying the posterior wall becomes adherent to and later erodes the pancreas (chronic perforation).

The ulcers situated at the antero-posterior aspect of the stomach can penetrate the liver while a saddle shaped ulcer situated on the lesser curve can, and often does, penetrate both the liver and the pancreas.

As with duodenal ulcer, gastric ulcers tend to occur in the non acid secreting mucosa at the boundary with the body of the stomach. This area is much smaller than the area of chronic gastritis which is the precursor of chronic gastric ulcer.

Microscopic examination:

The microscopic examination of chronic gastric ulcer is similar to that of chronic duodenal ulcer.

NB:

Chronic duodenal ulcers never become carcinomatous

Chronic gastric ulcers may become malignant.

CHRONIC DUODENAL ULCERS:

Etiology:

Most pts have gastric hypersecretion of acid.

Duodenal ulcer pts tend to have a larger than normal parietal cell mass.

Genetic and blood group:-

There is evidence that chronic ulcers occur in families

Persons of group O are 3 times more likely to develop P.U than those with other blood groups. It therefore seems that the ABO genes may modify the size of the parietal cell mass.

Neurogenic theory: stimulation of the vagus results in gastric hypersecretion and hypermotility. Stress and anxiety and may be a cause of duodenal ulcer and if so, may exert their via the vagus.

Accessory causes: inadequade mastication, alcohol, irregular meals, excessive smoking and vitamin deficiency have been blamed. Smoking delays healing and promotes recurrence of a previously healed ulcer.

Endocrine: Emotional effects as well as physical stress are hormonally transmitted to the stomach via the pituitary adrenocorticoid axis. Specific endocrine disorders which may be associated with severe or intractable ulceration include;

Zollinger Ellison syndrome- this is where a non –beta cell tumor secreting "gastrin" occurs in the pancreas.

Multiple adenoma syndrome- This is where adenomas occur in the pituitary, adrenal, pancreatic and parathyroid glands.

Hyperparathyroidism.

Infection: Helicobacter pylori, a spirochaete bacteria which exists in the duodenum deep to the mucosal layer. It has the ability to split urea with the formation of ammonia consequently this leads to rise to PH which causes epithelial cellular damage and ulceration. If the organism is removed by antibiotics, metronidazole, the recurrent ulcer rate is reduced.

INCIDENCE:

D.U is rare before the age of 16 yrs

Becomes frequent as middle age approaches

Male to female ratio is 2:1 due to occupation

D.U is found four times more common than gastric ulcer in patient under the age of 35yrs but after 45 yrs of age it is only one or two times more common.

pathology:

Ulcers, whether gastric or duodenal, tend to occur in alkaline mucosa.

A chronic D.U invades the muscular coats which it tends to penetrate. When a G.U or D.U heals, the site heals and is covered by mucosal scar. Fibrosis, the result of recurrent ulceration, causes deformities, including pyloric stenosis and hourglass contracture of the stomach. The duodenal ulcer is nearly always situated in the first part and sometimes two "kissing" ulcers are present. One on the anterior surface and one on the posterior surface of the 1st 3cm of the duodenum. An anterior ulcer may perforate, while a posterior one carries the risk of hemorrhage by erosion of a large vessel.

MICROSCOPIC EXAMINATION:

There is nearly always greater destruction of the muscular coat than of the mucosa. The base of the ulcer is covered by a thin layer of granulation tissue. The neighboring arteries show evidence of endarteritis obliterans. There are no nerves in the floor of the ulcer but always many in the edge.

Clinical features of chronic gastric ulcer.

NB: It is important to record the pt's history under seven headings:

Patient is usually beyond middle age. Thin because of restriction of diet. They usually appear anemic.

Periodicity: Attacks lasts for several weeks followed by interval of freedom from 2-6 months.

Pain: epigastric, may occur immediately or any time up to 2hrs after food.

Vomiting: Is present in 50% of cases. It reliefs pain and may be self induced

Hematemesis and malaena: at some time 30% of the pts bleed. Ratio of hematemesis to malena is 60: 40

Appetite: good but patient is afraid to eat

Diet: pt learns to avoid fried foods, stews, and curries. Milk, eggs and fried fish are the stapple food.

Weight: They usually lose wt.

On examination: There is frequent deep tenderness in the midline of the epigastrium, a few inches above the umbilicus.

Clinical features of chronic D.U:

Occurs any time during adult life, but commonly between 25-50yrs. It common in men who otherwise appear healthy.

Periodicity: Attacks are precipitated by work or worry or weather. The attacks usually last from 2-6 wks with decreasing intervals of freedom from 1-6 months.

Pain: It is severe and may curl up the patient. Usually occurs 1-2 and ½ hrs after food. It is often relieved by food and pain is usually known as "hunger" pain and classically patient always carries food which he eats at frequent intervals. It is also relieved by alkalis, often awakens the pt at around 2 a.m but usually absent at normal waking hours.

Vomiting: rare unless self induced or stenosis has occurred. Regurgitation of burning fluid or sudden salivation (water brash) + pain deep to the sternum (heart burn) due to reflux esophagitis are common (1:10).

Haematemesis and malaena: ratio is 40:60, but sometimes together are rather more frequent than in G.U.

Appetite: good but tries to avoid solid foods during attacks

Diet: usually don't discriminate until they are advised to avoid fried foods.

Weight: usualy wt gain (become plump).

On examination, pain is localized deep tenderness in the right hypochondrium.

SPECIAL INVESTIGATIONS:

Barium meal: I t is usually conclusive. In the lesser curvature, gastric ulcer will show a niche (a small hollow place) projecting from the usually smooth outline. In D.U it will demonstratean ulcer crater filled with barium, a positive evidence of an active ulcer. Appearance of pyloric stenosis and hourglass appearance.

Blood studies: HB may show evidence of chronic blood loss.

Gastric function studies:

Peak acid output (pentagastrin): increased levels are associated with increased acid secretion.

Insulin test (Hollander): insulin given to patient who has had vagotomy done should show no increase in acid production. Test is valuable postoperatively.

Chew and spit: To stimulate the vagus nerve. An N/G tube is passed and pt given food to chew but does not swallow but spits it into a receiver. The stomach is aspirated and contents analysed for the concentration of acid.

Gastroduodenoscopy:

TREATMENT OF UNCOMPLICATED GU AND DU

Conservative management:

If there is no life threatening complication the initial treatment should be conservative (medical). This therefore .means that there be collaboration between the surgeon and physician. The aim of conservative management is to relief symptoms. Since gastric acid is the main provocative factor, the control of acid secretion is the essential therapy. The introduction of drugs which selectively and specifically block acid secretion has increased the effectiveness of conservative therapy. The drugs don't alter the natural history of the chronic disorder.

Chronic gastric ulcer:

Once the lesion has been confirmed as benign, symptomatic treatment is instituted. Although secretion of acid may be subnormal, H2 receptor blockade with cimitidine or ranitidine will secure healing in more than half the patients in a period of 6 wks. Provocative agents should be avoided including chemical irritants such as aspirin, corticosteroids, NSAIDS, and alcohol. Cigarette smoking should be stopped.

Chronic duodenal ulcer:

Active duodenal ulcer disease is associated with increased gastric acid secretion and the pain of D.U arises from the contact of acid with the lesion.

Therefore the control of acid secretion is the logical treatment. Spontaneous healing of D.U within 6wks is common and the aim of treatment is the abolition of pain during the healing phase.

Antacids are helpful in the immediate relief of symptoms. Magnesium hydroxide mixture (120 ml per day) will neutralize gastric acid but are not acceptable because of the incidence of diarrhea.

H2 receptor antagonists: At the parietal level acid secretion is mediated partly by histamine acting on H2 histamine receptors. Such drugs include cimitidine 800mg nocte and ranitidine 300mg nocte.

Surgical treatment:

Indications for surgical treatment;

Intractable pain or recurrence of pain with frequent loss of work, failure to respond to adequate medical treatment.

Complications- E.g. pyloric stenosis, hourglass deformity, perforation or bleeding.

Ulcers which have lasted more than 5yrs are unlikely to heal.

In gastric ulcers

Billroth (partial gastrectomy), vagotomy

In duodenal ulcers:-

Vagotomy (vagus nerve section) with gastric drainage. Vagotomy reduces hypermotility and hypersecretion of the stomach.

Vagotomy causes;

Reduced gastric secretion

Reduced gastric motility

Gastric stasis

Episodic diarrhoea

Complications of p.u;

Acute complications-

Perforation

Hematemesis and or malaena (hemorrhage)

Intermediate: - Residual abscess

Chronic

Stenosis, (pyloricstenosis, tea pot deformity and hourglass contracture)

Penetration into neighboring viscera, notably the pancreas

Carcinoma (gastric ulcer)

PERFORATED PEPTIC ULCER:

Sex ratio is m:f 8:1

Age. Highest incidence is between 45 and 55 yrs

Perforation is common in the anterior surface of the duodenum. Perforation is less frequent on the anterior surface of the duodenum usually near the lesser curvature or the pyloric ant rum. There is long history of peptic ulceration (80%). But there is no such history in 20 % of the cases, it is a "silent chronc ulcer" that perforates, especially those pts on cortisone treatment. In this case the perforation is sudden in occurrence. The gastric or duodenal contents escape through the perforation into the peritoneal cavity and this leads into peritoneal irritation (peritonism). This is when the pt cries out in agony. The peritoneum reacts to the chemical irritation by secreting peritoneal fluid copiously and this gives relief of pain for a short time. This reaction lasts 3-6 hrs and is followed by diffuse bacterial peritonitis.

Clinical features of perforation;

Massive perforation

During the early stages of peritoneal irritation the pt is;

Pale, anxious and loaths more

Temperature may be subnormal

Pulse may be raised

Abdomen is held still, moving little or not at all with respiration.

Slow perforation:

Pain may less severe and

Generalized with definite tenderness

Guarding and rigidity are equivocal

Bowel sounds persist

Right iliac fossa pain due to some small amount of fluid tracking down the paralytic gutter and may simulate appendicitis. It is important to establish the site of the pain.

Diagnosis:

Plain abdominal x-ray with the patient erect will show a translucent area beneath the right cupora (round part or dome) of the diaphragm in 70% of the cases.

Aspiration of the abdominal cavity will reveal bile stained fluid which is alkaline to the litmus.

Treatment:

NB: don't give morphine to the pt because it causes spasms of the sphincter of Oddi.

This is a surgical emergency

Admit the pt

Resuscitate the pt by

Giving i.v fluids

N.G. tube suctioning

Antibiotics

Prepare for surgery as soon as the general condition of the pt permits by;

Getting an informed consent

Blood for grouping and cross matching

Premedications

Laparatomy in theatre and the perforation is closed with interrupted sutures

In case of G.U biopsy is taken for histopathology

Perotneal toilet (lavage)

Continue with NG tube suctioning

Drainage is left in situ.

Antibiotics

Breathing excercises

CAUSES OF HEMATEMESIS AND MALAENA:

Differentials:

Chronic peptic ulcers-65% of cases

Acute peptic ulcers and

multiple erosions-30%

Esophageal varices

Ca stomach

Mallory Weiss syndrome

Peptic ulcer in Meckel's diverticulum

Purpura

Hemophilia

Pernicious and other anemias

Eller's Danlo's syndrome-5%

Haematemesis due to chronic P.U

Bleeding is slight due to trauma from solid food

Occurs frequently from all chronic P.Us

Bleeding is demonstrated by finding traces of blood during gastric analysis and occult blood in stool

There is increased risk with advancing age due to arteriosclerosis and erosion of an artery in the base of the ulcer

The vessels involved occasionally are splenic or gastroduodenal artery.

Hematemesis due to acute P.U.

May be due to solitary or multiple erosions all over the stomach

Diagnosis is gastroscopy

Should be treated conservatively

All drugs should be withdrawn

I.V cimitidine 300mg bd or QDS if the renal function is normal may control the bleeding and is very useful in stress ulcers.

MALLORY-WEISS SYNDROME:

The patient is usually a male of over 50yrs and has a prolonged vomiting bout often after inhibiting (stopping) alcohol. After vomiting gastric contents, he suddenly starts vomiting blood profusely and persistently and becomes exhausted. As a result of straining and retching a longitudinal tear of the mucosa just below the cardiac occurs and gives rise to the sudden onset of hematemesis.the violent **vomiting is sometimes due to migraine or vertigo (dizziness, fear).**

Diagnosis:

From history

Confirmed by gastroscopy

In over 90 of the cases they respond to;

To secretion

;lood transfusion

Pitressin

10% may require laparatomy and repair of the incision.

General clinical features of haematemesis and malaena:

Initially;

Faintness

Sweating

Pallor

Occasionally pt collapses

Afterwards

Effortless haematemesis

Vomiting coffee ground material or bright red blood

Later

Black tarry stools (malaena) or red clotted blood may be passed per rectum.

Treatment of haematemesis and malaena:

On admission;

Collapsed pt is laid flat with a pillow under the head and foot of bed raised.

Cover him to keep him warm depending on the climate of the day

If evidently restless i.v. morphine 15mgand repeated 4hrly PRN

Plasma expanders are started e.g. haemacel to correct hypovolaemia.

NB: sometimes the pt can be given uncross matched blood if the condition is desperate to save life. The main objective is to prevent irreversible shock due to hypovolaemia which may lead to reduced cardiac output and hence poor peripheral perfusion. Maintain a well charted ; pulse chart,

Urinary output and Bp.

Note that Hb estimation after severe hemorrhage will remain unchanged and may not be helpful at this time. After 3 hrs the Hb then may change and its estimation may be helpful.

Signs of severe haemorrhage:

Cold nose

Increasing pallor

Ncreasing

Pulse rate

Beads of sweat on the fore head

Clammy palms of the hands

Blindness is rare and a serious complication

Factors to be considered on whether to treat the pt conservatively (medical) or surgical:

There should be consultation between the surgeon and the physician.

Response to treatment. If the is under adequate conservative treatment and is not responding then surgery is indicated.

Chronicity of the ulcer-the shorter the history the better the pt's response to conservative management.

Age- 70% of cases that bleed are >45yrs and surgery is increasingly necessary after this age.

Ingestion of drugs- if the pt has been taking drugs which predispose to gastric ulceration like A.S.A, NSAID, or cortisone, then their withdrawal will improve the pt's condition conservative

Condition of the arterial tree- presence of arteriosclerosis suggests evidence of recurrent bleeding.

Conservative management:

Bed rest

Blood transfusion

i.v morphine to allay anxiety

H2 receptor antagonists i.v

Chest physiotherapy to prevent pulmonary complications

Prophylactic antibiotics to prevent pulmonary complications

Light diet

Routine treatment for peptic ulcers

NB: Gynaecomastia has been reported in association with high doses of cimitidine.

Surgical management:

If the gastric ulcer is the source of bleeding then gastrostomy and underrunning of the offending vessel is all that is required. Reason is to limit the scale of operation in an already very sick pt.

Rarely gastrectomy may be required

Vagotomy and wide pyloroplasty may be carried out.

Chronic complications of P.U:

Pyloric stenosis- due to cicastration from D.U/juxtapyloric ulcer.

Also in ca situated at/ near the pylorus

Hourglass stomach occurs exclusively in women

Penetration of pancreas

Malignant change- only occurs in gastric ulcers and is rare in the lesser curvature.

Tea[pot stomach- shortening of the lesser curvature due to cicatrisation around a long standing gastric ulcer

CARCINOMA OF THE STOMACH:

"This is the captain of death in men"

It is more common in men than females

No age is excempt from early adult life to senility.

The highest incidence is between 40-60 yrs of age.

Occurs three times as many males as females (3:1)

Etiology:

The premalignant conditions and risk factors include;

Gastric polyp

Pernicious anemia

Postgastrectomy

Posttruncal vagotomy

Longstanding dyspepsia

Gastric ulcer

Genetic (possible)

Ingested carcinogens

Substances which cause irritative gastritis e.g. spirit

Cigarette smoking

Site:

The most common site for neoplasms is in the pre-pyloric region but when carcinoma follows pewrnicious anemia it is more likely to be fundal and polypoid.

Pathology:

Macroscopically;

There are five macroscopic types that are recognized

Cauliflower like growth with sharp defined edge whose surface is indurated and later ulcerated.

An ulcer with an irregular indurated edge.

Colloid carcinoma

Scirrhous localized or diffuse (leather bottle) with thick wall.

Carcinoma secondary to a chronic ulcer.

Early gastric cancer is classified into;

Type I – protruded tumor which protrudes above gastric mucosa

Type II – Superficial inconspicuous unevenness to the surface

Type III – excavated tumor which is centrally ulcerated and its base may reach the muscularis propria.

Microscopically;

Early gastric ca is restricted to the sub mucosa irrespective of lymph node metastasis

The prognosis is excellent and the 5yr survival rate is 90%

The growth is usually columnar celled but cubical and even squamous cells neoplasms arise near the esophageal orifice.

Spread:

Direct spread to the neighboring structures

Lymphatic spread by both emboli and permeation.

By blood stream

Trans peritoneal implantation- ca cells sometimes pass from the stomach into the peritoneal cavity.

Clinical features:

This disease is difficult to diagnose early because;

It's diverse presentation

Symptoms appear late

Gastric distension- inability to take normal meals, vomiting

Anorexia leading to wt loss

Anemia, tiredness, weakness and pallor.

Persistent pain – no response to treatment and noperiodicity.

Clinical types:

- New dyspepsia:

After 40yrs with vague persistent indigestion

- Obstructive type: ca of the cardia which presents with, fullness, belching, and vomiting (gastric output obstruction)
- Insidious onset: especially in men. He feels tired and weak with, Anorexia, Anaemia, Asthenia (3As).
- Lump: incidental discovery of a lump in the epigastrium with no any other symptom
- Silent: ca of the body of the stomach may be silent but give rise to features in other organs such as ;

Obstructive jaundice due to secondary deposits to the liver

Ascites from carcinomatosis of the peritoneum

Krunkenberg tumors- tumor of the ovaries due to metastasis

Phlebothrombosis of superficial veins e.g. legs – Trosseou's sign, left supraclavicular fossa- Troisier's sign.

Investigations:

Hb- 45% of the cases have anemia

Stool for occult blood- present in 80% of the cases

Radiology- cxr, barium meal

Gastric secretory studies

Gastroscopy

Exfoliative cytology

Treatment:

Depends on the stage at which it has been discovered;

If early gastrectomy

If late palliative

Prognosis: -poor.

ACUTE ABDOMEN DDX:

There are many conditions which can present as acute abdomen. It is with this reason that it is considered wise to carefully consider possible diseases of the throat, chest, abdomen, pelvis, the GUT, the CNS and spine.

It is for this reason that we should visualize the body as a house and compare the seven parts of the house to the appropriate anatomical regions.

Attic (the nasopharynx and throat)

Tonsillitis- Abdominal colic may follow swallowed exudates (tonsil tummy)

Pneumonia and pleurisy- especially at the right sided abdominal pain.

They are associated with an increased respiratory rate and the pain prevents deep inspiration.

The upper storey- (i.e diaphragm to the level of the umbilicus)

Perforate peptic ulcer

Acute cholecystitis

Cyclical vomiting

The ground floor (i.e umbilicus to the level of the pelvis)

Appendicitis

Enterocolitis

Non specific mesenteric lymphadeinitis

Intestinal obstruction

Regional ileitis

Ca of the caecum

Meckel's diverticulum

The basement (i.e. the pelvis)

Salpingitis

Ectopic gestation

Ruptured ovarian follicle

Twisted ovarian cyst

Diverticulum of the caecum

The backyard (the retroperitoneal structures)

Ureteric colic

Acute pyelonephritis

Electrical installation(central nervous system)

Pre-herpetic of the 10^{th} and 11^{th} dorsal nerves

Tabetic crisis

Other spinal conditions

Oil tank (blood)

Abdominal crisis of porphyria

Diabetic abdomen

Acute appendicitis:

This is one of the commonest requiring surgery. It is common in the second and third decade of life. It is rare in young children. Common in males than females.

Etiology:

Luminal obstruction in 80% of the cases. The obstruction is usually due to ;

Faecolith

Strictures and

Exceptionally a foreign body or

Round worm or thread worms

Less common:

Stricture or adhesions or kinking secondary to previous inflammation

Caecal or appendicular tumors

Abuse of purgatives particularly castor oil by pts with “stomach aches” leading to violent peristaltic action which results, favors and often determines, perforation of an inflamed appendix.

Pathology:

The severity of appendicitis lies in the frequency with which the peritoneal cavity is infected from this focus by;

Perforation

Transmigration of bacteria through the appendicular wall.

The omentum attempts to wall off the spread of peritoneal invasion, while violent peristalsis from ingested purgatives tends to spread it. If the inflame d appendix lies freely dangling, the risk of peritonitis is increased and should early perforation occur diffuse peritonitis is inevitable. There are two types of acute appendicitis.

non obstructive acute appendicitis:

An inflammation usually commences in the mucous membrane, les often in the lymph follicles and can terminate in one of the following ways:

Resolution

Ulceration

Suppuration

fibrosis

gangrene

Once the infection reaches the loose submucous tissues it progresses rapidly. The organ becomes rigid , dusky red, and hemorrhage occurs into the mucous membrane. The blood supply to the distal part of the appendix is often affected because at this point the artery is liable to occlusion by inflammation or thrombosis. This may lead to gangrene of the tip. Non obstructive appendicitis may progress slowly to form localized peritonitis. In many cases the infection never progresses beyond the mucous lining (i.e

catarrhal inflammation). Complete healing never occurs. Fibrosis of the tip and shrinking occurs and is usually a classical finding in recurrent appendicitis.

Obstructive acute appendicitis

About two of every three cases of acute appendicitis belong to this group. The obstruction can be:

In the lume- faecolith, foreign body, parasite

In the wall – usually inflammatory but may be due to a Ca of the caecum

Outside the wall- adhesions or kinking

Of these the most common is the faecolith. Fibrosis of the wall from previous attacks of appendicitis can cause appendicitis by causing narrowing the lumen and promoting faecolith impaction. Obstructive appendicitis proceeds more rapidly and more certainly to gangrene or perforation. Usually within 12-18 hrs the appendix distal to the obstruction becomes gangrenous. Perforation occurs most often at the site of an impacted faecolith before protective adhesions have had time to follow. Subphrenic and pelvic abscesses are common later complication if the pt survives the initial peritonitis.

Clinical features:

Age incidence:

Rare before the age of 2yrs

Becomes increasingly common during childhood and adolescence

Maximum incidence is 20-30 yrs but no age is excempt.

General features:

During the first 6hrs there is no change in temperature or pulse rate.

After that there is slight pyrexia of 37.2-37.7degrees centigrade with increased pulse rate of <.> 80-90/min.

In90% of the cases the WBC is greater than 10,000 cells per mm3 (10x10power9)liter.

Specific features:

Abdominal pain: it shifts. Usually at the umbilicus, epigastrium or may be generalized. This is visceral pain and vague, and is due to distension of the appendix. Pain is constant in non obstructive appendicitis but colicky in obstructive.

Upset of gastric function:

Anorexia

Nausea

Infrequent of short duration and stops as soon as the stomach is empty

Complication in majority of cases but occasional diarrhea occurs

Local tenderness at the site of the appendix.

As soon as the pain has shifted, there is localized tenderness at the Mcburney's point or elsewhere as is determined by the site of the appendix. This tenderness may be confined to the pelvis and therefore rectal examination must be done in every case of lower abdominal pain.

Rigidity in the right iliac fosa

As time passes, accurate localization becomes more difficult as muscular rigidity becomes evident in addition to the tenderness. A positive release sign is an indication of an acutely inflamed appendix adjacent to the parietal peritoneum.

Obstructive acute appendicitis:

Clinical features and their frequency occur much more quickly and early diagnosis and treatment are accordingly much more urgent. Onset is abrupt and there may be severe generalized abdominal colic from the start. The temperature can be normal. Vomiting is common, so that the clinical picture mimics acute intestinal obstruction. The diagnosis becomes clear when abdominal x-ray shows that the typical signs of obstruction are absent. If diarrhea is present, gastroenteritis may be suspected , but a pelvic appendix must be remembered. Ultra sound will help in making a diagnosis.

Specific features according to position.

Retrocaecal- Rigidity is absent (silent appendicitis) because the caecum is filled with gas and prevents pressure from examining hand to reach the inflamed appendix.

Acute appendicitis in the aged: Gangrene and perforation occur much more frequently because they have a lax abdominal wall. They also like self medication with laxatives.

Obese pts may harbor a gangrenous appendix with little evidence of its existence. Obesity diminishes or obscures all local signs of appendicitis

The clinical picture may mimic sub acute intestinal obstruction and an enema if given will spread peritonitis more widely.

Acute appendicitis in pregnancy: the appendix shifts to the upper abdomen favoring peritonitis. The nearer to term thereafter the danger even without perforation. After 6 months there is a maternal mortality of 20% (ten times greater than in the first three months). As pregnancy advances the pain becomes higher and more lateral. Acute perforated appendicitis causes abortion or initiates premature labor in 50% of the cases. While in acute non perforated appendicitis reduces the figure to 30%.

Treatment of acute appendicitis:

Appendicectomy- By use of Grid iron incision. An incision made at right angles to a line joining the anterior iliac spine to the umbilicus, its centre being at the Mcburney's point.

Antibiotics

Analgesics

Complications of acute appendicitis:

chronicity

Perforation

Peritonitis

Appendicular abscess

Complications after appendicectomy:

Early:

Ileus

Residual abscess(local, pelvic, paracolic or subphrenic)

Intestinal obstruction from adhesions

Wound sepsis

Faecal fistula

Pyelephlebitis

Postoperative thrombosis and embolism

Actinomycosis

Pulmonary complications like pulmonary collapse or pneumonitis

Late complications:

Intestinal obstruction from adhesions

Incisional hernia

Sterility In females from frozen pelvis

Acute peritonitis

Introduction:

The peritoneum is divided into two parts; the visceral (surrounding the viscera) and parietal (lining the rest of the cavity). The parietal is richly supply with nerves and when irritated, causes severe pain acutely localized to the affected area. The visceral is poorly supplied with nerves and pain arising from there is vague and badly localized. The peritoneal cavity is the largest cavity in the body nearly equal to that of the skin .This serous membrane is composed of flattened polyhedral cells, one layer thick resting upon a thin layer of fibroblastic tissue, the two layers constituting the peritoneum. Beneath the peritoneum, supported by a small amount of areola tissue, lies a network of lymphatic vessels and rich plexuses of capillary blood vsls from which all absorption and exudation must occur. Normally only sufficient peritoneal fluid, which is pale yellow fluid containing lymphocytes and polymorphs is secreted to ensure that more mobile viscera glides easily.

Nearly all types of peritonitis are due to bacterial invasion. To an extent that when the term "peritonitis" is used without qualification, bacterial peritonitis is implied.

Bacteriology:

Bacteria from the alimentary canal usually caused by two or more strains. The commonest are, E. coli, anaerobic and aerobic spores, the bacteroides.

Less frequently, cl. Welchi, staphylococci, klebsiella pneumonia

Many of the strains of E. coli, bacterpoides, and Cl. Welchi produce toxins which cause severe illness or death when they invade a large absorptive area (endotoxic shock).

Bacteroides:

Are gram-ve non sporing

Predominant in the lower intestine

Are slow to grow on culture media unless there is adequate Co2 tension in the anaerobic apparatus.

They are resistant to penicillin and streptomycin

They are sensitive to metronidazole, clindamycin and lincomycin.

Bacteria not from the alimentary canal:

Examples include;

Gonococcus

Beta hemolytic streptococci

Pneumococci

Mycobacterium tuberculosis

In young girls and women, pelvic infection via the fallopian tubes is responsible for high level of non alimentary infections e.g. gonococcus and streptococcus, but bacteroides is also found normally in the female genital tract.

Routes of infection:

Direct infection

Via perforation of the gastrointestinal canal

Through penetrating wounds of the abdominal wall

Operative e.g. drains, dialysis tubes, foreign material

Blood stream- part of general septicaemia

Even an initial sterile peritonitis (e.g. intraperitoneal rupture of the bladder or hemoperitoneum) soon becomes infected by transmigration of organisms from the bowel.

Natural factors which tend to cause diffusion of peritonitis:

Perforation of an inflamed appendix or other hollow viscus early before protective mechanisms have mobilized, there is a free gush of intestinal contents into the peritoneal cavity which spreads over a large area almost instantly.

Ingestion of food or water which stimulates peristalsis which hinders localization. Violent peristalsis occasioned by administration of purgatives or an enema promotes a widespread distribution of an infection that would otherwise have remained localized.

When the virulence of the offending organism is so great as to render the localization of the infection difficult or imposible.

In children the omentum is short

Rough handling of localized collection e.g. appendix mass or pericolic abscess.

Immune deficiency – may be due to drugs (e.g. steroids), disease (e.g. AIDS) or infancy or old age.

Clinical features of peritonitis:

Localized features;

If localized the initial feature s are those of the causative lesion

Fever

Increased pulse rate

Abdominal pain and associated vomiting

Guarding and rigidity of the abdominal wall over the area of the abdomen which is involved, with a positive release sign.

If inflammation arises under the diaphragm, shoulder tip (phrenic) pain may be felt.

DIFFUSE (GENERALIZED) PERITONITIS

Initial phase:

Severe pain made worse by moving or breathing

Vomiting may occur

Pt lies still

Tenderness and rigidity on palpation if anterior abdominal wall

Tenderness and rigidity are diminished or absent if the anterior abdominal wall is not affected, e.g. pelvic peritonitis

In pelvic peritonitis the pt may complain of urinary symptoms like tenderness on rectal or vaginal examination

Pulse rises progressively

Infrequent bowel sounds which cease with the onset of paralytic ileus

Intermediate phase

May dissolve so that;

Pain and tenderness diminishes leaving a silent and soft abdomen

The pulse slows

The condition may localize producing one or more abscesses with overlying swelling and tenderness.

Terminal phase:

If there is no resolution or localization the abdomen remains silent and increasingly distends

Circulatory failure ensues

cold and clammy extremities

Sunken eyes

Dry tongue

Thread and irregular pulse

Withdrawn and anxious

The pt finally lapses into unconsciousness

Diagnosis:

The most important is careful history and physical examination i.e.

Tenderness,

Guarding and rigidity,

A distending and silent abdomen

Other investigations may give doubtful diagnosis and even confuse it .

Full hemogram may show leucocytosis

Peritoneal diagnostic aspiration from each quadrant of the abdomen

Bile stained fluid indicates perforated P.U

Presence of pus indicates bacterial infection (peritonitis)

Blood indicates intraperitoneal bleeding

If aspiration fails, a small amount of physiological saline is introduced, followed with few minutes, by a repeat aspiration which may produce fluid of diagnostic value. Abdominal X-ray may show free air or dilated gas filled loops of bowel with multiple fluid levels.

Treatment:

General care of the pt;

I.v fluids- pts are usually hypovolaemic and with electrolyte imbalance. Plasma proteins are also depleted through the inflamed peritoneum.

Ng tube to aspirate the stomach contents until the paralytic ileus is restored and abdomen is soft and not tender and bowel sounds have returned.

Antibiotics-to prevent multiplication of bacteria and the release of endotoxins. Combine parenteral Ampicillin, gentamycin and metronidazole.

A fluid balance chart must be started and maintained

Analgesics – Nurse pt on a sitting position (morphine in small doses)

Physiotherapy to prevent D.V.T and pulmonary embolism.

Neutralization of local cause:

If the cause is treatable by surgery, operation must be carried out as soon as the pt is fit for anaesthesia. This should be within a few hrs. this applies to conditions like perforated appendicitis, diverticulitis, salpingitis, or in cases of primary peritonitis of streptococcal or pneumococcal origin, conservative treatment is the procedure of choice(you should be certain with the diagnosis.

Peritoneal lavage:

After dealing with the cause by surgery, the peritoneal cavity should be explored by a sucker and mopped dry until the purulent exudates is removed. Large volumes of saline are effective in this respect.

Prognosis:

The advent of modern therapy has reduced mortality due to diffuse peritonitis to 10%. Lethal factors include;

Bacterial toxaemia

Paralytic ileus

Bronchopneumonia

Electrolyte imbalance

Renal failure

Undrained collections

Bone marrow suppression

Multisystem breakdown.

Complications:

All complications of severe bacterial infection are possible, but the special complications of peritonitis are as follows;

Acute intestinal obstruction due to adhesions

Paralytic ileus

Residual abscesses, subphrenic, appendicular, pelvic.

GALLSTONES (CHOLELITHIASIS):

They are the commonest billiary pathology.

Classification:

They are classified according to their chemical composition.

a) Cholesterol stones;
- They comprise of 6% of all gallstones
- Consist almost entirely of cholesterol
- Are often solitary

b) Mixed stones;
- Account for 90% of all gall stones in the western world
- Cholesterol is the major component
- Other components include,
  - Calcium bilirubinate
  - Calcium phosphate
  - Calcium carbonate
  - Calcium palmitate and proteins
- Usually they are multiple and faceted

c) Pigment stones;
- Common in the far east
- Composed almost entirely of calcium bilirubinate
- Usually small, black and multiple
- Some are hard and coral like while others are soft.

Incidence of gall stones:

- A Fat, Fertile, Flatulent,Female, of Fifty is a classical sufferer of symptomatic gallstones.
- However;
  - It occurs in both sexes
  - It is often in much early age and even in childhood.
  - Is more common old age.
- Stones are rarer in Africa and South India.

Causal factors:

Metabolic – Cholesterol is insoluble in water but bile salts render it into solution. When cholesterol is in excess in relation to bile acids and phospholipids it allows cholesterol crystals to form. Such cholesterol is termed as "supersaturated"or lithogenic. Cholesterol (bile) increases with age and raised in women particularly those on oral contraceptives and the obese.

Infection: - The role of infection is unclear because bile from pts with gallstone s is sterile. However organisms have been cultured from the center of gallstones.

Bile stasis: - The contraction of the gall bladder is reduced by the following factors which in turn cause stasis of the bile; estrogen, pregnancy and truncal vagotomy.

Pigment stones are seen in pts with haemolysis in which bilirubin production is increased e.g. hereditary spherocytosis, SCD, thalasaemia, malaria mechanical destruction of RBCs by prosthetic heart valves.

Gallstones in relation to other disorders:

Gallstones, diverticulitis, hiatus hernia frequently coexist (SAINT'S TRIAD). It is therefore important to find out which lesion is the cause of the pt's dyspeptic symptom.

Complications of gallstones:

1. In the gall bladder
- Silent stones
- Chronic cholecystitis
- Acute cholecystitis – gangrene, perforation, empyema
- Mucocele
- Carcinoma
2. In the bile duct

Obstructive jaunduice

Cholangitis

Acute pancreatitis

In the intestine:

Acute intestinal obstruction (gallstone ileus)

CHOLECYSTITIS

The gall bladder and the bile duct:

Surgical anatomy:

The gall bladder is pear shaped

Measures 7.5-12.5 cm long

Normal capacity is about 50ml, but is capable of considerable distension in certain pathological conditions

Its wall is made of muscle fibers arranged in criss-cross manner especially in the neck. Its mucous membrane contains indentations of the mucosa (crypts of Lushka) that stick into the muscle coat.

The cystic duct: is about 2.5 cm long and contains the spiral valve of Heister

The common hepatic duct is usually less than 2.5 cm long and is made of a combination of left and right hepatic ducts.

The common bile duct is about 7.5 cm long and is made of the cystic and common hepatic ducts.

Surgical physiology:

As bile leaves the liver it is composed of;

- 97% water
- 1-2% bile salts
- 1% pigments, cholesterol and fatty acids.

The liver secretes bile at a rate of 40ml per hr.

Functions of the gall bladder:

1. Reservoir for bile

During fasting, resistance to flow through the sphincter is high and bile excreted by the liver is diverted to the gall bladder.

After ,feeding, the resistance to flow through the sphincter of Oddiis reduced, the gall bladder contracts and bile enters the duodenum. These motor responses of the billiary tract are in part affected by the hormone cholecystokinin produced by the upper intestinal mucosa in response to foodparticulary fats.

2. Concentration of bile:

By active absorption of water, sodium, chloride and bicarbonate by the mucus membrane of the gall bladder. The hepatic bile which enters thgall bladder becomes concentrated 5-10 times with a corresponding increase in the proportion of the bile salts, bile pigments, cholesterol, and calcium it contains.

3. Secretion of mucin about 20 ml per a day.

CHOLECYSTITIS ACUTE CALCULOUS:

The gall bladder already affected by chronic cholecystitis is now acutely inflamed. In 90% of all the cases a stone is found impacted in the Hartman's pouch or obstructing the cystic duct. In most cases bacteria can be cultured from the bile or bladder wall. The common organisms include;

E. coli

Klebsiella

Streptococcus feacalis

Strict anerobes e.g. bacteroides are rare.

Gas forming organisms e.g. clostridia are rare

Salmonella are also rare.

Outcome:

Mucocele- the bladder is distended and mucous membrane is lifted away from the sides of the stone . The stone then slips back into the body of the gall bladder leaving mucoid or mucopurulent cyst at the duct.

Empyema- alsi known as a pyocele and less common

Perforation which may itself lead to local abscess or generalized peritonitis.

Clinical features:

Sudden onset

Pain at the hypochondrium

Severe nausea and vomiting

A mass may be palpated if the pt is able to relax

Pyrexia of up to 38 degrees celcius

Tenderness and rigidity at the right hypochondrium

BOAS'S SIGN – An area of hyperparaesthesia between the 9^{th} and 11^{th} ribs posteriorly on the right side.

DIFFERENTIAL DIAGNOSIS:

Appendicitis

Perforated p.u

Acute pancreatitis

Right acute pyelonephritis

Myocardial infarction

Right lower lobar pneumonia.

Treatment:

Conservative followed by cholecystectomy. 90% of the cases subside with conservative measures. non operation is based on four principles;

Ng tube aspiration and i.v fluids

Analgesics

Broad spectrum antibiotics

Subsequent management.

By the 3rd day when signs and symptoms have subsided the Ng tube is removed, fluids given orally and a fat free diet started. Then cholecystectomy performed after the acute episode has resolved.

NB: conservative treatment is not advised when there is uncertainty about diagnosis. Conservative treatment must be abandoned if the pain and tenderness spread across the abdomen and pulse.

Routine early operation- Occasionally early surgery is advocated by some surgeons in acute cholecystitis. Usually done within 48hrs.

Other causes of acalculous cholecystitis:

Cholesterosis (straw berry gall bladder)-the interior looks like a straw berry, it has yellow specks due to accumulation of cholesterol.

Polyposis

Ademyomatosis

Cholecystitis glandularis proliferans

Typhoid fever.

CHRONIC CALCULOUS CHOLECYSTITIS:

The bladder wall is fibrotic and thickened. Bacteria is cultured from the bile in less than 30% of the cases. Chronic acalculous cholecystitis can be;

a) Asymptomatic: - An only incidentally discovered on laparatomy or autopsy
b) Symptomatic: - The symptoms are due to either , inflammation of the gall bladderwall or obstruction of the outlet of the gall bladder.

Symptoms:

- Rt hypochondrial pain which is episodic with varying severity. It may be a mere discomfort.
- Flatulent dyspepsia. This is a feeling of fullness after food associated with belching and heart burn.

Diagnosis:

1. Usually ultrasonography is the only investigation

2. Abdominal x-ray following oral cystography may reveal the stones. You may give opiates though they cause Oddi spasms but counter themwith hyosine butylbromide.

Treatment:

1. Analgesica for biliary pain
2. Gall bladder should be removed provided the pt is fit. cholecystectomy is rarely indicated.
3. Dissolution of gallstones- the bile acids, chenodeoxycholic and ursodeoxycholic acid taken orally will dissolve the stones as long as; they are radioluscent and the gall bladder is not non functioning. This treatment however causes diarrhea in half of the pts and has to continue for 6/12. Not all the stones will disappear and may reccur on cessation of treatment

PANCREASE:

Surgical anatomy:

Pancreas is a Greek word meaning (pan- all,kreas –flesh)

Initially thought to act as a cushion for the stomach.

It weighs 80gm

It is retroperitoneal

It is comprised of a head and neck (comprises of 46% of the whole organ) moves up and down with respiration

Moves forward and backward with the aortic pulse

Head lies within the curve of the duodenum

It has the superior mesenteric vsls as a posterior relation

The pancreatic acinar tissue is organized into lobules

The main duct ramifies into interlobular and intralobular ducts, ductules and finally acini

The acinar cells are clamped around a central lumen to form an acinus which communicates with the duct system.

Acinar cells form 84% of the pancreas

Duct cells and bld vsls 4%.

Endocrine cells (Islets of Langerhans)

The rest is connective tissue and fat.

PANCREATITIS:

It is classified on two ways:

According to clinical presentation

According to etiology.

Classification according to presentation;

Acute pancreatitis- returns to normal when primary cause is removed

Relapsing acute pancreatitis- returns to normal when the primary cause is removed.

Chronic pancreatitis- functional or structure damage still remains even if the cause is removed.

Classification according to etiology;

Etiology is very vital in relating the natural history of the disease to prognosis, long term treatment and prognosis.

Billiary tract disease

Alcoholism

Post operative

Traumatic

Rare causes (mumps, hyperthyroidism, vascular disease)

Idiopathic.

However the most agreed is the division of pancreatitis into acute and chronic.

ACUTE PANCREATITIS:

Incidence:

About 5 in every 10000 per yr in the UK

-same as in men and women

In men the peak age is 30-40yrs

In women the peak age is 50yrs

There is a varying degree of edema, hemorrhage and necrosis of the pancreas and surrounding fat.

Causes:

Billiary calculi 50%

alcoholism 25%

others- after biliary or gastric surgery 25%

after trama e.g. blow to the pancreas

when there is distortion of Ampula of Vater due to P.U or carcinoma

as a result of generalized disorders e.g. hypocalcaemia

hyperlipidaemia, D.M and pophyria

Rxn to some drugs e.g corticosteroids

Viral infections like mumps

Some autoimmune conditions like polyarthritis Nodosa

Impaired blood flow e.g. after cardiopulmonary bypass.

The main cause of damage is autodigestion of the gland by its own enzymes. If due to calculi, the stone is passed through the ampulla of Vater distending or splinting the sphincter. The duodenal pressure rises with spasms or contractions and duodenal contents reflux into the pancreas triggering the proteolytic enzymes and the inflammation. High level of alcohol consumption alters metabolism in the acinar cells and alters the composition of pancreatic juice forming protein plugs within pancreatic ducts causing alcoholic pancreatitis.

Clinical feature:

d) Epigastric pain- frequently severe and radiates to LT and RT, through the back.
e) Anorexia
f) Nausea and vomiting
g) Abdominal guarding
h) Bowel sounds may be reduced or absent
i) Fever in 60% of the pts
j) Tarchyopnoea in 50% of the patients
k) Jaundice in 10% of pts
l) Retroperitoneal hemorrhage may be occurring in 5% of the pts producing a bluish ecchymotic discoloration of the flanks (Grey Turners syndrome) and periiumbilical area (cullens syndrome)

Investigations:

Blood serum for amylase is elevated to above 100 somogyl units

Plain abdominal x-ray may show;

An air containing, slightly dilated loop of small bowel over the upper quadrant called sentinel loop.

Mild distension of the transverse colon with collapse of descending colon

Pancreatic ultra sound confirms pancreatic edema and may demonstrate calculi in the gall bladder or bile duct.

Treatment:

Criteria to identify high risk pts;

Old over 50 yrs

Wbc> 16000/cm

Fasting blood sugar>200mg/100ml

Increased LFT (SGOT)

Haematocrit fall>10%

Serum calcium drop< 8mg/100ml

Pao2<60mmhg

Main stay of treatment; bed rest, i.v fluids, ng tube decompression. Pain control by opiates preferably pethidine with an antispasmodic

Attempt to reduce pancreatic secretions with anticholinergics (glucagon, calcitonin, somatostatin, vasopressin, acetazolamide and isoprenoline have been shown to be of no value).

Peritoneal lavage but has got little value

Sphincterotomy if due to calculi

In pts whose condition doesn't improve after 7-14 days, ultrasound and Ct canning should be done and surgery if there is evidence of local complication

Complications:

Shock due to;

G.i.t fluid loss

Retroperitoneal hemorrhage

Electrolyte imbalance

Pulmonary insufficiency(hypoxia) due to;

Retroperitoneal edema

Elevation of the diaphragm

Reduced ventilation due to pain

Rt to left arterial shunting of blood in the lungs

Intravascular coagulation of platelets in the lungs activating phospholipase A with loss of surfactant and increased affinity of oxyhemoglobin for oxygen.

Secondary for edematous pancreatitis

Hypocalcaemia in 3-30% of the cases. Mechanism not fully understood.

Colonic stricture due to scarring of transverse colon

Pseudocyst.

Prognosis:

- Varies with severity- 3-10% for edematous pancreatitis

- 40-50% for necrotizing pancreatitis.

- 100% for completely necrotized pancreas.

Patients who have recovered from pancreatitis due to gallstones should undergo

Cholecystectomy and removal of bile duct stones. This should be done within a month of the attack before another attack develops.

CHRONIC PANCREATITIS:

It is characterized by persistence of pancreatic damage even if the primary cause of pancreatitis has been removed. It occurs more frequently in males than females (M:F 4:1). Mean age of onset is 40yrs. However preference in women seems to be increasing.

Pathology:

The pancreas enlarges and becomes hard due to sclerosis while the ducts become distorted and dilated with areas of ectasis. Calcified stones weighing a few mg to as much as 200mg may form within the ducts. The changes affect a large part of or all of the pancreas and the pancreas may be surrounded by sclerosis which can narrow the arteries, lymphatics, portal and splenic veins, bile ducts and transverse colon. The most frequent cause of chronic pancreatitis is high alcohol consumption accompanied with a

diet rich in proteins and fat. Occasionally, stenosis of the ampulla of vater can result to chronic pancreatitis.

Clinical features:

1. Epigastric pain in 95% of all cases

   Reffered to the left in 29% of the cases and through to the back

   Reffered to the right in 44% of the cases and through to the back

   Reffered to the back in56%

- The pain is worsened by taking alcohol

2. Wt loss due to loss of appetite brought by pain and malabsorption.
3. Gross malabsorption
4. D.m
5. Jaundice in 3% of cases
6. A tender epigastric mass may be palpable - may be due to cyst formation or cancer

   Investigations:

a) Serum amylase
b) A plain abdominal x-ray - may show calcification in 65% of the cases
c) Ultrasound may be useful in the pancreas with cysts and dilated ducts
d) CT scan

   Treatment:

1. Low fat diet
2. No alcohol
3. Pancreatic enzyme supplements even in absence of steatorrhoea
4. Strong analgesics for pain
5. Control DM if present
6. Medical management is often able to sustain life but surgery is occasionally indicated when the disease is not controlled

INTESTINAL OBSTRUCION:

This is a common and serious surgical emergency. It requires early diagnosis and quick relief. It may be classified into two classes;

Dynamic (mechanical) intestinal obstruction;

This is where peristalsis is working against a mechanical obstruction. The obstructing lesion may be;

Intraluminal - like impacted feces, foreign bodies gallstones.

Intramural (in the wall) – like malignancy or inflammatory strictures

Extramural (from outside the wal) like intraperitoneal bands and adhesions, hernias, volvulus or intussusceptions.

Adynamic intestinal obstruction. It may occur in two forms;

Peristalsis may be absent (e.g. paralytic ileus) or

May be present in a non propulsive form (e.g. mesenteric vascular occlusion or pseudo - obstruction). In both types a mechanical element is absent.

DYNAMIC (MECHANICAL) INTESTINAL OBSTRUCTION:

The diagnosis is based on the quartet of pain, distension, vomiting and absolute constipation. Itmay be classified as;

- (i) **Simple**: when there is obstruction to the passage of contents but blood supply is intact.

(ii) When there is obstruction to the passage of contents plus obstruction of blood supply of the involved segment.

Can also be classified according to etiology, thus;

Causes in the lumen - gallstones, food bolus fecal impaction.

Causes in the wall - congenital atresia, neoplasm, inflammatory or malignant strictures

Causes outside the wall - strangulated hernias, adhesions, bands volvulus, intussusceptions.

Can also be classified according to the site i.e.

High small bowel obstruction. The features include;

Early, profuse and frequent vomiting leading to

Rapid dehydration due to increased fluid loss

Oliguria

Dehydration and early collapse

Sunken facial features

Distension +/- in early stages and later limited to

Epigastric region

Feces or flatus may be passed

Shows no fluid levels

Low small bowel obstruction:

The onset is gradual

Severe and colicky pains

Vomiting comes later and less frequent

Distension involves the central abdomen

X-ray shows multiple central fluid levels

Large bowel obstruction- is characterized by early abdominal distension, mild pain. Vomiting and dehydration are late.

Can be classified according to the speed of onset.

Acute- usually involves small gut with sudden severe onset of colicky central abdominal pains, early vomiting, central abdominal distension and constipation.

Chronic obstruction –seen in large bowel obstruction with lower abdominal colicky at first and absolute constipation. Distension comes later and involves the periphery.

Acute on chronic i.o. –it spreads from the large bowel to involve the small intestine giving rise to pain and constipation on a variable time scale, later followed by general distension and vomiting.

Pathology:

The proximal bowel dilates and alters motility.Increased peristalsis continues for about 48hrs to several days. The more distal the point of obstruction the longer it remains vigorous. If obstruction is not relieved, a time reaches when increasing distension causes peristalsis to become feebler and finally ceases and the obstructed intestine becomes flaccid and paralyzed. The intestine below the point of obstruction show normal peristalsis and absorption from it continues for 2-3 hrs following the obstruction, until the residue of its contents has been passed onwards. Then the distal empty intestine become immobile, contracted and pale, and so remains, until the obstruction has been overcome or death ensues.

Distension:

Occurs proximal to the obstrusion and begins immediately after the obstruction occurs.

Two factors account for the distension;

Gas –

Swallowed atmospheric air (68%)

Diffusion from the blood into the lumen (22%)

The product of digestion and bacterial activity (10%)

When oxygen and Co2 has been absorbed into the bloodstream the resultant mixture is made up of nitrogen (90%) and Hydrogen sulphate.

Fluid

It is made of whatever fluid the pt swallows as well as the various digestive juices. It is about 8000 ml per 24 hrs .

Above the pylorus- 4000 ml =saliva 1500ml

= gastric juice 2500ml

Below the pylorus- 4000ml = bile + pancreatic juice 1000ml

= Succus entericus 3000ml

In i.o, absorption from the gut is retarded but excretion of water and electrolytes into the lumen

Persists and may even be increased. Therefore the causes of dehydration and electrolyte imbalance are;

- Vomiting
- Defective intestinal absorption
- Sequestration in the bowel lumen
- Reduced oral intake

Severity and speed of clinical manifestation depends on the level of obstruction.

It is most severe in high intestinal obstruction

Later in ileal obstruction

Slow to appear in colonic obstruction.

APPROXIMATE ELECTROLYTE CONTENTS OF THE GUT:

Fluid	Na+	K+	Cl-(mmol)
Gastric juice	60	10	100
Bile juice	145	5	100

Pancreatic juice	140	5	75
Small bowel	140	5	100

Intestinal toxins:

Death may occur even if the obstruction is relieved especially in strangulated obstruction. In unrelieved strangulation, toxic substances appear in the peritoneal fluid only when the viability of the bowel wall is affected. However when the obstruction is relieved, this toxins may pass on the bowel where the absorption can occur . Most peobably the toxins are endotoxins from gram negative bacilli.

Strangulation of the bowel:

It occurs when the bowel is trapped by a hernia or a band or involved in a volvulus or intussusceptions cause progressive interference to blood supply. This is very dangerous and requires urgent treatment before gangrene sets in. mesenteric vascular occlusion alone gives rise to gangrene without mechanical obstruction.

Onset of gangrene:

The strangulation compresses the veins causing strangulation on the bowel, involving the mes entery which becomes b lue and congested. Severity depends on the tightness of the constricting agent. When the venous return is completely occluded the color of the intestine turns from purple to black. At this time due to increased edema at the point of obstruction, the arterial supply is jeopardized. The peritoneal coat loses it's glistering appearance, the mucous membrane ulcerates and gangrene is imminent. Loss of blood into the congested segment is proportional to the length of the segment.

Distension:

For a considerable time the strangulated segment alone distends, the greatest distension occurring when the venous return is completely obstructed while the arterial supply remains uninterrupted.

CLOSED LOOP OBSTRUCTION:

It occurs when the bowel is obstructed at both distal and proximal points.

Diagram

It presents with late distension. When it starts turning gangrenous distension occurs in both ends of the obstructed segments. If not relieved it results in necrosis and perforation.

Clinical features of acute intestinal obstruction:

Abdominal pain:

It is the first symptom

Commences suddenly without warning

Becomes increasingly severe, then passes off gradually only to return at intervals of a few minutes.

The attacks of intestinal colicky lasts about 3-5 minutes spreading all over the abdomen, but mainly localized at the umbilicus.

The pain is associated with increased peristaltic activity

When distension occurs the pain becomes diffuse and constant.

Distension associated with severe pain indicates strangulation.

Vomiting:

The interval depends on the level of obstruction

As acute obstruction progresses, the character of the vomitus alters. Initially it contains partly yellow or green from regurgitation of bile.

Finally it is faeculent.

Distension :

In early cases of obstruction of the small intestine abdominal distension is often slight, or even absent.

Centrally placed distension is present in fully established cases of obstruction to the ileum.

Visible peristalsis may be present

Borborygmi are sometimes loud enough to be heard by an unaided ear.

External hernia may be present although the pt is entirely unaware of it.

It is important to examine the hernia sites in case of intestinal obstruction. An irreducible external hernia may be present though

Distension depends on site of obstruction. It is greater if theobstruction is low down.

Constipation:

It is classified as absolute (neither faeces nor flatus is passed) or relative (when only flatus is passed). Absolute constipation is a cardinal sign of complete intestinal obstruction. Some pts may pass faeces or flatus after the onset of obstruction owing to evacuation of the distal bowel contents. The rule that constipation is present in i.o does not apply in Ritcher's hernia, gallstones, mesenteric vascular obstruction and i.o associated with pelvic abscess, partial obstruction (faecal impaction, colonic ca) where diarrhea may often occur.

Other features

Dehydration

On due to vomiting and loss of absorptive power by the distended gut

Dry skin

Dry tongue

Sunken eyes

Reduced urine output, concentrated and contains little

Increased blood urea

Increased haematocrit (which may cause a misleading raised hemoglobin figure)

Hypokalaemia may occur but is rare

Pyrexia indicates septic shock

Abdominal tenderness is localized in ischemic areas. May also be due to peritonism or peritonitis if there is infarction or perforation.

Clinical features of strangulation:

All features mentioned above+

There may be presence of shock indicating ischemia

Symptoms are usually sudden and reccur regularly.

Strangulation of external hernia presents with tense, tender, irreducible lump which is non expansile on cough impulse.

Obstruction where pain persists even in the absence of these symptoms indicates strangulation

General tenderness and presence of rigidity requires urgent laparatomy.

Investigations:

X-Ray diagnosis:

This is by far the most important of all investigations.

5% of the pts may have normal x-ray findings

Should be taken with the patient standing and lying down

In case of chronic obstruction, this should be preceded by an enema.

Methods of taking an x-ray:

Plain abdominal- erect (standing) or lying

Barium studies – barium meal and follow through or barium enema

Gas shows:

The diameter of the viscus is no criteria as to whether it is small or large intestine.

Obstructed small intestine is revealed by relatively straight segments that generally lie more or less transversely

Obstructed large intestine is disclosed by its haustration (folds that don't completely traverse the width of the gut). The folds are spaced irregularly and the indentations are not placed opposite one another. A distended caecum is shown by a rounded gas shadow usually in the right inguinal fossa. In total i.o no gas shows in the rectum.

Fluid levels:

Infants < 2yrs a few fluid levels in the small intestine are a normal occurrence.

In adults, two inconstant fluid levels must be regarded physiological. One is at the duodenal cap, while the other is within the terminal ileum. In i.o it takes a little time for gas to separate from the fluid and therefore fluid levels appear later than gas shadow. When paralysis sets in fluid levels become more conspicuous and more numerous. The number of fluid levels is proportional to the degree of obstruction and to its site in the small intestine. The nearer the obstruction is to the ileocaecal valve, the larger the number of fluid levels. Obstruction low in the colon does not commonly give rise to fluid levels in the small gut but in case of obstruction high in the large intestine the phenomenon is not unusual because the ileocaecal valve is incompetent in many individuals. In obstruction of the large gut, a plain X-ray always shows a large amount of gas in the caecum.

Summary of x-ray:

Supine abdominal film

Obstructed small gut is characterized by straight segments

The jejunum is characterized by its vulvae convente which pass across the width of bowel and are regularly spaced giving a ladder picture.

Caecum is distended and shows a round gas shadow.

Large bowel shows haustral folds which are irregularly spaced.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION:

Gastrointestinal drainage:- in every case of i.o the first step is to empty the stomach by a nasogastric tube by withdrawing the contents by continuous suctioning.

Replacement of the fluids and electrolytes – this should be the second step in the management of i.o.

Relief of the obstruction by operation should be done as soon as dehydration and electrolyte imbalance have been corrected. The main indications for early operation are;

Obstructed or strangulated external hernia

Internal intestinal strangulation

Acute on acute on chronic i.o

Supportive:

Continuous Ng tube decompression

i.v.f usually plasma expanders, the volume depending on the biochemical disturbance

broad spectrum antibiotics should be started early. It is a must in a healthy pt undergoing large bowel surgery.

Parenteral analgesics

Treatment of pressure points

Vital signs monitoring

Chest physiotherapy

Daily cleaning and dressing of the wound

Social toilet

Input output chart

Follow up at the S.O.P.C after discharge.

ACUTE INTUSSUSCEPTION:

It occurs when one portion of the gut becomes invaginated into another immediately adjacent. It is almost always the proximal one invaginating into the distal.

Etiology:

Obvious causes in a few cases include;

A polyp

A papilliferous carcinoma

A submucous lipoma

A meckel's diverticulum

In infants it is generally agreed that;

Idiopathic intussusception occurs usually between the 6^{th} and 9^{th} month of life.

Change in diet (weaning period)

Idiopathic intussusceptions usually commences in some part of the last 50cm of the small intestine

The maximum aggregation of the peyer's patches is the lower ileum

NB:-

Mainly found in children 3-9 months

70-95% is idiopathic in nature

It is associated with the following conditions; URTI e.g adenovirus or rotavirus, Diarrhoea, UTI.

Pathology:

It is composed of three parts:

The entering (inner) tube= the proximal segment

The returnin (middle) tube=intussuscipiens

The sheath (outer) tube.=intussusceptum

Intussusceptions is an example of strangulation obstruction as the blood supply of the inner layer is usually impaired. Ischemia follows depending on the tightness of the invagination.

Clinical features:

Usually the pt is a fine child of between 6-9 months.

Onset is sudden.

The child has bouts of abdominal pain, drawing up her legs and screams.

He may vomit after onset of the attack but this is not a constant occurrence but after 24hrs it becomes obvious.

The attack occurs after every 15 minutes accompanied by facial pallor.

Bld plus mucus are passed at late stages (red-currant jelly stools)

Abdomen is not distended

Lt sausagelike lump may be felt at the lt side of the umbilicus

Per rectal blood plus mucus on an examining finger

Diagnosis:

1) Plain abdominal x-ray usually reveals increased gas in the small intestine and sometimes an absence of caecal gas shadow
2) Barium enema may show a claw sign if ileocolic intussusceptions. NB- barium may be therapeutic.

DDX:

1. Acute enterocolitis- has abdominal pain, vomiting, occ.blood and mucus in stool. Diarrhea is the leading symptom.
2. Rectal prolapsed – the projecting mucosa is in continuity with perianal skin while in intussusceptions the examining finger may pass into the sulcus
3. Henoch's Schonlein purpura – has a characteristic rash and abdominal pain.

Treatment:

1. Barium enema may reduce it.
2. Surgery and manual reduction.

VOLVULUS:

Def. It is a twisting or axial rotation of a portion of a bowel about its messentry. When complete it forms a closed loop of obstruction resulting in ischaemia due to vascular occlusion.

Types:

1. Volvulus neonatorum
2. Volvulus of the small intestine. It is usually in the lower ileum and is favoured by the presence of adhesions.

In Africans volvulus involves many feet of small intestine without causative adhesions occurs commonly. Consumption of a large maize meal and vegetables seems to to predispose the condition.

Treatment:

1. Untwist the loop if possible.
2. The causative band must be divided.

VOLVULUS OF THE CAECUM:

Occurs when the Rt half of the colon is lax and mobile.

It is nearly always in clockwise direction.

It occurs between the of 14 to 88yrs.

It is about twice common in females than males.

The first twist obstructs the ascending colon and if the second twist occurs, it obstructs the ileum also.

It usually presents as acute abdominal pain in 90% of the pts.

There is nausea and vomiting in 70% of the cases.

There is constipation in 60% of the pts.

There is apalpable tympanic swelling in 25% of the cases in the Rt iliac fossa but not as a rule.

A plain x-ray shows loops of gas filled ileum and sometimes an especially large gas shadow which can be recognized as the caecum.

Barium enema shows bird neck deformity and there will be no barium in the caecum.

Treatment:

Operation- In early stages untwist the bowel

Before untwisting the ballooned caecum is deflated by insertion of needle

Untwisting should be followed by caecostomy to relief distension

The caecum should be fixed to the wall to prevent reccurence.

If the caecum is gangrenous Rt hemicolectomy is performed.

SIGMOID VOLVULUS:

It is rare but common in Africans and is predisposed by high residue diet and chronic constipation

Clinical features:

Presents with signs of large bowel obstruction

There is early progressive distension

Retching

Hiccups

Late vomiting

Absolute constipation

X-RAY:

Massive colonic distension

Dilated loop running diagonally across the abdomen, Rt to Lt

Fluid levels within the loops

Treatment:

Do sigmoidoscopy or

Pass flatus tube to deflate the gut, if it succeeds arrange for laparatomy with untwisting. If it fails then urgent laparatomy and untwisting is done. The bowel may be fixed to the posterior abdominal wall to avoid reccuring.

ACUTE OBSTRUCTION IN A NEWBORN:

It occurs in 1:2000 births

Causes:

1. Congenital atresia/ stenosis

Incidence

Duodenum 35%

Jejunum 15%

Ileum 25 %

Ascending colon 10%

Multiple sites 15%

a) Duodenal atresia or stenosis

Both stenosis and atresia occur in the same proportion

This is due to an intrauterine accident occurring during pregnancy such as volvulus intussusceptions or strangulation at the umbilical region.

The ischaemic portion is absorbed and disappears because the fetus is germ free. If the obstruction is complete the fetus presents with persistent peristalsis with no distension at the Lt upper quadrant.

Treatment: Surgery after resuscitation.

b) Atresia or stenosis of the jejunum or ileum.

Child is born with abdominal distension and presents within 24hrs of birth (ileal)

c) In jejuna atresia vomiting occurs early

The vomitus contains bile and some meconeum

X-RAY shows air or fluid in late stages

MECONEUM ILEUS:

This is a neonatal manifestation of cystic fibrosis.

The condition is due to an autosomal recessive genetic defect.

Meconeum is usually kept fluid by action of pancreatic enzymes.

The terminal and viscid mucus, results in progressive insipissation in utero and meconeum obstruction.

This may be palpated as a rubbery swelling.

40% of the cases are associated with complications like volvulus, atresia, and meconeum peritonitis.

There is absence of trypsin in stool or bile.

Concentration of sodium in sweat.

TREATMENT:

Radioactive opaque fluid enema(gastrographin) for confirmation or reliefing.

If it fails, surgery is done to resect the dilated segment(with end to end anastomosis.

MEGACOLON:

A relatively uncommon condition which occurs in two varieties;

i) Primary or true mega colon (Hirchsprung's disease or congenital aganglionic mega colon)
ii) Secondary or acquired mega colon

HIRCHSPRUNG'S DISEASE:

It is also known as primary or true megacolon or congenital aganglionic mega colon

Pathology: - it is characterized by dilatation and hypertrophy of the pelvic colon which sometimes extends to the descending colon. It rarely involves the more proximal portions of the large intestine. The pelvic mesocolon is elongated and thickened and its blood vsls are large and prominent. All coats of the dilated intestine show gross pathological changes. The mucosa is chronically inflamed and ulcerated. There is a terminal constricted, non hypertrophied segment of bowel usually involving the anal canal, rectum, and a variable part of large intestine. In 9 out 10 cases the upper limit of the contracted segment is at or below the pelvirectal junction. Occasionally the deficiency extends to a higher level. It is in this contracted segment that physiological obstruction lies, and the dilatation, hypertrophy of the normal colon above is due to absence of peristalsis in the spastic segment. Surprisingly the anal sphincters retain their normal physiological function.

Clinical features:

Affects 1:4500 births

Shows familial tendency

Common in males than females

Symptoms appear within 3 days in 90% of the cases

More frequent in babies with Down's syndrome

i) Constipation:
- Infants fail to pass meconeum during the first 2-3 days and occurs only after insertion of small finger or tube into the rectum
- Subsequently the motions are tooth paste like and inadequade in amount
- Straining is obvious during passage of motions
- Diarrhea also occurs and baby may die.

ii) Distention with borborygmy and visible peristalsis are evident.

iii) On per rectal examination, the anus is free from fissures with no peri anal soiling. The rectum is empty and grips the examining finger

iv) Complete intestinal obstruction occurs quite frequently within a few days of birth and may be fatal. As a rule attacks reccur. Relief is by a small enema, passing a greased examining finger or by spontaneous passage of large stools sometimes followed by diarrhea.

v) Chest infection may be present due to massive abdominal distension.

vi) If the child survives, malnutrition and stunted growth are obvious

Investigations:

X-ray following barium enema shows a typical picture;

Dilated , normal proximal bowel

A narrower segment the transitional zone and

Undilated distal bowel which is aganglionic portion

Treatment:

Excision of the entire aganglionic segment. No reparative operation should be done till the child is 18kg I wt and thriving.

ACQUIRED OR SECONDARY MEGACOLON :

The obstruction is due to suppressed defaecation and faecal impaction. Dilatation and hypertrophy of an otherwise normal large bowel extends to the anal canal.

Characteristically there is;

Anal fissure

A spastic sphincter and

Much peri anal soiling

As a rule faulty bowel care and training are the sources of the trouble, and usually, they can be traced to infancy. The onset however is never from birth. Sometimes this condition is encountered in the insane and the old.

Investigations:

1. Per rectal examination- is usually painful and there is a mass felt just inside the anus which is contrary to the findings in hirchsprung's disease
2. Sigmoidoscopy- chronic insertion of aperients for many yrs to relief constipation may lead to a dark discoloration of the mucus of the colon and rectum (melanosis coli)
3. X-ray – in all cases of megacolon the dilatation is shown by a barium enema ends at the anal canal

Treatment:

Conservative should an anal fissure in ano or stricture be present appproperiate treatment must be done. The essential thing is anal training so that a regular habit is developed. It is wise to start with regular enemas and laxatives.

ADYNAMIC INTESTINAL OBSTRUCTION: (Is commonly known as paralytic ileus)

Definition: A state where the intestine fails to transmit peristaltic waves due to failure in the neuromuscular mechanism i.e the myenteric plexus (Auerbach) and the submucus plexus (meissner). This leads to;

Collection of fluid and gas in the intestine

Diste

nsion

Vomiting

Absent bowel sounds

Absolute constipation (failure to pass flatus and stool)

Varieties of paralytic ileus;

1. Postoperative

- May be local or general following abdominal operation
- Not serious if not accompanied by infection
- Motility and absorption usually return in about 16hrs ahead of gastric and colonic activity
- Paralysis may be prolonged if there is;

- hyponatraemia
- Latent renal failure

Gastrointestinal suction is continued beyond the point at which effective bowel sounds have returned.

2. Infective

Peritonitis gives rise to prolonged ileus but several factors may be involved. At first peristalsis stops as a normal response to spread but afterwards, bacterial toxins prevent the normal activity of the nerve plexuses. When the bowel begins to recover the early weak (feeble) peristaltic waves may not be able to overcome the obstructive effects of the newly formed adhesions between loops. In this kind of ileus, there are therefore mechanical as well as neurogenic factors to be considered. Typhoid is associated with ileus in its acute form.

3. Reflex

This form may occur following fractures of the spine or ribs, retroperitoneal hemorrhage,or even the application of a plaster jacket.

4. Uremia

This type is characterized by distension, vomiting and hiccups. It is se en in renal failure. It may follow prostatectomy.

5. Hypocalaemia. Low serum potassium may cause ileus

Clinical features of ileus

Suspect ileus if after laparatomy;

1. There has been no passage of flatus
2. There is no return of normal bowel sounds on auscultation

Abdominal distension becomes more marked and drumlike (tympanic) if operation has not been done

Effortless vomiting of large dirty fluids

There is no colic nor pain at all

Respiratorydistress from the abdominal distension

Pulse rate may increase

Prolonged distension may lead toburst abdomen(dehiscence)

X-ray shows gas filled loops of intestine with multiple fluid levels

NB: it is important to note the three types of bowel sounds;

1. The normal borborygmy each lasting about one second and occurring every 20 -30 seconds or so
2. The prolonged, rapidly reccuring and noisy borborygmy of dynamic obstruction
3. The high tinkling note " like bells at evening pealing" which occur after every 10-30 seconds and is distinctive of paralytic ileus. It is not due to peristalsis but due to overflow of fluid from one distended loop to another.

Management:

- To prevent paralytic ileus routine NG tube suction and withholding fluid by mouth after laparatomy until return of normal bowel sounds or passage of flatus
- Electrolyte balance should be achieved before and maintained during operation
- Remove primary cause
- Decompress the GIT distension by NG tube(non sphigoted) to allow swallowed air to be evacuated.
- Small doses of morphine or pethidine are valuable in this cases
- Antibiotic prophylaxis is essential
- Gentle handling of bowels intraoperatively
- Sound technique that reduces possibilitry of leakage

NB: never stimulate peristalsis. The objective is to rest the bowel not to stimulate.

RUPTURED SPLEEN:

It should be suspected after any trauma particularly if there had been direct injury to the upper quadrant of the abdomen from any angle. Occasionally a fall without direct trauma to the trunk can rupture the spleen especially if it is diseased or enlarged as in malaria or infectious mononucleosis.

Spleen rupture may be divided into three groups;

1. Pt succumbs rapidly, never recovers from the initial shock:
- This type is rare
- There is tearing of the splenic vsls and complete avulsion of the spleen from its pedicle giving rise to rapid bld loss which can be fatal within minutes.
2. Initial shock; recovery from shock; signs of ruptured spleen:
- The usual type seen in surgical practice
- After initial shock has passed off, there are signs which point to intraabdominal bleeding
- General signs of internal haemorrhage are variable;
  - Increasing pallor
  - A rising pulse
  - Sighing respiration

Local signs;

- abdominal guarding in 50% (more in the Lt quadrant)
- local bruising and tenderness in the Lt abdominal quadrant
- abdominal distension in about 3hrs after the accident
- restlessness Kehr – pain reffered to the Lt shoulder
- shifting dullness in the flunks is often present
- rectal examination frequently reveals tenderness and sometimes a soft swelling, due to bld or clot in the retrovesical pouch.

3. The delayed type of case:

After the initial signs have passed off, the symptoms of serious intraabdominal catastrophe are postponed for a variable period even upto 15/7. This time the pt appears to have recovered from the blow, for example a rugby player, may continue to play after a short rest, only to collapse later from internal haemorrhage. The cause for delayed haemorrhage is local vasoconstriction with or without formation of bld clots which seal the tear. The cause of the haemorrhage are those of reactionary or secondary haemorrhage.

Investigation:

1. Ultrasound examination is the investigation of choice.
2. Abdominal X-ray may show;
- Obliterated splenic outline
- Obliterated psoas shadow
- Indentation of the Lt side of the gastric air bubble
- Fracture of one or more lower ribs on the Lt side in 27% of the cases)
- Free fluid between gas filled intestinal coil.
- Elevation of the left side of the diaphragm.

Treatment:

- Immediate laparatomy and splenectomy is the only step.
- Bld is evacuated
- Abdomen is closed after exclusion of injury to other viscera.

DDX OF SPLENOMEGALLY:

1. Infective

a) Bacterial

- Typhoid and paratyphoid
- Typhus
- Anthrax
- Tuberculosis
- Septicaemia
- Abscess of the spleen

b) Spirochaetal

- Weils' disease
- Syphilis

c) Viral

- Infectious mononucleosis
- Psittacosis

d) Protozoal and parasitic

- Malaria
- Trypanosomiasis
- Schistosomiasis
- Kalar azar
- Hydatid cyst
- Tropical splenomegally

2. Blood diseases

- Myelofibrosis
- Acute leukaemia
- Chronic leukaemia (lymphocytic or granulocytic)
- Pernicious anaemia
- Polycytaemia vera
- Hereditary spherocytosis
- Autoimmune haemolytic anaemia
- Idiopathic thrombocytopaenic purpura
- Thalasaemia (meditteranean anaemia)
- SCD

3. Metabolic

- Rickets
- Amyloid disease
- Porphyria

- Gaucher's disease

4. Circulatory

- Infarct- infective endocarditis, mitral stenosis
- Occlusion of the portal vein- portal Ht, thrombophlebitis, neoplastic like ca head of pancreas.

5. Collagen diseases- still's disease, Fetty's disease

6. Non-parasitic cysts- congenital or acquired

7. neoplastic – angioma, primary fibrosarcoma, Hodgkin's disease and other lymphomas.

HERNIA:

Def. A protrusion of a viscus, or part of a viscus, through an abnormal opening in the wall of its containing cavity.

External abdominal hernia is the commonest form (spontaneous) hernia. Of these hernias, the inguinal hernia is the commonsest (73%)

- Femoral (17%)
- Umbilical (8.5%)
- 15% is left for the rarer form of spontaneous hernias.

Etiology:

Any condition which raises intra abdominal pressure e.g;

- A powerful muscular effort or
- Strain occasioned by lifting heavy wt.
- Whooping cougfh(in children)
- Chronic cough
- Straining on micturition or defaecation
- Intra abdominal malignancy
- Stretching of abdominal musculature because of an increase in contents as in obesity, pregnancy.
- Fat which separates muscle bundles and layers weakens aponeurosis, and favours appearance of
- Para- imbilical, direct inguinal and hiatus hernia.

Composition of a hernia;

As a rule, a hernia consists of three parts; the sac, the covering of the sac, and the contents of the sac.

i) The sac: it is a diverticulum of peritoneum consisting of mouth, neck, body and fundus. The neck is usually defined but not so in some direct inguinal and many incisional hernias. There is no actual neck. The diameter of the neck is important, because strangulation is common where the neck is narrow as in femoral and umbilical hernia

ii) The body of the sac: in cases occurring in infancy and childhood, the wall is thin. In long standing cases, especially after yrs of pressure the wall is thick. The size varies and is not necessarily occupied.

iii) The covering: these are from the layers of the abdominal wall through which the sac passes.

iv) Contents: this can be any abdominal viscus,except the liver, but most commonly are;

- Fluid (peritoneal exudates),
- Omentum (omentocele)
- Intestine (enterocele usually small intestine but occasionally large intestine or the appendix)
- A portion of the circumference of the intestine(Ritcher's hernia)
- A portion of the bladder or diverticulum of the bladder
- A meckel's diverticulum(Littre's hernia)

Classification of hernia irrespective of site;

1. Reducible
2. Irreducible (a complication of reducible hernia)
3. Obstructed (a complication of irreducible hernia)
4. Inflamed (a complication of irreducible hernia)

Reducible hernia;

The hernia reduces itself when the patient lies down or can be reduced by the patient or by another person. The intestine gurgles on reduction and the last portion is more difficulty to reurn than the first.it impacts an expansile impulse on cough.

Irreducible hernia

The contents cannot be returned to the abdomen and there is no evidenceof other complications. This is due to adhesions between the sac and its contents or from overcrowding within the sac. Irreducible without other symptoms is almost diagnostic of an omentocele especially in femoral and umbilicalhernia.

Obstructed hernia:

There is an irreducible hernia containing intestine which is obstructed from without but there is no interference to the bld supply to the bowel. The symptoms are less severe and the onset more gradual than is the case in strangulation.

Strangulated hernia:

The bld supply to the contents is seriouslyimpaired, rendering gangrene imminent. Gangrene may occur as early as 5-6 hrs after the onset of the first symptom of strangulation. Although inguinal hernia is four times common than femoral hernia, a femoral hernia is more likely to strangulate because of thenarrowness of the neck of the sac and its rigid walls.

Indirect (oblique) inguinal hernia:

- This is the commonest of all the hernias.
- Most common in the young whereas direct is common in middle life or after.
- In early life it is more common on the right side in the male because of the deffered descent of the right testis
- After the second decade the left inguinal hernia are as common as the right.
- The hernia is bilateral in nearly 30% of cases. There are three types of inguinal oblique hernia i.e.

i) Bubonocele – the hernia is limited to the inguinal canal

ii) Funicular – the peocessus vaginalis is closed just above the epidymis. The contents of the sac can be felt separately from the testis which lies below the hernia.

iii) Complete – also known as scrotal. It is rarely present at birth but commonly encountered in infancy. It also occurs in adolescence and adulthood. The testes appear to lie within the lower part of the hernia.

Clinical features of hernia:

- It occurs at any age
- males are 20 times affected than females
- pt complains of pain in the groin or reffered to the testicles when performing heavy work or strenuous exercise.
- Transient swelling may occur (seen and felt) when pt coughs (in the inguinalregion).
- Positive expansile impulse on coughing
- When the sac is still in the inguinal canal the bulge may be seen (better) by observing the region from the side or even looking down the abdominal wall while standing slightly behind the the respective shoulder of the pt.

How to examine for inguinal hernia:

- The clinician is seated infront of a standing pt with legs apart.
- The pt is instructed to look up at the ceiling and asked to cough.
- The hernia usually comes down
- The examiner looks for the impulse and should be able to come out with the following;

➢ Whether the hernia is right or left or bilateral
➢ Whether it is inguinal or femoral
➢ Whether it is direct or indirect inguinal hernia
➢ Whether reducible or irreducible
➢ Whether complete or incomplete
➢ The contents.

DDX:

In males;

- A vaginal hydrocele
- Encysted hydrocele of the cord
- Femoral hernia
- An incompletely descended testis
- Lipoma
- Spermatocele
- Abscess

In females;

- Hydrocele of the canal of Nuck
- Femoral hernia.

Treatment of indirect inguinal hernia:

Surgery is the treatment of choice

- Herniotomy- the hernia sac is dissected out and opening the sac, the contents are reduced and the neck transfixed and the remainder removed.
- Herniotomy and repair (herniorrhaphy)
- Hernial sac is excised
- Repair of thestretched internal inguinal ring and the transversalis fascia.
- Reinforcement of the posterior wall of the inguinal canal

NB the first two steps should be done without tension.

Femoral hernia:

- It is the third commonest hernia (incisional is the second).
- Accounts for about 20% of hernia in women
- Accounts for about 5% of hernia in males
- It can never be controlled by truss like any other hernia
- It is the most liable to become strangulated because of the narrowness of the neck of the sac and rigitidy of the femoral ring.

Sex incidence:

- The female to male ratio is 2:1
- Female pts are frequently elderly while the
- Male pts ar ebetween 30-45 yrs
- It is more prevalent in women who have deliverd than in nulliparous
- The broader female pelvis also predisposes to the condition.

Pathology:

The hernia passes down the femoral canal descending verticall as far as the saphenous opening. It is confined to the inelastic walls of the femoral canal and is narrow but once it escapes through the saphenous opening into the loose areolar tissue of the groin, it expands sometimes considerably. A fully distended femoral hernia assumes the shape of a retort (closed bottle with a long narrow bent spout), and its bulbous extremity may be above the inguinal ligament.

Clinical features:

- Rare before puberty
- Prevalence rises between 20 and 40 yrs and continues to old age.
- The right side is affected twice as much as the left
- It is bilateral in 20% of the cases
- The symptoms of a femoral hernia are more pronounced than those of inguinal hernia
- Adherence of the greater omentum sometimes causes a dragging pain
- A large sac is rarely present

DDX:

1. Inguinal hernia
2. A saphenous varix (varicosity)
3. An enlarged femoral lymphnodes
4. Lipoma
5. Femoral aneurysm
6. Psoas abscess
7. A distended psoas bursa
8. Ruptured adductor longus with haematoma

Umbilical hernia:

Exomphalos (omphalocele)

- Occurs once in every 6000 births

-It is due to failure of all or part of the mid gut to return to the coelom during early life

Sometimes a large sac ruptures during birth.

When it remains unruptured it is semi transluscent. Although very thin it consists of three layers;

- Outer layer of amniotic membrane
- Middle layer of wharlou's jelly
- Inner layer of peritoneum

Types of exomphalos;

i) Exomphalos minor:

- the sac is relatively small and the umbilicus is attached to its summit.
- A loop of small intestine or meckel's diverticulum can be included in the ligature applied to the base of an umbilical cord containing this protrution

Treatment:

The cord is only twisted to reduce the contents of the sac through the narrow opening into the peritoneal cavity and to retain them by firm strapping. Despite a seropurulent discharge on no account must the strapping be removed for fourteen days.

ii) Exomphalos major:

The umbilical cord is attached to the inferior aspect of the swelling which contains small and large intestine and nearly always a portion of the liver. Half the cases belong to this group.

Treatment:

Operation within the first few hrs of life is the only hope, otherwise the sac will burst. In order to prevent further distension of the contents of the sac, the infant sould not be fed. A few newborns with a ruptured sac have survived following immediate operation and antibiotic therapy

Umbilical hernia of infants and children:

- This is a hernia through a weak umbilical scar usually as aresult of neonatal sepsis.
- The ratio of males to females is 2: 1
- Often symptomless but increases with crying and this causes pain which makes the infant to cry more.
- Small hernias are spherical
- Big hernias are conical
- Obstruction or strangulation below the age of three yrs is extremely uncommon.

Treatment:

- Conservative treatment is successful in 93% of cases
- If symptomless, reassure the parents
- A big percentage of hernias disappear spontaneously during the 1st few months
- Cure may be hastened by putting the skin and abdominal musculature together by adhesive strapping placed across the abdomen.

Paraumbilical hernia (supra or infra umbilical hernia):

- In adults the hernia does not occur through the umbilical scar.
- This is a protrusion through the linea alba just above or sometimes just below the umbilicus.
- As it enlarges it becomes rounded or oval in shape with a tendency to sag downwards
- It can become very large

- The neck of the sac is often narrow as compared to the size of the sac.
- The sac contains greater omentum often accompanied by small intestine
- May contain portion of the transverse colon.
- In long standing cases the sac sometimes becomes localized due to adherence of omentum to its fundus.

Epigastric hernia:

Occurs through the linea alba anywhere between the xiphoid process and the umbilicus, usually midway between these structures. Usually commences as a protrution of extraperitoneal fat through the linea alba. Sometimes more than one hernias are present. A swelling the size of apea consists of a protrusion of extraperitoneal fat only (fatty hernia of the linea alba), if the protrusion enlarges, it drags a pouch of peritoneum after, and so becomes a tru epigastric hernia. The mouth of the hernia is rarely large enough to permit a portion of hollow viscus to enter it. Consequently the sac is either empty or it contains a small portion of greater omentum. The cause may be the direct result of a sudden strain tearing the interlacing fibres of the linea alba. The pts are often manual workers between 30- 45 yrs.

Clinical features:

1. Symptomless- may present as a small fatty hernia of the linea felt than can be seen. It may be symptomless only to discovered on routine abdominal examination (palpation).
2. Painful – there may be attacks of local pain which is worse on physical exertion. May be tender to touch and tight clothing.
3. Referred pain - the pt may complain of pain suggestive of a peptic ulcer. The pt may have not noticed the hernia.

Treatment:

If it gives rise to symptoms surgery is the only remedy.

INCISIONAL HERNIA:

Incisional hernia (ventral, postoperative hernia):

Etiology:

- Most often in obese pts
- Persistent postoperative cough
- Postoperative abdominal distension
- There is a high incidence of incisional hernia foll oiwngoperations for peritonotis, because, as a rule, the wound becomes infected.

Clinical features:

- The degree varies

- The hernia may occur through a small portion of the scar usually at the lower end
- There may be a diffuse bulging of the whole length of the incision
- Post operative hernia especially one through a lower abdominal scar usually increase steadily in size the contents becoming irreducible
- Sometimes the skin overlying it is so thin and atrophic so that normal peristalisi can be seen
- Attacks of subacute intestinal obstruction are common and strangulation liable to occur at the neck of the sac.

Treatment:

1. Palliative- an abdominal belt is sometimes satisfactory especially in cases where a hernia through an upper abdominal incision.
2. Operation.

FAECAL FISTULA:

An external fistula communicatin with the caecum sometimes follows;

- An operation for gangrenous appendicitis or
- The opening of an appendicular abscess.

A faecal fistula can occur from;

- Necrosis of a gangrenous patch of intestine after the relief of a strangulated hernia or
- A leak after an intestinal anastomosis
- The opening of an abscess connected with chronic diverticulitis or
- Carcinoma of the colon
- Other causes include;

➢ Tb peritonitis
➢ Ileocaecal actinomycosi
➢ Amoebiasis
➢ Regional ileitis (always follows operation)

External faecal fistulae are divided into three;

1. A track lined by mucus membrane protruding above skin level
2. A direct track lined by granulation tissue communicating with the exterior
3. A long ortous track lined by fibrous tissue and partly epithelialized

- The discharge from a fistula connected with the duodenum or jejunum is bile stained and causes severe excoriation of the skin.
- When the ileum or caecum are involved the discharge is fluid faecal matter
- When the distal colon is involved it is solid or semi-solid faecal matter
- When the leak from the small intestine or caecum is small it may be difficulty to distinguish a faecal discharge from a faeculent pus.

- Methylene blue is given orally and if a faecal leak is present the blue colour will be distinguished easily in the discharge a few hrs later.

Treatment:

- Those fistulas which connect to the small intestine tend to heal spontaneously provided there is no obstruction beyond the fistulous opening.
- The abdominal wall must be protected form erosion by the use of a disposable ileostomy bag
- Give nil orall or minimal residual diet and Supplement by intravenous feeding, this will facilitate closure by resting the bowel while maintaining the condition of the pt.

ANAL FISSURE (FISSURE IN ANO)

Def. An elongated ulcer in the long axis of the lower anal canal.

Site:

Midline posteriorly (90%)

Next mot common is the midline anteriorly)

Aetiology:

Not completely understood. during defaecation the pressure of a hard faecal mass is mainly on the posterior anal tissues, in which event the overlying epithelium is greatly stretched and being relatively unsupported by muscle, is vulnerable by hard stool.

Other causes:

1. Incorrectly performed haemorrhoidectomy where too much skin is removed leading to anal \
2. Leading to tearing when hard stool passes.

Acute anal fissure:

There is a deep tear through the skin of the anal margin extending into the anal canal. There is little inflammatory induration or oedema of its edges. There is accompanying spasm of the anal sphincter muscle.

Chronic anal fissure:

There are inflamed indurated margins,and a base consisting either scar tissue or the lower border of the internal sphincter muscle. The ulcer is shaped like a canoe and has a skin tag, which is usually oedematous, at the inferior extremely. The tag is known as a sentinel pile be cause it guards the fissure. There may be spasm of internal sphincter. Chronic fissure in ano have a specific cause, often a granulomatous infection e.g. crohn's disease or syphilis. biopsy is adviced.

Clinical features:

- Common in women especially in the child bearing age
- Uncommon in the aged because of muscular atony
- Not rare in children. May occur in infancy and may cause megacolon

1. Pain – sharp agonizing pain starting during defaecatio, often overwhelming in intensity and lasting an hour or more. As a rule it stops suddenly and comes over in the next bowel action. Pts tend to become constipatedrather than enduringthe agony of defaecation.
2. Bleeding: usuallyslight and consists of bright streaks of blood on the stool or tissue paper.
3. Discharge: fully established caseshave a slight discharge.

On examination:

- A sentinel tag can usually be seen
- Together with a typical history of a tightly closed, puckered anus,is pathognomonic of the condition.

By gently parting the margins of the anus, the lower end of the fissure can be seen.

Digital examination should not be done because of pain unless;

1. The fissure cannot be seen
2. You want to exclude a major intrarectal pathology.

It is therefore importantto apply a local anaesthetic such as 5% xylocaine before examination

DDX:

i) Ca anus in its early stage
ii) Multiple fissures
iii) Anal chancre
iv) Tuberculosis ulcer
v) Proctalgia fugax- A disease not related to organic disease characterized by attacks of severe pain arising in the rectum, reccuring at irregular intervals

Treatment:

The aim of treatment is complete relaxation of the internal sphincter. The pain is intense and needs to be addresed.

Conservative management:if the fissure is acute and superficialand the inflammation is minimal, conservative management is advised. Xylocaine 5% in a water soluble lubricant is applied. After 5 minutes a well lubricated finger is introduced into the anal canal. Alternatively a small dilator is introduced and even a bigger one if the anaesthesia allows.

Operative measures:

A wide, forcible dilatation of the sphincter under G.A the index and middlefinger of eachhand are inserted spontaneouslyinto the anus and pulled apart to maximal dilation. Pt may go home after the procedure but should be warned of possible faecal incontinence for between 7-10 days.

If the above procedure is not successful or if the fissure is chronic with fibrosis, a skin tag. Or a mucus polyp then surgery is advised.

HAEMORRHOIDS (PILES):

They are veins occurring in relation to the anus.

Classification:

A. It is customary classified by degree;
- First degree – only bleeding announces their presence
- Second degree – spontaneously reducing prolapsed at defaecation
- Third degree – prolapsed requiring manual replacement
- Fourth degree – permanent prolapsed.

B. They may be external or internal in relation to the anal orifice.

The external ones are covered by skin while the internal ones lie beneath the anal mucus membrane. When the two varieties are associated, they are known as interoexternal heamorrhoids.

The veins forming internal haemorrhoids become engorged as the anal lining descends and is gripped by the anal sphincters.

Haemorrhoids may be symptomatic of some other conditions and this important fact must be remembered. They appear in;

- Carcinoma of the rectum
- During pregnancy
- Straining at micturition due to stricture or prostate
- From chronic constipation

A great majority of haemorrhoids are not symptomatic.

INTERNAL HAEMORRHOIDS:

- They are exceedingly common.
- They include interoexternal haemorrhoids
- Essentially it s a dilatation of the internal veinous plexus within an enlarged displaced anal cushion.

1. Heredity
2. Morphological – veins in both internal and external haemorrhoidal vein (plexuses) don't have valves. This produces a high pressure in the lower rectum. Haemorrhoids are rare in animals except a few fat old dogs.

3. Anatomical.

a) The collecting adicles of the superior haemorrhoidal veins lie unsupported in the very loose submucous connective tissue of the anorectum
b) The veins pass through muscular tissue and are liable to be constricted by its contraction during defaecation
c) The superior rectal veins, being tributaries of the portal veins have no valves

Pathology:

Internal haemorrhoids are arranged in three groups, at 3, 7, and 11 o'clock with the pt in lithotomy position. The distribution is attributed to the internal supply of the anus whereby there are two subdivisions of the right branch of the superior rectal artery, but the Lt branch remains single. There may be smaller secondary haemorrhoids in between the three primary haemorrhoids. A principal haemrhoid can be divided into three parts;

i) Pedicle – is situated at the anorectal ring. On proctoscopy it is seen to be covered with a pale pink mucosa. Occasionally a pulsating artery may be felt.
ii) The internal haemorrhids – commences just below the anorectal ring. It is bright red or purple and covered by mucous membrane
iii) An external associated haemorrhoid - it lie between dentate line and the anal margin. It is covered by skin, through which blue veins can be seen, unless fibrosis has occurred.

Clinical features:

1. Bleeding: - at first the bleeding is slight, it is bright red and occurs during defaecation. Haemorrhoids that bleed but do not prolapsed are called first degree haemoorrhoids.
2. Prolapsed: - a much later symptom. Initially slight occurring on at stool with spontaneous reduction. Later they don't reduce unless returned by the patient. Those that prolapsed on defaecation and return or need to be replaced manually and then stay reduced are called second degree haemorrhoids.

Haemorrhoids that are permanently prolapsedare called 3rd degree haemorrhoids

3. Discharge: - a mucoid discharge is a frequent feature. Pruiritis will almost always follow this discharge.
4. Pain: - pain is absent unless complications set in. any pt complaining of painful piles must be suspected of having another condition (possibly serious) and examined accordingly.
5. Anaemia: - due to persistent profuse bleeding.

On examination:

There may be no evidence of internal haemorrhoids. Redundant folds or tags of skin can be seen in the position of one or more of the three primary haemorrhoids. On straining the internal haemorrhoid may come into view transiently, or if they are of the 3rd degree they are and remain prolapsed.

- **Digital examination:**

Internal haemorrhoids cannot be felt unless they are thrombosed

- **Proctoscopy**: - a proctoscope is passed to the fullest extent and the obturatoris removed. The proctoscope is then slowly withdrawn just below the anorectal ring. Internal haemorrhoids if present will bulge into the lumen of the proctoscope.
- **Sigmoidoscopy**: - Should be done as a precaution in every case to rule out other conditions like ca.

Differential diagnosis;

1. Anal tags – they are cutaneous protruberances at the junction of the anderm and perianal ski n whose origin is unknown.
2. Fibroepithelial polyp
3. Sentinel pile
4. Fissure
5. Dermatitis
6. Perianal haematoma
7. Rectal prolapsed
8. Rectal tumour

Complications:

1. Profuse haemorrhage
2. Strangulation
3. Thrombosis
4. Ulceration
5. Gangrenous
6. Fibrosis
7. Suppuration
8. Pyelephlebitis

Treatment:

a. Non operative:

Recommended when a haemorrhoid is a symptom of other conditions or disease except when a carcinoma is present. The bowels are regulated by hydrophilic colloids (isogel etc) and a small dose of senokot nocte prn. Various creams can be inserted into the rectum from a collapsible tube filled with a nozzle at night and before defaecation suppositories are also useful.

b. **Incase of inflamed and permanently prolapsed haemorrhoids**:

Oedema should be reduced by repeated dressings of glycerine and then surgery gives permanent cure. However severe MAD is frequently successful in relieving symptoms even in advanced cases of the piles.

c. **Active treatment**:

- Injection treatment – indications include; (i) ideal for 1st degree internal haemorrhoids which bleed. (ii) early 2nd degree haemorrhoids are often cured by the method but some relapse. 3-5 ml of 5% phenol in almond oil is injected.

d. **Banding**:

For 2nd degree haemorrhoids which are too large for successful handling by injection. A tight elastic band is slipped to the base of the pedicle of thee haemorrhoid with a special instrument. The band causes ischaemic necrosis of the piles which slough off within a few days. The procedure should be painless if done properly. Not more than two piles should be banded at one sitting.

- **Cryosurgery** – application of liquid nitrogen (extremely cold at -195 degrees celciuc) causes coagullative necrosis of the piles which subsequently separate and fall off. It causes troublesome mucus discharge which has limited its use.
- **Photocoagulation** – this is application of infrared coagulation by a specially designed instrument. It is effective and painless.
- Operation -- indicationfor peration include;(the following cases are unsuitable for injection or banding)

➢ 3rd degree haemorrhoids
➢ Failure of non operaton treatment for 2nd degree haemorrhoids
➢ Fibrosed haemorrhoids
➢ Interoexternal haemorrhoids- when the external haemorrhoid is well defined

Therefore the above are theindications for heamorrhoidectomy.

Haemorrhoidectomy:

Preoperative management;

- An aperients on the evening prior to operation
- A soap and water enema is administered
- The anal region is shaved
- On the morning of the operation the rectum is evacuated

MAD (which greatly reduces post operative pain) is done before the ligation and excision of the haemorrhoid. Petroleum jelly gauze are tacked into the anus so as to cover the area denuded of skin. A pad of gauze and wool are applied and a T bandage applied.

Postoperative managmeet:

- Saline sitz baths twice daily

- The dressing can be removed after the first day
- Enema is not necessary because bowel usually move at day 4-5.
- Baths and dry dressing are best
- Antibiotics.
- Analgesics.

Postoperative complications:

1. Pain
– May necessitate pethidine PR.
– Xylocaine jelly into the rectum by a nozzle may help.
2. Urine retension
- Especially in males due to a rectal tube or pack or both
- Reassure pt an give an analgesic before resorting to catheterization.
3. Reactionary haemorrhage
- More common than secondary haemorrhage
- The haemorrhage may be mainly or entirely concealed but becomes eveident on rectal examination.
- Treatment is a suitable dose of morphine and if there is no response the patient is taken to theatre to identify and secure the bleeder.
4. Late/secondary haemorrhage:
- Uncommon
- Occurs at the 7^{th}/8^{th} day
- Controlled by morphine and is not a suture is applied to the bleeder
5. Anal stricture
6. Fissure
7. Submucous abscess

External haemorrhoids:

They comprise a group of distinct clinical entities i.e.

1. A thrombosed external haemorrhoid: -- commonly known as a perianal haematoma. A small clot occurring in the perianal subcutaneous connective tissue and usually superficial to the corrugators' cutis ani muscle. It is due to back pressure on an anal venule as a result of straining at stool; coughing or lifting of a heavy wt. the condition appears suddenly and is very painful .

O/e

– A tense, tender swelling is seen.
– The haematoma is usually situated in a lateral region of the anal margin.

If untreated it may;

➢ Resolve

- Suppurate
- Fibrose leading to a cutaneous tag
- Burst and extrude the clot or continue bleeding.

In the majority of the cases resolution or fibrosis occurs. It is reffered to as a 5 day , self curing lesion. If seen within the first 36 hrs of onset it should be treated as an emergency and the haemorrhoid should be bisected and the two halves excised together.

2. Associated with internal haemorrhoids i.e interoexternal haemorrhoids
3. Dilatation of the veins of the anal verge. It becomes eveident only if the pt strains, when a bluish cushionlikering appears.
4. A sentinel pile is associated with an anal fissure

FISTULA IN ANO:

Def. A track lined by granulation tissue which connects deeply in the anal canal or rectum and superficially on the skin around the anus.

Usually results from an anorectal abscess which bursts spontaneously or was opened inadequadely. It continues to discharge and because of constant reinfection from the anal canal or rectum, it never closes permanentlywithout surgical intervention. An anorectal abscess may produce a track to the orifice of which has the appearance of a fistula, but it does not communicate with the anal canal or the rectum. This is not a fistula but a sinus.

Types of anal fistulae:

They are divided into

a) Low level-they open into the canal below the anorectal ring.
b) High level –they open into the anal canal at or above the anorectal ring.

A low level fistula can be laid open without fear of permanent incontinence, while a high level fistula can be treated only by staged operations often with a protective colostomy to prevent septic complications and to shorten healing time between the stages.

Clinical features of low level fistulas:

- Persistent seopurulent discharge which irritates the skin in the neighbourhood and cau ses discomfort.
- The history usually dates back for many years
- Pain is not present so long as the opening is large enough to allow pus to escape.
- If the orifice is occluded pain increases until discharge erupts
- Usually there is a solitary external opening within 3-7 cm of the anus which presents as a small elevation with granulation tissue protruding from the mouth of the opening. Sometimes superficial healing occurs, pus accumulates and an abscess reforms and di scharges through the

same opening, or anew opening. Therefore there may be two or more external openings usually grouped together on the right or left of the midline.

NB: fistulas with an external opening in relation to the anterior half of the anus tend to be of the direct type. Those with an external opening or openings in relation to the posterior half of the anus (are more common), usually have curving tracks).

- **On digital examination**:

Usually an internal opening can be felt as a nodule on the wall of the anal canal. Irrespective of the number of external openings, there is almost always only one internal opening.

- **Proctoscopy**: sometimes reveals the internal opening of the fistula.
- **Probing**: used to be done in the wards or outpatient but are of no value and painful and liable to spread infection.
- **CXR** should be done to rule out PTB.

Treatment:

The fistulous track must be laid open from the terminal end to the source.

NB: fistulas of high level are difficulty to treat. If the track is laid open as for low level fistulas incontinence will follow.

THE URINARY SYSTEM:

Urinary symptoms:

The three most common symptoms of the tract are;

1. Haematuria

This is the presence of in urine and should not be ignored. Haematuria is described as;

- Initial haematuria
- Blood throughout the urtinary stream
- Terminal haematuria
- Bleeding with clots and perhaps pices of tissues.

These variations may be used to indicate the site of bleeding, thus;

a) Renal causes
- Infarcts
- Injury
- Stone
- Tb
- Hypernephroma

- Papillary tumours
- Carcinoma
- Wilm's tumour

b) Ureteral causes

- Stone
- Neoplasm
- Focal and glomerularonephritis

c) Bladder causes

- Cystitis
- Tumours
- Tb
- Stones
- Bilharzias
- Tauma

d) Prostate cauases

- Benign tumours
- Malignant tumours

e) Urethral causes

- Injury
- Stones
- Tumours

f) Blood dyscrasias

- Purpura
- Sickle cell disease
- Anticoagulants

2. Pain

Different forms of pain arise from the urinary tract;

a) Renal pain

- It arises from the kidney
- May be associated with inflammation or obstruction at the level of the pelvi-ureteric junction and is well localized
- If due to obstruction it will be described as an ache
- If infection is present, there may be irritation of the psoas muscle. There may be tenderness at the costovertebral angleand the right hypochondriac region

b) Ureteric pain

This is pain passing from the loin to the groin. It is associated with the passing of a stone.

- It is usually sudden in onset and severe
- Pt is unable to get comfortable and prefers to pace about
- It is different from the painof peritonsm which is excercabation of the pain.

- The pain is restricted to the loin when the stone is just coming out of the kidney. As the stone moves down to the upper ureter the pain moves into the upper abdomen and gradually down towards the groin.
- When the stone is in the lower ureter the pain radiates into the perineum or to the vulva in women and sometimes to the base of the penis in men. It may also go down the inner thigh.

c) Vesical pain

- It is usually sited at the suprapubic region
- It is made worse by the filling of the bladder or emptying and sometimes by defaecation.

d) Prostatic and seminal vesicle pain

- It is usually deeply seated in the rectum or the perineum
- Usually described as an ache which may also occur both in the suprapubic region and in one or both iliac fossa.

e) Urethral pain

- Typically scalding in nature and associated with active cystitis
- May also be felt in the base of the penis, particularly with the presence of vesical calculus (at the neck of the bladder)

3. Frequency

It may be due to;

- Incomplete emptying as in prostatism
- Irritability of bladder by inflammation or stones
- A contracted bladder.
- A dieresis
- Sphincter weakness

URINARY TRACT INVESTIGATIONS:

A complete history and physical examination is vital before investigation of any condition. Remember that haematuria may not arise from primary disease of the urinary tract but because of some other systemic illness e.g. bleeding secondary to thrombocytopaenia (reduced platelets) caused by leukaemia.

1. The urine:

The urine may be examined;

a) Macroscopically for;
- Presence of RBCs
- " " WBCs
- Bacteria

- Crystals and
- Casts

b) Biochemically for;

- Electrolytes
- Glucose
- Bilirubin and its products

c) PH

d) Bacteriologically – by simple culture which may reveal infection

Mid stream specimen is better because

- It avoids contamination and
- There is no risk of catheterization

Early morning specimen should be cultured on a Lowenstein Jensen medium when looking for Tb.

e) Malignancy

The papanicolaou stain used to demonstrate the presence of ca in situ in the cervix has been adopted for cells exfoliating from the urinary tract. This has a place in the detection of urothelial malignancy

2. Renal function tests

Structural damage to the may occur before the functional damage becomes apparent. Kidney function impairement can occur in three principal ways:

i. Pre-renal – reduced renal plasma flows
ii. Renal - damage to the glomeruli
iii. Postrenal – impaired tubules

Range of specific gravity (concentration)

- The ability of the kidney to concentrate or dilute urine is a good test of their functional integrity.
- Fluid is withheld for 12hrs overnight.
- The specific gravity of the first two specimens should reach 1.020
- A specific gravity of 1.025 in a urine free of protein indicates good renal function
- A liter of water is given orally. Within 4hrs the specific gravity should be as low as 1.002.
- A fixed gravity of 1.010 under these varying circumstances is an evidence of impaired function of the distal renal tubules.

Blood urea – is normally between 2.5-6.5 mmol/l (15-40mg/100ml)

Creatinine – It is 42-130mmol/l (0.2-1.5mg/100ml). A more sensitive test of renal function

Creatinine clearance – it measures the glomeruli filtration rate. One sample of bld and an accurately timed collection of urine for a period of aout 24 hrs are all that is required. The normal clearance is between 90 and 130 ml/min. it decreases in old age.

3. Radiology:

- Plain abdominal x-ray can reveal a lot of information like;
  - Gastric air bubble on the left
  - Liver on the right
  - Presence of previous pelvic fractures
  - Kidney shadow
  - Calculi
- Intravenous urogram
  - It relies on the glomeruli filtration of sodium diatrizoate (this substance is allergenic)
  - A laxative is given prior to the investigation and pt starved for 6-8hrs
  - The contrast (urographin or niopam 370) is given i.v on the forearm. Observe for any rxn. An x-ray is then taken to demonstrate the urinary system. At the end of the study the pt is asked to to micturate and a final film taken to demonstrate the bladder.
- Infusion urography (rarely used this days)
  - In pts with a bld urea of 16.6-33.2 mmol/l (100-200mg/100ml), an infusion of 50% hypaque 2ml/kg in an equal amount of isotonic saline over a period of 10 minutes.
- Retrograde uretropyelography

A systoscope is initially passed and subsequent passage of ureteric catheter into the ureteric orifice. It normally requires GA. A contrast media is injected into the ureter if there is doubt about the presence of intraluminal lesion.

- Renal arteriography
- Cystography
- Urethrography
- Venography

4. Ultrasonography

- It can demonstrate the size of kidneys and the thickness of the cortex. Individual calyces can be demonstrated and the width of the collecting system (hydronephrosis can be diagnosed within seconds). Fluid can be differentiated from solid

5. Computed tomography
6. Endoscopy.

ANURIA

It is also called suppression of urine

Definition: absence of secretion of urine for 12hrs

Oliguria: excretion of urine less than 300ml in 24hrs

Classification of anuria;

1. Pre-renal anuria

The Bp in the glomeruli is normally about 90mmhg. When the systolic Bp falls bellow 70mmhg, filtration from the glomeruli stops.if the glomeruli are diseased, a higher pressure of up to100mmhg may be inadequate to maintain filtration.

Causes of pre renal anuria are;

- Traumatic shock
- Severe haemorrhage
- Spinal anaesthesia
- Extensive burns
- Dehydration from vomiting, diarrhea or excessive sweating
- Cardiac failure

Treatment:

- Bld transfusion if due to haemorrhage
- i.v fluids (plasma expanders) if due to dehydration. If hypotension is prolonged damage to the renal epithelium results leading to tubular necrosis.

2. Acute renal anuria (acute tubular necrosis)

- It occurs due to damage to or ischaemia of the renal tubular epithelium

The principal causes (surgical) are;

a) Severe shock (hypotension) lasting 2hrs or more.
b) Incompatible BT
c) Bilateral pyelonephritis
d) Crush syndrome
e) Concealed accidental haemorrhage and abortion
f) Certain poisons e.g. media used for arteriography, toxins of eclampsia, chemicals like mercury salts and carbon tetrachloride.
g) Acute pancreatitis
h) Operation on jaundiced pts
i) Drugs like aminoglycosides, cephalosporins esp. in pts on lasix.

3. Post renal anuria (obstructive anuria)

Causes;

a) Calculi anuria – it arises in one of the following ways;

- Impaction of calculi in the ureter of the only functional kidney, the other kidney being congenitally absent, previously removed or destroyed by disease.
- Both ureters become obstructed by stones or crystals

b) Sulfonamide crystalluria
c) Uric acid crystalluria
d) Accidental ligation of the ureters
e) Involovement of both ureters in a neoplastic process e.g ca cx, ca prostate
f) Involvement of the urters in retroperitoneal fibrosis

KIDNEYS AND URETERS

Congenital abnormalities of the kidneys:

1. **Absence of one kidney**:

Pyelography reveals only one functioning kidney and at cysytoscopy only one ureteric orifice is present. Sometimes a ureter and a pelvis are present on the non functioning sid e, but the parenchyma is almost or entirely absent. In either case the functioning kidney is hypertrophied. An absent or congenitally atrophic kidney is present in about 1:1400 individuals.

2. **Renal ectiopia**:

Occurs once in 1000 cases. The kidney is arrested in some part of its normal ascent, usually at the brim of the pelvis. As a rule the kidney the kidney of the opposite side is present and in its normal position. The left kidney is far more often affected than the right kidney. The reason is not known. Ectopia may present a diagnostic problem when acute disease develops in the ectopic kidney.

3. **Horse shoe kidney**:

During embryonic life, the most medial subdivision of the primary mesophrenic bud of each side fuse and the kidney fail to ascend completely. The adrenal glands develop separately and are in their normal positions. It occurs one in every 1000 of renal cases.the bridge joining the lower poles lies infront of the 4^{th} lumbar vertebra. Fusion occurs very early, when the embryo is about 30-40 days old, at which time the two masses of mesoblast destined to form the kidneys lie very close together. Exceptionally, it is the upper poles that are fused.

Clinical features:

The kidneys are prone to become diseased mainly because the ureters are angulated as they pass over the fused isthmus. This causes urine stasis and consequently simple infection, tuberculosis, and calculus formation are common complications.

Investigations:

1. Urography – the most characteristic finding, the lowest calyx on each side is reversed in position (directed towards the vertebral column). Rarely most or all of the ureterscurve like a flower

vase. Urinary complications are more frequent in pregnancy but horse shoe kidney is not a contraindication to pregnancy. Surgery is only indicated when trying to correct anabdominal aortic aneurysm.

4. Congenital cystic kidney (polycystic kidney

In 18% of the cases there is a congenital cystic liver. Occasionally the pancreas and lung are similary affected. The disease is hereditaryand can be transmitted by either parent. The disease is not easily demonstrable in a urograph before the late teen (13 – 19yr). It rarely gives clinical manifestations until the 4th decade of life.

Pathology:

The kidney becomes enormously enlarged. The surface appears as many bubbles. On section the renal parenchyma is occupied with cysts of varying sizes, some containing clear fluid, others thick brown material while others contain coagulated blood.

Clinical features:

- the condition is slightly common in women than men.
- Renal enlargement
- pain due to the wt of the organ dragging upon its pedicle or totension within the cyst.
- Haematuria ue to to rupture of one of the cysts due to overdistension. It may be profuse.
- Infection. The most common complication being pyelonephritis.
- Hypertension usually those aove the age of 20yr. Nobody understands why some pts escape this complications
- Uraemia – Pts complain of anorexia, head ache and vague gastric symptoms. Later drowsiness and vomiting occurs.

Investigations:

- Excretory urography is the best way of confirming the diagnosis
- The kidney shadows are enlarged in all directions
- Renal pelvis is enlarged and may be elongated
- The calyces are stretched over the cysts and are oten narrow (like the legs of a spider) or bell like.

Treatment:

1. Conservative;

- Drink large quantities of water routinely
- Have low protein diet
- Iron supplements to prevent anaemia
- Infections, when present, should be treated with appropriate drugs

2. Operative;

To remove pressure on the remaining renal parenchyma. Many surgeons advocate this operation now.

3. Renal failure should be treated by dialysis possibly bilateral nephrectomy and kidney transplantation.

Other congenital abnormalities of the kidney:

a) Infantile polycystic disease
b) Unilateral multicystic disease
c) Solitary renal cyst
d) Aberrant renal vsls

Congenital abnormalities of the renal pelvis and ureter:

I. Duplication of the renal pelvis

- Is the commonest congenital anomally of the renal tract
- Is found in about 4% of the pts
- Usually unilateral
- Common in the left side than the right

ii. Duplication of a ureter

- Double ureters are present in addition to double renal pelvis in a bout 3% of the cases
- The ureters usually join in the lower 1/3 of theeir course and have a common opening into the bladder
- Less frequently the ureters open into the bladder independently.

iii. Ectopic ureteric orifice

- This is a rare anomally
- In both sexes the existence of double ureter is determined by excretory urography.

iv. Congenital mega ureter

- May be unilateral or bilateral
- In later stages it is accompanied by other congenital abnormalities
- Common in males
- Usually symptomless until infection sets in.

v. Post caval ureter

The right ureter passes behind the inferior vena cava instead of lying to the right of it. It may cause obstructive symptoms. If the symptoms occur it may require surgery to divide the ureter at the dilated portion and reanastomosed infront of the vena cava.

vi. Ureterocele

- This is due to congenital atresia of a ureteric orifice which causes a cystic swelling of the intramural portion of the urete.
- It may sometimes involve the muscle coat
- It may be discovered at childhood but is more often discovered during adulthood

- Women are affcted more than men
- It is bilateral in 10% of the cases.

Treatment:

- Many cases are symptomless and require no treatment
- It may complicate to;
  - Hydronephrosis
  - Stone formation
  - Reccurent ifections

This complications may necessitate treatment, by cauterizing a hole through the wall of the cyst with a diathermy.

CONGENITAL ABNORMALITIES OF THE URINARY BLADDER:

1. **Diverticula**:

A vesical diverticulum consists of a pouch like eversion or evagination of the bladder wall. It may arise as a congenital defect but are more commonly acquired lesions from persistant urethral obstruction. The congenital type may be due to a focal failure of development of the normal musculature or to some urinary tract obstruction during fetal development. Acqu ired diverticula are more often seen with prostatic enlargement (hyperplasia or neoplasia), producing obstruction to the urine outflow and marked muscle thickening of the bladder wall. The increased intravesical pressure causes outpouching of the bladder wall and the formation of diverticula. Diverticuli are important because they constitute sites of urinary stasis and predispose to infection and the formation of bladder calculi. They may also predispose to vesicoureteral reflux. Rarely, carcinomas may aris e in bladder diverticula.

2. **Extrophy:**
   This is the presence of a developmental failure in the anterior wall of the abdomen and the bladder. The bladder either communicates directly through a large defect with the surface of the body or lies as an open sac. The exposed bladder mucosa may undergo colonic glandular metaplasia and subject to the development of infection this infection often spreads to upper levels of urinary system. When chronic infections persists the mucosa becomes converted into an ulcerated surface of granulation tissue and the marginal epithelium becomes transformed into a stratified squamous type which may later develop into carcinoma (adenocarcinoma). May surgically be corrected with long term survival.

   Congenital abnormalities of the penis:
   They range from congenital absence and hypoplasia to hyperplasia, duplication etc. most of these are extremely rare and apparent on inspectioncertain other abnormalities are more frequent and have greater clinical significancy.

Hypospadias:

It occurs once in every 350 cases of congenital anomalies of the renal system.

This is when the external meatus is situated at some point on the undersurface of the penis or in the perineum.it is the commonest congenital abnormality of the urethra.

Classification:

1. **Glandular hypospadias:**

There is an ectopic opening on the undersurface of the glans penis which is separated from a blind depression at the normalsite of the external urinary meatus. Sometimes achannel connec ts the ectopic too the normal meatus.

2. **Coronal hypospadias:**

The meatus is situated at the junction of the undersurface of the glans with the body of the penis.

3. **Penile**: Meatus opens at some point of the undersurface of the penis.
4. **Penoscrotal**: the urethra opens at the junction of the penis with the scrotum.
5. **Perineal**: The scrotum is split and the urethra opens between its two halves. Is sometimes associated with bilateral maldescended testes, in which even the sex of the child is difficult to determine.

Glandular hypospadias is the most frequent variety and due to failure of canalization of the glans.

In all except the glandular variety the penis is curved in a downward direction. The furtheraway the opening is from the normal position, the more pronounced is the bowing. In all the cases the inferior aspect of the prepuce is poorly developed (hooded prepuce).

Treatment:

No treatment is required in the glandular type unless the opening is too small in which case meatotomy is performed. In either varietie, plastic operation (reconstruction) is carri ed out.on this account, circumcision during infancy should not be carried out in this cases because the skin is required for this reconstruction.

Epispadiasis:

- It is very rare.
- Occurs 1/30000 males and 1/400000 females
- The defect may be glandular, penile,or total, the later usually associated with ectopia vesicae. In
- the first two varieties the urethral orifice is situated at the dorsum, and in the penile variety, the penis curves upwards.
- The female variety is associated with many other abnormalities.

Phimosis:

This is the narrowing of the preputal orifice

Causes;

1. Acquired as a result of chronic or acute inflammation of the lining of the prepuce.
2. Congenital narrowing of the preputal orifice which is associated with an unduly long foreskin.

Clinical features:

In extreme cases;

- When the pt micturates the prepuce balloons out first, and a thin weak stream of urine follows.
- Difficulty of micturition, with residual urine, hydroureters, hydronephrosis are rarely due to phimosis but due to atresia of the meatus which may be hidden by the phimosis.

Treatment:

Circumcision.

Paraphimosis:

This is when a tight prepuce has been retracted but cannot be returned and is constricting the glans penis which is engorged and oedematous.

Treatment:

1. Injection of 1ml of isotonic saline containing 150 turbidity units of hyalurodinase into each lateral aspect of the swollen ring of prepuce. 15 minutes later the swelling is much reduced and in early cases reduction is done with easy.
2. Circumcision when the above fails.

Undescended testes:

This could be in a form of;

1. Incomplete descent – The testis is arrested in some part of its route to the scrotum
2. Ectopic testis: The testis is abnormally placed outside this route

Incompletely descended testis:

Incidence;

a) In neonatal period – Incomplete descent in one or both sides is 4% in full term infants and 30% in premature infants. The testis or testes reach the scrotum in 50% of children in their 1st month of life. Incomplete descent of testis is not usually detected during infancy.

b) In late childhood and puberty: - The incidence is 2%. Still remains unrecognized unless a routine medical examination is done. In a few cases, presence of hernia pain or acute torsion in that order is what makes the abnormality to be detected.
c) In adult life: - it is believed that a good number of cases have this problem but don't seek advice unless symptoms develop.

The incidence is 0.8% and in 10% of unilateral cases there is a familial history.

Pathology:

Up to the age of 6yrs there are no microscopical differences between an incompletely descended and a normal testis. After 6yr, due to the higher temperature to which it is subjected, the development of the undescended testis is progressively retarded. By the time puberty is reached, the incompletely descended testis is flabby and hardly more than half the size of its intr ascrotal counterpart. Histologically, the epithelial elements are grossly immature and at the age of 16yrs irreversible destruction has occurred in the germinal epithelium. The internal excretory mechanism of an incompletely descended testis functions but feebly and, often after a few months or yrs, stops and therefore the power of spermatogenesis may be negligible. The internal excretory activity of an incompletely descended testis is reduced.in bilateral cryptoorchidism about half the normal amount of androgen is produced. If an incompletely descended testis is brought down satisfactorily before puberty, it develops and functions satisfactorily.

Clinical features:

- Rt testis 50% of the cases
- Lt testis 30% of the cases
- Bilaterally 20% of the cases
- Other abnormalities of the urinary tract may be present
- The testes may be;
  - Retained within the abdomen
  - In the inguinal canal
  - In the superficial sub-inguinal pouch
- When both testes are in the abdomen or inguinal canal and are impalpable, the condition is known as cryptorchidism (hidden testes)
- When the testes can be placed in anormal position temporarily it is not truly imperfectly descended testes. It is known as a retractile testis. They require no endocrine nor operative treatment. Retractile testes should be suspected if the scrotum is normal.

Hazards of an incompletely descended testis:

1. Sterility in bilateral cases
2. Pain: - A inguinal testis is liable to repeated trauma
3. An associated indirect inguinal or interstitial hernia in 70% of the cases. It is the hernia that causes symptoms in the adolescents and adults.

4. Torsion
5. Epididymorchitis. The right one is not possible to differentiate from acute appendicitis.
6. Atrophy
7. Increased liability to malignancy. 1 in 20 abdominal becomes malignant. 1 in 80 inguinal becomes malignant.

Treatment:

- Operation is never performed in the 1st 2yrs
- Operation is performed between 4-8 yrs
- Percentage of success after puberty falls considerably
- Orchydopexy which involves , mobilization of the spermatic cord and testicular vsls, retaining the mobilized testes in the descended position
- In cryptorchidism one side should be operated at a time with an interval of 6 months between the operations.

Haematocele:

Can be recent or old clotted bld.

Recent: - It is usually as a result of injury of small bld vsls during tapping or aspiration of a hydrocele. Refilling of the sac with considerable pain and tenderness and poor or absent transillumination (this confirms the diagnosis).the treatment should be urgent surgery with evacuation of bld and excision or eversion of the sac. Exploration also confirms whether the testes are ruptured. Neoplasms may also present this way.

Old clotted haematocele:

Slow haemorrhage into the tunica vaginalis can occur spontaneously apparently painless. There is no history of trauma to the testis nor pain to the organ. An old clotted haematocele mimmicks a neoplasm of the testis soo closely that pre-operative differential diagnosis is sometimes impossible.

Treatment:

Unless exposure of the organ leaves no doubt as to the innocent nature of the swelling, unquestionably orchidectomy should be done. As a rule it is impossible to be certain of the diagnosis until the mass has been bisected. The testes are usually compressed as to be virtually functionless.

Ddx of scrotal swelling:

- Seminoma
- Teratoma
- Lymphoma
- Interstitial tumour
- Varicocele

- Elephantiasis
- Sebaceous cyst of the scrotum
- Carcinoma of the scrotum

CARCINOMA OF THE PENIS:

Aetiology:

NB: circumcision correctly performedsoon fter irth gives almost total immunity against ca penis (reason not understood). Circumcision ater ear infancy does not provide the same degree of protection (reason not known). The best example are the muslims who circumcise their boys at the age of between 4 and 9 yrs. Ca penis therefore occurs in men whohave not been circumcised in early infancyand is favoured by chronic balanitis.

The following are also precarcinomatous;

1. Leukoplakia of glans
2. Longstanding genital warts due to human papilloma virus
3. Pagets disease of the penis

Pathology:

The condition presents squamous carcinoma of two types;

i) The flat or infiltrating – This t is associated with leukoplakia
ii) The papilliferous type – commences in papilloma of longstanding period

The growth strictly remains local for many months. The earliest spread is to the inguinal and then to the iliac lymphnodes. Direct spread to the body of the penis is prevented for many months by the fascial sheath of the corona. Once this barrier is broken the growth extends more rapidly and the illiac lymphnodes become onvolved. Distant metastatic deposits are not frequent.

Clinical features:

It is a big mistake to belief that the disease is confined to the elderly because 40% of the cases are under the age of 40yrs.

- The progress of the disease is slow
- The first symptoms are a mild irritation and purulent discharge from the prepuse
- These symptoms are usually neglected and sometimes more than
- A yr after symptoms have appeared, there is blood stained discharge which has a foul smell
- Pain is absent
- Inguinal lymphnodes are enlarged in 60% of the pts, half of these enlargement is due to secondary deposits and the remainder is due to sepsis.
- The prepuse cannot be retracted and must be slit to view the lesion.
- In all cases biopsy must be taken.

Treatment:

1. Radiotherapy gives good results with small, well differentiated tumours. The five yr survival rate is 60-70%.
2. Surgery

Indications for surgery;

- Large anaplastic growths, if there is infiltration of the shaft of the penis
- When RT has failed
- Elderly men who don't mind the mutilation as much as the pain to be expected from the extensive reaction to RT.

PARTIAL AMPUTATION: is used for distal growths provided there is at least 2cm of the dependent shaft which is not involved.

Total amputation: done when there is an advanced , infiltrating, or anaplastic lesion (must).

SURGICAL EMERGENCIES:

RUPTURED URETHRA:
the rupture of the urethra is categorized into two distinguished classes, thus, bulbous and membranosus (intrapelvic) urethra. Each is again subdivided into complete and incomplete, which relates to the circumference of the urethral wall. When it is said to be total or partial it relates to the thickness of the wall.

Rupture of the bulbous urethra:

This is a more common accident. There is usually a history of a fall astride on a projecting object. The common cases involve;

- Cycling
- Loose manhole
- Gymnasium accident. Falling astride on the beam.

Clinical features;

The triad sign of a ruptured b urethra is

- Haemorrhage
- A perineal haematoma
- Retension of urine

Preliminary treatment and investigation:

If suspected and to diminish the possibility of extravasatio, the pt should be advised against trying to pass urine.

- No attempt should be made to catheterize him until fit to be taken to theatre where asepsis can be assured and operation done in necessary cases.
- Start on pain killers and sedative like morphine
- Start on antibiotics
- If circumstances don't allow and the bladder is full it should be treated by suprapubic puncture until the pt is fit to be taken to theatre.
- If the pt has passed urine when first seen and there is no extravasation, then the rupture is partial and catheterization should be avoided.

Treatment:

a) Catheterization under aseptic precautions should be attempted (folley's catheter). The roof of the urethra is likely to be intactand the peak of the catheter should be directed towards it.
b) If catheterization is successful it should be left in situ for 48hrs. A perineal haematoma should be drained and the midline wound packed.
c) If the catheter fails to pass, the site of obstruction should be explored with the pt in lithotomy position, through a midline incision. If not possible a suprapubiccystostomy should be performed and a fully curved sound passed down from the internal meatus. The urethra is then repaired and a suprapubic catheter left in situ. If the bladder has been repaired without cystostomy, a cathetercan be passed into the bladderfrom the urethrostomy. The perineal wound is packed but not stitched.

- A course of antibiotics is continued until healing is complete
- Urine must be recultured after a wk
- Perineal wound irrigated daily with a weak eusol solution
- Remove catheter after 8-10 days
- Perineal leak may persist for a few days
- P.O.S must be passed 2wks after healing and a urethrogram performed after 2wks to reval the result

Complications:

1. Subcutaneous extravasation of urine. In case of total rupture if the pt tries to pass urine.
2. Stricture – usually due to infection.

Rupture of membranous urethra (extraperitoneal rupture of the bdladder)

Intrapelvic rupture of the urethra occurs in the membranous portion near the apex of the prostate. As in extra peritoneal rupture of the bladder it may be due to;

- Penetrating wounds
- Fracture of the pelvis

The prostate is firmly attached to the pubis by the pubo-prostatic ligaments. A displaced fracture of one ischiopubic ramus infront of the corpus carvenosum usually ruptures the urethra. A "butterfly" fracture of both pubic rami on each side usually springs back into place and the urethra remains intact.

- Complete rupture with floating prostate -1-2%
- Incomplete intrapelvic rupture of urethra - 4-6%
- Extraperitoneal rupture of the bladder – 4-6%
- Combined urethra and bladder damage – 1-2%

NB: clinical features.

The most common cause of pelvic rupture are; R.T.A, severe crash injuries and falls from heights. The clinician should therefore bear in mind that there could be accompanying injuries like, head, thoracic, abdominal or fracture of long bones. The pts are usually in a state of shock from either bld loss and may be unconcous. A careful assessment of the whole pt must be made including radiographs of the head (if there is hx of head injury or loss of consciousness), cxr to rule out pneumothorax, abdomen, pelvis and appropriate long bones.

- The classic sign of urethral injury is bld at the external urinary meatus
- It may be associated with gradual distension of the bladder (assuming it is not injured).
- Signs of peritonitis will develop if there is an intraperitoneal rupture of the bladder
- If the rupture is extraperitoneal the diagnosis is often difficult.
- For the extraperitoneal rupture there is some suprapubic tenderness and perhaps a little dullness to percussion.
- There will be blood in urine and often clots
- A plain X-R may show pelvic or any other bony structure.

Treatment:

- Treat for blood loss and shock

Mitchel's theory:

- Injury to the urethra is incomplete in majority of pts.
- Catheterization through the urethra might lead to further damage and cause the rupture tto be complete
- Suprapubic cystostomy and the bladder drained which allows the pt recover.
- Primary recovery of the urethra should be discouraged
- Endoscopy is then attempted after 3/52 after the injury which may allow a cystoscopy to be passed past an area of bld clot and granulation tissue into the urethra.

Brandy's theory:

It recommends a single attempt to pass a small soft urethral catheter.

- This should be done by an experienced using aseptic technique.
- If this fails, a suprapubic cystostomy should be performed and the bladder drained for a day or two until the pt is fit for management of the urethra.
- The pt should be given a broad spectrum antibiotic. **Complications:**

1. Urethral stricture
2. Urinary incontinence – damage of external sphincter.
3. Impotence – erectile impotence.

Causes are usually through;

- Blows
- Fall upon the loin
- Road traffic accident

Haematuria following

Minor injuris should suggest the possibility of pre existing renal abnormality like stone, hydronephrosis or tuberculosis.

Types of renal injuries;

a) Small subcapsular haemorrhage
b) Large subcapsular haemorrhage
c) Cortical laceration with perinephric haematoma
d) Medullary laceration with bleeding into the renal pelvis
e) Complete rupture

- The degree of injury varies.
- The kidney may be partially or wholly avulsed from its pedicle.
- Tears of the renal parenchyma follow the lines of the uriniferous tubules.
- The whole of one pole may be detatched
- The injury is usually peritoneal
- Occasionally in children the peritoneum may be torn (due to little adipose tissue) involving the renal capsule and this may allow urine to escape into the peritoneum.

Clinical features;

- Superficial bruising (rarely)
- Local pain and tenderness
- Haematuria;

➢ A cardinal sign but may appear hrs after injury. If haemorrhage is profuse, it may be followed by clot colic.

- Severe delayed haematuria – sudden profuse haematuria can occur between the 3rd day and 3rd wk after injury. This is due to a clot becoming dislodged.
- Meteorism (abdominal distension) due to retroperitoneal haematoma
- Perinephric haematoma – should be suspected if there is even a slight flattening of the normal contour of the loin (provided there is no scoliosis).

Management:

- Conservative management is usually successful
- Bear in mind that other organs may me injured
- Pt rests flat in bed until macroscopic haematuria has been absent for 1/52
- Morphine for pain and sedation
- Hourly pulse and blood pressure
- Prophylaxis (antibiotics)
- Save a sample of each specimen of urine passed
- GXM
- Urgent i.v.u if no change with the above management then explorative laparatomy should be performed.

Exploration should be done if;

- There are signs of progressive blood loss
- An expanding mass develops in the loin
- Signs of perirenal infection develops

RENAL CALCULI:

ETIOLOGY;

1. **Dietetic**

Deficiency of vitamin A which causes a desquamation of epithelium forming a nidus around which the stone is deposited.

2. **Altered urinary solutes and cholloids;**

In hot climates, the concentration of solutes will rise. Also reduction of urinary cholloid which absorb solutes, or excess microproteins which may seize calcium, predisposes to the formation of insoluble complex. Common in

3. **Decreased urinary output of citrate.**

The presence of urine (300- 900ml/24hrs) as citric acid, tend to maintain in solution otherwise relatively insoluble calcium phosphate and carbonate.

4. **Renal infection**

Infection favours the formation of urinary calculi. Usually common in Streptococcal, staphylococcal or proteous infection. The predominant bacteria found in the nuclei of urinary calculi are staphylococci and E. coli.

5. Inadequate urinary drainage.

Stones are prone to occur in pts with obstruction to the free passage of urine

6. Prolonged immobilization

From any cause in paraplegia. This may lead to poor renal drainage favouring the deposition of calcium phosphate calculi. In uninfected cases spontaneous dissolution sometimes occurs.

7. Hyperthyroidism

Rare but occurs in 5% of the cases hyperthyroidism results in a great increase in the elimination of calcium in the urine.

8. Randal's plaque

When there is erosion at the apex of one of the renal papillae. On this erosion are deposited urinary salts (Randal's plaque) minute concretions (microliths) occur normally in the renal parenchyma.

Varieties of renal calculi

1. *Oxalate calculus (calcium oxalate)* – Popularly known as the mubery stone. It is covered with sharp projections. These causes the kidney to bleed and altered blood is precipitated on the surface of the stone. An oxalate calculus which is usually single casts a good shadow radiologically. A calcium monohydrate stone is very hard.
2. *Phosphate calculus*

- Usually magnesium phosphate but
- Sometimes combined with ammonium magnesium phosphate and rarely composed of phosphate and is smooth and dirty white
- In alkaline urine it enlarges rapidly and often filling the renal calyces, taking their shape (staghorn calculi). Because it is smooth, a phosphatic calculus gives rise to few symptoms, until it has attained a large size. Because of itssize but not density it is demonstrated readily by x-rays

3. *Uric acid and urate calculi*

- Are hard and smooth
- Because they are usually multiple, they are usually faceted
- Their colour varies from yellow to reddish brown.
- Pure uric acid calculi are not opaque to x-rays
- But absolutely pure uric acid calculi are not common. The majority contain enough calcium oxalate crystals making them radio –opaque
- Calculi of ammonium and sodium urate are sometimes found in children. Such stones are yellow, soft and friable and unless they contain impurities they do not cast an x-ray shadow.

4. *Cystine calculi*

- Usually appear in the urinary tract of pts with cystinuria
- Sometimes encountered in young girls
- Cystinuria occurs because of reduce d or absent reabsorption of cystine from the renal tubules
- Cystine crystals are hexagonal, white, transluscent, and appear only in acid urine
- They assume a cast of the renal pelvis and calyces
- Are hard and pink or yellow when first removed
- On exposure the colour changes to a greenish colour
- They are opaque due to the sulphur they contain

5. *Xanthine calculi*

- They are extremely rare
- They are smooth and round
- Brick red in colour
- They show a lamellar structure

Clinical features of renal stones

- 50% of pts with renal stones are between 30 and 50yrs
- The male – female ratio is 4:3
- Symptoms are not stereotype and sometimes the diagnosis remain obsequire until radiological examination has been done

1. **Quiescent calculus**: - Especially those mainly composed of phosphate lie dormant for a long time during which there is progressive destruction of the renal parenchyma, uraemia may be the first indication. Secondary infection usually supervenes.
2. **Pain:** - I s the leading symptom in 75% of the cases

a) Fixed renal pain – is located in the renal angle posteriorly often worse on movement, particulary on walking upstairs

b) Ureteric pain: -

- An agonizing pain passing from the loin to the groin coming on suddenly.
- Causes the pt to draw up his kneesand roll about
- Often accompanied by vomiting and profuse sweating
- Strangury may occur if the stone is in the intramural ureter (strangury is the passage of few drops of urine, often blood stained, after painful straining.
- The pulse quickens and as the attack progresses the temperature becomes subnormal.
- An attack of colic rarely lasts more than 8hrs
- The condition is often due to stone entering the ureter but also occurs when a stone in the renal pelvis temporarily blocks the pelvi-ureteric junction. Colick may be caused by passage of a shower of oxalate crystals e.g after eating an excess of strawberries.

3. Haematuria:

- Rarely haematuria, is a leading , or the only symptom.
- As a rule haematuria occurs in small amount (enough to render the urine smoky) during or after an attack of pain.

4. **Pyuria:**

- Infection of the kidney is liable to supervene and pus will be found in the urine in varying amounts.
- Stones lead to an increase in the number of white cells in the urine even in the absence of infection

Investigations:

1. Radiographs of K.U.B

DDX of renal calculi on a radiograph;

- Calcified lumbar/mesenteric lymphnode
- Gallstone or concretion in the appendix
- Drugs like fesolate or FB in the G.I.Tphleboliths
- Ossified tip of the 12th rib
- A chip fracture of the transverse process of the lumbar vertebrae
- Calcified tuberculous lesion of the kidney
- A calcified suprarenal gland

2. Urine analysis and culture
3. Blood urea nitrites
4. Cystoscopy

Treatment:

1. Surgical removal
2. Extracorporal shock wave lithotripsy (stone banger). A shock wave is generated within an ellipse and if the kidney stone is placed where the the waves focus will be broken up.

TORSION OF THE TESTES:

It is also known as torsion of the spermatic cord.

Predisposing factors (causes):

- Torsion of the testis is not common
- It does not occur in a normal, fully descended testis.
- It's anchorage prevents rotation.

Therefore one of the testicular anomalies must be present;

1. Inversion of the testis – This is the commonest predisposing cause
2. High investiment of the tunica vaginalis – I t causes the testis to hang within the tunica like a clapper in the bell. Occasionally, torsion is extravaginal.

3. In cases where the body of the testis is separated from the epididymis. –Torsion of the body can occur without involving the cord. The twisting is confined to the messentry that joins the testis to the epididymis

Exciting causes:

Normally the cremaster contracts concurrently with violent contraction of abdominal musculature. Contraction of the spirally attached cremaster favours rotation around the vertical axis in the relevant cases.

- Straining at stool
- Lifting heavy wts and
- Coitus ,

Are all exciting causes but usually hx does not reveal any of them.

- Occasionally torsion develops during sleep.

Clinical features:

- Highest incidence is between 10 and 25 yrs of age
- The second most common age period is during infancy
- Symptoms vary with the degree of torsion
- The most common is the sudden agonizing pain in the groin and lower abdomen and
- Vomiting

NB: it is difficult to distinguish torsion of an imperfectly descended testis from a strangulated inguinal hernia until the parts have been exposed on operation.

- The side of the scrotum is empty and oedematous
- A tender lump at the external abdominal ring
- Torsion of a completely descended testis is less difficult problem to diagnosis
- In other times it can be confused exactly by a small, tense, strangulated inguinal hernia compressing the cord and causing congestion.
- Torsion of the fully descended testis can also simulate closely acute epididymo-orchitis.

After 6hrs or so have elapsed the skin of the scrotum becomes reddened and slight elevation of temperature (up to 37.2 oc).

- Scrotal elevation relieves pain of epididymitis but increases the pain of torsion of the spermatic cord.
- It is therefore important to rule out mumps, in a boy, and urethritis in an adult. If these two are ruled out then the diagnosis should be insisted upon.

Treatment:

In the first hr or so, attempt to untwist the testis may immediately relief the pain. Try gentle twisting in one direction.relief is obtained if towards the right direction. Pain increases if it is towards the wrong direction.urgency is removed if untwisting is achieved but operation and fixation should be performed as quick as possible.

- Urgent exploration of the srotum is indicated if untwisting is not successful. If after exploration the testis is viable, then it mustbe fixed to avoid reccurence. The opposite testis should also be fixed at the same time.
- A totally infracted testis shoud be removed
- If the testis is seen days or wks later the pain is subsided and little is to be gained.
- The testis will slowly become woody and shrink to a fibrous nodule.
- The opposite testis must be fixed at an early date.

HYDROCELE:

Definition – it the collection of serous fluid in some part of the processus vaginalis, usually the tunica.

Anatomical varieties of congenital hydrocele:

1. Vaginal hydrocele
2. Infantile hydrocele (unusual)
3. Congenital hydrocele
4. Hydrocele of cord

Aetiology:

a) Excessive production of fluid within the sac e.g. secondary hydrocele
b) Defective absorption of hydrocele fluid by the tunica vaginalis. This appears to be the common variety of primary hydrocele. The cause is not clearbut damage to the endothelial wall by low grade infectionis thought to be the cause.
c) Interference with the drainage of fluid by the lymphatic vls of the cord.
d) By connection with the peritoneal cavity as in the congenital type.

Hydrocele fluid:

- Amber coloured
- Specific gravity is 1.022-1.024
- Contains water, inorganic salts, 6% albumin and some fibrinogen.
- If the fluid is run through a cannula into a receptacle, it does not clot but,
- If a few drops of bld come into contact the hydrocele fluid clots firmly.
- In old standing cases the fluid may contain so much cholesterol

Diagnostic rules for hydrocele:

- 99% out of every 100 hydroceles are transluscent
- On examination it is possible to get above the swelling.

1. **Primary vaginal hydrocele:**

- most common in middle aged or elderly men but may
- occur (not common) in children
- common in tropical countries
- the only comlaint is the swelling and pt only seeks aid when the swelling is massive
- in acute hydrocele in a young man, there may be an underlying testicular neoplasm which can be confirmed by an ultra sound
- About 5% of inguinal hernias are associated with a vaginal hydrocele of the same side.
- Usually alarge hydrocele obsecuresa small inguinal hernia.
-

2. **Congenital hydrocel:**

- The processus vaginalis communicates with the peritoneal cavity but;
- The communicating orifice is too small to develop a hernia
- The fluid disappears into the abdomen when the pt lies horizontal but reappears when an erect position is resumed.
- Ascites or ascetic TB peritonitis should be suspected in bilateral cases.

3. **Infantile hydrocele:**

- Does not necessarily appear in infants
- The tunica and processus vaginalis are distended up to the internal abdominal ring but there is no connection with the general peritoneal cavity.

4. **Encysted hydrocele of the cord**

- Forms a smooth, oval swelling associated with the spermatic cord.
- May be mistaken for irreducible inguinal hernia

- On traction upon the testis it moves downwards and becomes less mobile (and this confirms the diagnosis).

HYDROCELE OF THE CANAL OF NUCK:

- This is a condition comparable to the encysted hydrocele of the cord
- It occurs in females
- The cyst lies in relation to the round ligament
- Unlike hydrocele of the cord, a hydrocele of the canal of Nuck is always wholly, or partially, in the inguinal canal.

HYDROCELE en BISAC:

- Rare
- Has two intercommunicating sacs, one above, one below the neck of the scrotum
- The upper sac lies supervicial to or partly within the inguinal canal and it may move itself into muscle layer.

Complications of a hydrocele:

1. Rupture
- Usually traumatic but may be
- Sometimes spontaneous
- On rare occasions cure results after absorption of the fluid
2. Hernia of the hydrocele in longstanding cases. This is caused by tension of fluid within the tunica.
3. Transformation into a haematocele. May be spontaneous or as a result of trauma
4. Calcification of the sac wallsometimes occurs in the longstanding cases

Management:

1. Surgery –Hydroelectomy
2. Tapping. After transillumination, the swelling is made tense by manual compression. A canula is inserted into an unquestionably transluscent area and the fluid evacuated.the sac usuallyrefills. Repeated tapping is liable to be followed by oozing of blood into the sac. Deposits of blood on the walls of the sac increases its thickness and so diminishes its transluscence

SECONDARY HYDROCELE:

- An effusion into the tunica vaginalis which accompanies certain conditions affecting the testes.
- It is usually associated with acute and chronic epididymoorchitis.
- Is nearly always present in syphilitic testis.
- Occasionally complicates malignant conditions of the testes
- Secondary hydrocele rarely attains a large size.
- Usually lax and does not interfere with the palpation of the testes and its epididymis.

POST HERNIORRHAPHY HYDROCELE:

Appears after an operation for inguinal hernia in 0.2% 0f the cases where there wasn't an evidence of a hydrocele pre-operatively. This could be due to damage of the lymphatic vsls of the tunica vaginalis.

HYDROCELE OF A HERNIAL SAC:

The neck of a hernia sac becomes plugged with omentum or occluded by adhesions and a hydrocele develops.

FILARIAL HYDROCELE AND CHYLOCELE:

- Filarial hydrocele usually follows repeated attacks of filarial epididymitis.
- It develops rapidly or gradually.
- Can be large or small.
- It is frequently bilateral.
- In early cases, the hydrocele fluid is similar to that found I the idiopathic variety.
- In longstanding cases, if the fluid is placed in a tall glass, after afew hrs a film of liquid fat (chyle) will be floating on the surface.
- This is rich in cholesterol, and is derived from rupture of a lymph varix into the tunica.
- The presence of chyle is proof (positive) of the filarial origin of a hydrocele.

INCONTINENCE OF URINE:

Passage of urine depends on a fine balance between the bladder having a storage capacity. The brain being aware of fullness, and the subsequent coordination of the detrussor and the urethral sphincters to perform the act of voiding. The basic prerequisites of urinary incontinence include;

- Anatomical intergrity
- Cerebral awareness
- A degree of mobility and
- Competent sphincter control.

A careful history and physical examinationwill usually be sufficient to disclose the cause. Investigations of the urinary system as a whole, is nearly always indicated. The urine should be cultured for evidence of infectionand biochemical estimation of the bld to assess renal function.it may be approperiate to have anatomical visualization of the urinary tract with intravenous urography, although ultrasound examination will often provide adequate details.

Causes of incontinence:

Common causes may be classified into male, female or mixed sex groups;

Male;

- Chronic urine retension with overflow
- Secondary to BPH which may be coexistent with ca prostate
- Hypetrophy of the bladder neck usually in a yuoinger age group.
- Rarely urethral stricture
- The key to the diagnosis is history of prolonged hesitancy and a poor urinary stream with both daytime and nocturnal "drippling incontinence".

Examination may reveal;

- A visible distended bladder
- A lost transverse suprapubic crease
- A painless distension of bladder may be palpable or percused.
- Post-operative (post prostatectomy) – may result from injury to the external sphincter mechanism.

Females;

- The commonest is stress oincontinence secondary to laxity of the pelvic floor with incompetence of the bladder neck and sphincter mechanism. It most commonly occurs in the late multiparous group. The pt complains of loss of urine associated with coughing, sneezing and even with change in posiure.
- Cystocele.

Common on both sexes;

1. Congenital;
- Ectopiae viscae and severe epispadiasis
- Abnormal entry of a ureter below the sphincter complex or into the vagina in females
2. Trauma from pelvic injury or pelvic surgery
3. Infection – simple cystitis especially women
4. Endocrine – occasionally women suffering from thyroitoxicosis
5. Neoplasia – local advanced cas in the pelvis.

Other causes :

a) Neurogenic ;
- Myelodysplasia
- Multiple sclerosis
- Spinal cord injuries
- Cerebral dysfunction (CVA, Dementia)

b) Psychogenic;
- As in hysteria in women or

- Depressive illness in both sexes

c) Capacity disorders – reduced bladder capcity as in TB bladder which heals with fibrosis.

d) Drug induced;

- Anticholinergics
- Tricyclic anti-depressants
- Lithium
- Some anti-depressants
- A side effect of phenothiazides

Treatment of functional incontinence:

1. **Drugs;**

- To increase strength of bladder neck (adrenergic agonists)
- To decrease strength of neck (adrenergic blockers)
- Mixed action on bladder neck and CNS (tricyclic drugs)

2. **Intermittent self catheterization**
3. **Devices for collection or control;**

- Condoms
- Indwelling catheters
- Penile clamp (cunnigham device)

4. **Outlet surgery;**

- Prostatectomy
- Bladder neck widening
- Sphinctectomy

5. **Denervatio n of bladder;**

- Neurectomy
- Transection of bladder

6. **Augumentation of bladder capacity;**

- Ileocystoplasty
- Caecocystoplasty

7. **Artificial sphincter**
8. **Urinary diversion** –ureteroileostomy
9. **Correction of bladder neck distortion;**

- Raising bladder neck
- Correction of cystocele
- Levatoplasty procedures

10. **Control of infection**

URINE RETENSION

It can be acute or chronic

Chronic leads to ultimate retension with overflow.

ACUET RETENSION:

Aetiollogy;

In males:

Prostatic enlargement

Urethral stricture

Post operative

In females:

Retroverted gravid uterus

Multiple sclerosis

Hysteria

In male child:

Meatal ulcer with scabbing

Other causes;

Following spinal analgesia

Blood clot in bladder

Rupture of the urethra

Neurogenic (injury to the spinal cord)

Fecal impaction in the rectum

Acute urethritis/prostatis

Urethral calculus

Phimosis

Certain drugs

Muscular atony due to advanced age

Anal pain (e.g. following haemorrhoidectomy)

Clinical features:

- History of not passing urine for some hrs and is unable to do so.
- Suprapubic sweeling by a full bladder which is tender on palpation and dull on percussion above the symphysis pubis.
- Acute pain due to spasm.

Treatment:

NB- never relief or attempt to relief acute retension and send the pt home.

- Always admit for observation for at least 24hrs to;
- Relief anxiety – chemotherapy
- Warm bath usually reliefs (pt may void)
- Pass a urethral catheter using aseptic technique

If catheterization fails the the other option will be tried i.e;

1. Suprapubic puncture — with a wide bore needle to relief acute retension
2. Suprapubic cystostomy with catheterization- through a 1.25 cm incision made under L.A 2.5 cm below the level at which the anterior surface of the bladder curves upwards and backwards to form a dome.
3. Immediate prostatectomy incase of benign enlargement of the prostate if the pt fit.
4. Urethral instrumentation- incase of stricture to dilate the stricture. A catheter is usually passed afterwards to empty the bladder

After catherterization it is important to record the volume drained and a pt's abdomen examined, a few minutes after the procedure to rule out any intra abdominal pathology.

CHRONIC URINE RETENSION:

e) Distension is usually painless
f) Bld urea must be estimated before any attempt is made to relief the retension. If urea is < 70 mg% (12mmol/l) treatment is as outlined for retension. > 70mg% slow decompression is advisable.

RETENSION WITH OVERFLOW:

The pt has no control over his urine, small amounts passing involuntarily from time to time from an overflowing bladder. It may follow a neglected acute retension or chronic retension. Treatment is as for acute retension but decompression of the bladder must be done slowly.

Summary of digfferentials of urine retension

1. *Intraluminal*:
- blood clot
- Stone (rare): acute pain in the penis and glans.
- Prolapsing bladder tumor
- Congenital Urethral valves- neonates, males, recurrent UTI
- Foreign body (rare)
- Tumours (rare): TCC or squamous cell carcinoma. History of haematuria, working in dye or rubber industry.

2. *Intramural:*
- BPH: frequency, nocturia, hesitancy, poor stream, dribbling, urgency.
- Prostatitis
- Prostate carcinoma
- Urethral stricture: history of trauma or serious infection, gradual onset of poor stream
- Urethral trauma: blood at meatus

3. *External*:
- Ovarian cyst: mobile iliac fossa mass
- Pregnancy
- Fibroids: palpable, bulky uterus, menorrhagia, dysmenorrhoea
- Pelvic mass
- Faecal impaction: spurious diarrhoea

4. *Neurological*
- Spinal injury: acute phase is lower motor neurone type, late phase is upper motor neurone type
- Multiple sclerosis
- Polio
- Prolapsed disc
- Diabetes: progressive upper motor neurone lesion
- Drugs: narcotics, anticholinergics, antihistamine, antipsychotics
- Postoperative: pain, drugs, pelvic nerve disturbance

FUNCTIONAL ANATOMY OF THE PROSTATE:

It lies in the pelvis infront of the rectum and behind the symphysis pubis

It surrounds the first part of the urethra

It sits at the base of the bladder

Seminal vesicle lies posteriorly and receives the vas deferens from the testis and drain into the posterior prostatic urethra.

Seminal vesicles don't store sperms but produce secretions which nature the spermatozoa

The prostate gland lies in the fibromuscular stroma and their ducts open into the posterolateral grooves on either side of the veromentosum. The epithelium is columnar commencing peripherally, and passing centrally, beneath the anatomical capsule lie the long branched prostatic gland proper.

This region is named as carcinomatous zone. Beneath this envelop, and separated from it by an indefinite capsule, lie another mass of secreting elements, also branched (the submucosal gland). The zone that they occupy is known as the adenomatous zone. Nearer the urethra are the unbranched urethral glands whose mouths opens directly into the urethra.

Into the urethra therefore, open the prostate ducts proper, the ducts of the submucisal and mucosal glands, and the common prostatic duct. That is whyinfection of the prostatic urethra is difficulty to eradicate.

The middle lobe is that part of the prostate between the common ejaculatory ducts and prostatic urethra.

An enlarged prostate is invested with three capsules;

- The compressed outer zone (true capsule)
- The anatomical capsule (false capsule)
- The prostatic sheath of pelvic fascia

Between the anatomical capsule and the prostatic sheath lies the prostatic venous plexus.

PHYSIOLOGY:

The prostate is purely a genital organ because is usually rudemental except during the

rutting season in animals (as goats).

Hormonal influence;

The prostate is governed by two hormones, thus,

1. Male (androgenic) – usually the one in large amount and most important testicular hormone. It is supplemented by the adrenal glands.
2. Female (estrogenic) – causes retrogressive changes.

Semen;

This is the fluid ejaculated at the time of orgasm

It contains sperms, secretions from seminal vesicals, prostate, cowpers glands and urethral glands

The volume is approximately 2.5 3.5 ml after several days of continence

The volume decreases with repeated ejaculations

Normally 100million sperms per ml

Acid phosphatase:

These are enzymes that split organic phosphates and most active at a PH of 5.

They are present in many human tissues but their concentrations in the adult prostate are greater than in any other organ or tissue.

These higher levels are attained after puberty.

Most of the acid phosphatasesecreted by the prostate drains along the prostatic duct into the urethra to keep the bld levels low.

When the cells producing this enzyme cannot discharge their products externally, the serum levels of acid phosphatase rises. The serum acid phosphatase is usually normal if the growth is confined to the gland and almost never in BPH slightly increased values commonly occur in;

- Acute prostatitis
- Paget's disease of bones
- Liver cirrhosis.

BENIGN ENLARGEMENT OF THE PROSTATE:

BENJAMIN BRODIE'S THEORY;

"When the hair becomes grey and scanty, when specks of earthly matter begi ns to be deposited in the artery and when a white zone is formed of the cornea, at this same time the prostate gland becomes increased in size."

Incidence:

Usually occurs in men above 50yrs of age

Most often between 60 and 70yrs

Is less frequent in Indians and more often in younger age groups

Rare in Negroes (the reason for these discrepancies is not known)

Aetiology:

1. Hormonic theory – as age advances the male hormones dimish while the quantity of estrogenic hormone is not decreased equally. The prostate therefore, enlarges due to predominance of the oestrogenic hormone.

2. The neopllastic theory – the prostatic enlargement is a benign neoplasm because the prostate is composed of fibrous tissue, muscle tissue, and glandular tissue, the neoplasm is a fibromyoadenoma.

Pathology:

Hyperplasia affects the glandular element and connective tissues but in variable degrees. The changes are similar to those occurring in breast dysplasia where adenosis, epitheliosis, and stromal proliferation occur in different proportions. Benign adenomatous hyperplasia affects the submucous group of glands, forming a nodular enlargement, "the typical lateral lobe" which compresses the external group of glands into a false capsule. As the gland enlarges extravesically, it it tends to displace the seminal vesicles so that instead of lying on the base of the bladder, these structures become a direct posterior relation of the upper limit of the prostate. When the h yperplasia affects the subcervical glands a "middle" lobe develops which projects into the bladder within the internal sphincter. Sometimes both the lateral lobes project into the bladder, so that when viewed from within the sides and back of the internal urinary meatus are surronded by an intravesical prostatic collar.

SECONDARY EFFECTS OF PROSTATIC ENLARGEMENT:

URETHRA;

Prostatic urethra becomes elongated

The canal is compressed laterally

BLADDER:

The musculature hypertrophies to overcome theobstruction

When the middle lobe projects upwards into the bladder it acts as a dam to the last ml of urine which remains in the prostatic pouch.

Calculi are prone to form in the stagnant pool of urine.\the enlarged prostate may compress the prostatic venous plexus leading to congestion of veins (vesical piles) at the base of the bladder and may cause haematuria.

If no relief to the obstruction the bladder hypertrophies to become atonic and tired bladder and makes no attempt to overcome the obstruction.

URETERS AND KIDNEYS:

Increased intravesical pressure causes dilatation of the ureters, followed by some degrees of hydronephrosis. The bladder hypertrophy wanes and the sphincter mechanism around the ureteric orifice ceases to function leading to reflux of urine from the bladder to the dilated ureters. This progresses leading to ascending infection

SEXUAL ORGANS:

In the early stages of prostatice enlargement there is increased libido.

Later impotence is the rule.

Clinical features of urine retension:

1. Frequency;

Is the earliest symptom

At first norcturnal

It becomes progressive and then becomes present day and night

When the vesical sphincter becomes stretched a little urine escapes into the normally empty prostatic urethra, causing an intense reflex desire to void and uregency is added to the frequency.

Later on, as residual urine increases frequency becomes more and more evident leading to terminal drippling.

Lastly cystitis and nocturia develops due to renal insufficiency.

2. Difficulty in micturition – pt waits patiently for urination to start. It is useless to strain.
3. Variable stream – usually weak, tends to stop and start and dribbles towards the end of micturition.
4. Pain – due to cystitis or acute retension of urine. Dull pain in the loins is indicative of hydronephrosis
5. Fullness of the perineum or rectum
6. Acute retension of urine is usually the first symptom that compels the pt to seek relief
7. Retension with overflow – urine constantly dribbles away. Norcturnal enuresis should be a warning
8. Haematuria – from an enlarged prostatic vein or ulceration of the prostate.
9. Renal insufficiency.

Examinations:

Pt lies supine on the bed or couch

Inspection;

- Suprapubic distension may be seen
- Loss of suprapubic transverse skin crease
- Tongue may be dry and brown, a sign of renal insufficiency

Palpation;

- Suprapubic mass
- Renal areas for tenderness and possible enlargement of the kidneys
- Epididymis for signs of inflammation

Percussion;

- Dull percussion note suprapubically

Urine;

- Low specific gravityindicates renal insufficiency

Rectal examination:

- Done in absence of full bladder
- Done bimanually on dorsal position

If benign;

- The lateral lobes are increased in size
- They are smooth, convex, typically elastic but may feel firm due to fibrous tissue
- Rectal mucosa is moved off the prostate
- Median sulcus is lost
- Feels rubbery.
- Bimanually an intravesical lobe can be felt
- If pressure is exerted on the apex of the prostate while finger already in the rectum, there will be a degree of mobility of the gland.
- Residual urine may be fely as a fluctuant mass over the prostate

If due to prostitis;

- Indurated and tender

If due to carcinoma;

- Stony hard and nodular

If due to prostatic calculi

- Stony hard and nodular

Conditions which mimmick prostatic obstruction;

- Diabetes mellitus
- Tabes dorsalis
- Disseminated sclerosis
- Cervical spondylitis
- Parkinson's disease and other neurological states

Investigations:

1. Micturography to record pt's stream and volume
2. Bld urea
3. Bld count
4. Serological tests for syphilis
5. Urinalysis for glucose, bacteria, albumin, c/s
6. i.v.u
7. plain abdominal X-ray – K.U.Bultrasound
8. cystourethroscopy

Management of retension:

- Acute retension is painful and should be relieved by decompression by the passage of urethral catheter.
- Chronic retension is painless and if no symptoms of coexisting infection and have normal creatinine (serum), they do not necessarily require catheterization
- In uraemic pts decompressing is a must
- Uraemic pts are usually dehydrated and therefore should be rehydrated
- Due to the chroinic back pressure on the dilated tubules within the kidney salt and water are not properly reabsorbed hence post obstructive diuresis (so replace the fluid)
- Pts are usually anaemic (transfuse)
- Catheterization or
- Suprapubic puncture
- Prostatectomy

Indications for prostatectomy:

- Prostatism – difficulty in micturition, increased, delay in starting, and a poor stream.
- Frequency alone is not an indication
- Enlargement rarely gets worse after 10 yrs
- Acute retension unrelieved by catheterization, emptying the bladder, and immediately removing the catheter.
- Chronic retension – residual urine of 200ml or more, raised bld urea, hydronephrosis and uraemia
- Complication – stone, infection, diverticulum formation

- Haemorrhage

Complications of prostatectomy:

Local:

1. Reactionary haemorrhage
2. Perforation of the bladder
3. Infection
4. Incontinence due to damage to external sphincter
5. Retrograde ejaculation and impotence
6. Stricture
7. Bladder neck contracture
8. Osteitis pubis

General complications:

9. Cardiopulmonary;
10. Pulmonary atelectasis
11. Pneumonia
12. Myocardial infarction
13. CCF
14. DVT
15. Water intoxication – reabsorption of water during transurethral irrigation which may lead to ccf, hyponatraemia and haemoptysis.
16. Confusion

URETHRAL STRICTURE:

Causes;

1. Congenital
2. Traumatic(accidental) – bulbous – membranous
3. Inflammatory – post gonococcal – posturethral chancre - tuberculous
4. Instrumentation - indwelling catheter - following passage of large caliber endoscope, notably a rectoscope
5. Postoperative – postprostatectomy – amputation of penis
6. Postgonococcal stricture: (It has reduced since the advent of antibiotics)

Sites;

- In the bulb – 70%

- At the penoscrotal junction
- Distal part of the spongy urethra (in that order).

NB; the membranous and prostatic parts are excempt

Multiple strictures are common

Where there are two strictures, the deeper is the narrower

When the strictures are three, the deepest is the narrowest

If a penile urethral stricture orifice is very narrow, there is rarely another stricture behind it.

Pathology:

Usually follows an inadequately treated gonorrhoea. The infection persists in the periurethral glands and spreads to the periglandular tissues. The tissues become infiltrated with round cells and fibroblasts. The infiltrated tissues then contract with the formation of scar tissue. There is also localized thrombophlebitis of the corpus spongiosum as seen in more dense varieties. Fibrosis in the bulbous urethra is mostly seen in the roof, while it predominates in the floor of the penile urethra. Most strictures are said to develop in the first yr after gonorrheal infection and may not give rise to difficult in micturition for 5-15yr.

Clinical features:

In large caliber stricture;

Passage of flakes (desquamated epithelium) in urine

There may be varying amount of urethral discharge (gleet) which is evident in the early mornings "dew drops".

The above symptoms are neglected until the caliber narrows causing difficulty in micturition.

NB; unlike obstruction due to an enlarged prostate, the pt feels he must strain to empty the bladder

Another distinguishing feature is the age. The pt is often younger than the prostate sufferer.

The stream becomes progressively narrower

Micturition is prolonged and dribbling occurs after the end of stream due to trickling of urine from the dilated urethra above the stricture

Frequency of micturition at first during the day and then both day and night and this is due to incomplete emptying of the bladder at each act of micturition, cystitis or both

The stricture may be palpated as an induration in the urethral floor in long standing cases.

If untreated, sooner or later retension of urine ensues.

In some cases, narrowness of the stricture leads to inability to expel residual urine, and acute on chronic retension, or retension with overflow occurs.

Investigations:

1. Urethras copy
– locates the precise site of the stricture
– May show false passages of recent penetration of the urethra infront of the stricture due to unskillful attempt to pass a bougie. The false passages are bound to bleed.
2. Urethrography – gives information concerning the length of the stricture

Treatment:

A. Instrumentation treatment:

Intermittent dilation carried out gently with bougies of increasing size.

- Don't forcibly dilate or overdilate the stricture as they may result in inflammatory oedema and subsequent formation of more fibrous tissue
- Small strictures should be dilated twice a week at first, then once a week for a month and then once a month for a yr.

B. Operative treatment:

- Urethrotomy
- Urethroplasty

Complications of urethral stricture:

1. Retension of urine
2. Urethral diverticulum- due to increased pressure behind the stricture
3. Periurethral abscess
4. Urethral fistula
5. Hydronephrosis > urinary infection> urinary calculi
6. Due to straining the following may be induced;

- Hernia
- Haemorrhoids
- Rectal prolapsed

TRAUMATOLOGY:

Introduction:

Bone- This is a supporting connective tissue forming the frame work of the body. Apparently it may look inert but it is in dynamic equilibrium with body metabolism. It is the main store house of calcium and phosphorous. It grows in length by endochondral ossification and in width by intramembranous ossification. It has bld vsls, nerves, and lymph vsls.

Blood supply:

1. Nutrient artery
2. Epiphyseal artery
3. Metaphyseal artery
4. Periosteal artery which enters bone through minute canaliculi (volkmann's canal) and supplies the outer 2/3 of the cortex.
5. From attached soft tissues, muscles and ligaments.

Functions of bone:

a) It forms the rigidy framework of the body
b) Levers muscles
c) Protection of the;

Brain and spinal cord

Heart and lungs

Liver and spleen

Urinary bladder

d) Contains marrow which produces Rbcs, Wbcs, and Platelets.
e) Stores calcium, phosphorous, magnesium and sodium

Biochemical composition of a bone:

Organic substances - 35%

Inorganic substances - 45%

Water -- 20 %

Histologically bones are;

1. Immature (woven)
2. Mature i.e. cortical (compact) or cancellous(spongy)

FRACTURE:

Defition:

It is a complete or incomplete break or crack in the continuity of a bone or

A structural break in the normal continuity of bone

The fracture may also occur through cartilage, epiphysis, and epiphseal plate.

Dislocation:

- A partial disruption of a joint with partial remaining, but abnormal contact between the articulating plates. The articular surfaces are nolonger in contact.

Sublaxation:

- This is a partial dislocation. Some of the articular surface is in contact, the congruence of the two joints has been lost.

Mechanism of injury: sufficient violence is required to cause a fracture of normal bone, but trivial (minimal) trauma can cause fracture in a fragile bone. The violence can be;

- Direct- when violence is applied directly and causes fracture at site of violence. The bone breaks at the point of impact and soft tissues are also damaged. A direct bow usually causes a transverse fracture and damage to overlying skin.
- Indirect – violence is transmitted to distant part. The bone breaks ata distance from where the force is applied for example when the head of the radius is fractured in a fal on an outstretched hand or fracture clavicle in a fall on the shoulder.

Tubular bone may be broken either by direct or indirect violence. Cancellous bone may be fractured either by compression or by tension. May compression fractures may occur if a pt falls from a height and lands on his heels. Traction injuries only occur in cancellous bones to which a ligament or tendon is attached for example medial malleolus.

Periosteum:

A dense fibrous tissue covering a bone. It is connected to marrow space with connective tissue through volkmann's canals. It supplies bld to bone and stimulates osteogenesis. It has two layers, thu fibrous (outer) and cellular (inner). In children the periosteum is thick, loosely attached to cortex and produces new bone rapidly. In adults it is thin, adherent to the cortex and produces new bone less rapidly. For these differences healing of fractures in children is rapid.

PAHOLOGY OF FRACTURES AND FRACTURE HEALING:

A fracture can be;

Simple (losed) – The overlying skin remains intact, the bone is incompletely divided and the periostuem in continuity. This is what happens in greestick and compression fractures.

An open (compound) fracture -- the skin or one of the body cavities is breached and is liable to contamination and infection.

Classification of fractures:

Classification of fractures is wide and varied. It may take any of the following the modes;

- Classification by quality of bone in relation to load;

Here the fracture occurs when the load to which they are subjected exceeds their intrinsic strength.

- Simple fracture which is produced when an excessive load is applied to normal bone.
- Pathological fracture which is produced when the strength of the bone is reduced by disease. In this case a force which is within normal limits leads to a fracture as in osteoporosis and metastasis of tumours.
- Stress fracture in which is fatigued and then breaks due to continous mechani cal stress as in people playing high levelsport.
- Partial or greenstick. Bones in young people are flexible. Th ey bend and may buckle or partially break, instead of breaking cleanly when overloaded.

- Classification by direction of force;s
  - Compression fractures. The load applied along the length of the bone exceeda that of its strength then it may collapse into itself. This is common in the elderly due to osteoporosis
  - Avulsion or distraction fractures. Here two fragments of bone are pulled apart. They are common where srong muscles insert into small bones. Examples are the patella (the quadriceps), olecranon(triceps).
  - Spiral fractures. When a long bone is twisted along its axis
  - Transverse fractures. When a long bone is bent along its axis
  - Butterfly fractures. If a bone is struck by a direct blow, a more complex fracture may follow where two break lines spread outward from the point of contact of the blow, producing a free floating “butterfly” fragmentbetweenthe two fractures
  - Comminuted fractures. They occur when a large amount f energy is dissipated (unrestrained) into a bone. The bone breaks into fragments which may impact into each other or separate and become displaced.
- Classification by anatomical site

A long bone is divided into three main zones;

- The diaphysis which is the marrow part of the shaft. It has a thick cortex and a medulla filled with trabecular bone
- The metaphysic the part which flares at each end between the diaphysis
- The epiphyseal plate. In infants and children, in whom the bones are still growing, the epiphyseal plate will be open. The plate is weaker than the bone around it and so fractures tend to track along it or even across it.epiphyseal fractures are important because they can have a poor prognosis.

A. Etiological

i) Traumatic fracture (a single traumatic incident). It is due to sudden injury and is by far the largest group and may be direct or indirect. A simple traumatic fracture occurs when excess load is applied to a normal bone.

ii) Pathological fracture: The term is applied to a bone already weakened by disease. Often the bone gives way from trivial violence or even spontaneously. It is produce when the strength of the bone is reduced by disease. In this cause the force which is within normal limit leads to a fracture. The disease could be generalized osteoporosis or a localized lytic lesion from metastasis.

Causes;

4. Infection

- pyogenic osteomyelitis usually the chronic form.

- syphilitic infection (syphilitic metaphysitis).

> Benign tumours

- haemangioma

- chondroma

- giant cell tumours (osteoclastoma)

> **malignant tumours**

- osteosarcoma

- Ewing's tumour

- Solitary myeloma

- Metastatic carcinoma(from the lungs, breast, thyroid, kidneyand prostate)

> Miscellaneous

- Simple bone cyst

- Monostatic fibrous dysplasia

- Bone atrophy in paralytic conditions such as polio

- Tabes dorsalis

- Eisinophilic granuloma

- Brittle state after irradiation

General affection of the skeleton

7. Congeneital disorders like osteogenic imperfecta
8. Diffuse rarefaction as in senile osteoporosis, cushing syndrome, infantile rickets, uraemic osteodystrophy (renal rickets), cystinosis (fanconi syndrome) and nutritional osteomalacia.
9. Miscellaneous – including pagets disease, polyostotic fibrous dysplasia, Gauchers disease and Hand-Schiiler-christian dsease.

iii) Stress (fatique) fractures: This is due to repeated trauma at same site. The bones are subjected to a large number of loads, none of which could be enough to break the bone . The mechanical structures of the bone can gradually fatique and the bone will then break. Cracks cann occur in bone, as in metatarsals and attached materials due to repeated stress. This is commony seen in tibia, fibular metatarsals and neck of femur especially in athletes dancers army recruits who go on long route matches with a few exceptions. This fractures are usually confined to bones of lower limbs with a great majority occurring in the metatarsals.

B. Clinical

i) Simple or closed
ii) Compound or open
iii) Complicated – the nerves, vsls and vital organs are affected.

C. Radiological

i) Transverse fracture due to angulation force
ii) Oblique fracture due to twisting force
iii) Spiral fracture due to twisting force
iv) Comminuted fractures – more than 2 fractures
v) Avulsion fractures- a small fragment pulled and shifted by muscles
vi) Impacted fracture – force a long axis of bone e.g. neck of femur or humerus
vii) Burst/ shattered – multiple small fragments with separation.

D. **In children (radiological**)

i) Epiphyseal separation
ii) Epiphysealfracture separation
iii) Greenstick fracture

m) fracture of one cortex and bending of the appropriate cortex

iv) Buckle fracture – cortex is punched out by force along a long axis.

Site of fracture:

Other than recording the actual bone which is fractured it is necessary to describe the situation within the bone. The fracture may be at an end of the bone in which case it will involve the joint in which the bone articulates. These are known as articular fractures. Such fractures in children are known as epiphyseal fracture.

Displacement:

It refers to the deformity which may be present following a fracture or dislocation. It describes the position of the distal component relevant to the proximal component. The causes of deformity include;

Initial force

Gravity

Effects of contraction of muscles attached to the fractured fragment.

Movement of the pt during administration of first aid, transport to hospital and during the cause of initial assessment is also an important cause of deformity. This can be diminished by early application of temporary splintage. The fragments may be shifted sideways, backwards or forward in relation to the other fragment, such that the surfaces lose contact. The fracture heals even if apposition is imperfect or even if the bone ends lie side by side without making contact with the fracture surfaces.

Types of displacement:

1. Shift – There is loss of alignment of the cortices of the shafts
2. Angulation - Loss of normal longitudinal axis of the shaft.
3. Shortening – There is overlap of the bone fragments but may result from impaction of one fragment onto the other.
4. Twist (rotation) – Rotation of the distal fragment around the long axis of the bone, either external or internal.
5. Distraction – Prolonged by overvigorous traction during treatment.

Fracture healing:

It starts to heal as soon as the bone is broken. Repair of tubular bones occurs in five stages;

i) Stage of haematoma formation

ii) Stage of periosteal and endosteal cellular proliferation

iii) Stage of callus formation

iv) Stage of consolidation
v) Stage of remodelling

(a) Stage of haematoma formation:

When the bone is fractured bld seeps out through torn vsls and forms a haematoma between and around the fractured surface.

The haematoma is contained by the surrounding soft tissue – periosteum and muscles which may be stripped up from the bone end to a variable extent.

The fracture divides most of the capillaries that run longitudinally in the compact bone, a ring of bone adjacent to the side of the fracture becomes ischaemic over a variable length, but usuall few mm, deprived of bld supply the osteocytes near the surface die.

(b) **Stage of periosteal and endosteal cellular proliferation**:

In the early stages there is proliferation close to the fracture

These cells or precursors of the endoblasts which will later tay down the intercellular substance

They form a collar of active tissue that surround the fragments

Simultaneously(at the same time)with the subperiosteal proliferation there is cellular activitywithin the medullary canal, where the proliferating cells appear tobe derived from the endosteum and th e marrow tissue of the fragment. This tissue grows forward to meet and blend with similar tissue growing from the other fragment.

(c) Stage of callus formation:

As cellular tissue that has grown out from each fragment matures, the basic cells give origin to to osteoblast, chondroblasts which form the cartilage.

The osteoblasts lay down intercellular matrix of collagen and polysaccharide which soon become impregnated with calcium salts to form the inactive bone of fracture callus. This from its tyexture has been termed woven bone

The formation of this bridge of woven bone imparts obvious rigidity to the fracture and when an injured bone is a superficial one the callus may be felt as a hard mass surrounding the fracture.

The mass of a bone is also visible in the radiographs and gives the 1st radiological evidence of bone union.

(d) Stage of consolidation:

The woven bone that forms the primary callus is gradually transformed by activity of the oateopblasts into mature bones which has a typical lamella structure.

(e) The stage of remodeling:

When union is complete, the newly formed bone forms a bulbus collar which surround the bone and obliterates the medullary canal

Callus is usually profuse in children because the periosteum is easily stripped from the bone by extravastated bld, allowing bone to form beneath it.

In the months that follow, union is strengthened along the lines of stress which is slowly removed and reabsorbed elsewhere and the bone is thus restored more or less to original form. The process of remodeling is going on constantly, but inconspicouslyin every bone throughout life but it becomes especially obvious after a fracture.

Clinical and radiological features of fractures:

a) **History** –

A statement that the pt is unable to stand or walk after an injury or to use the injured part should arouse suspicion of a fracture.

A history of visible bruising appearing a day or so after an accident is also suggestive.

In fatique or pathological fractures there may be spontaneous onset of pain and disability without any causative agent

And incase of a malicious injury to a baby an accurate account is deliberately withheld

Caution:

A clinician is often misled to belief that no fracture exists because the pt has retained the use of painful limb in certain causes function is preserved despite the fracture such fractures include; fatique fractures, impacted fractures, fractures of small bones.

When common sites for these fractures are borne in mind, diagnostic mistakes can be avoided. Impacted fractures of the neck of the femur, neck of humerus and lower end of the tibia.

Fatique fractures are seen in the 2nd and 3rd metatarsal, fracture of tibia, fibula. Fractures of carpal bones like the scaphoid and greenstick fractures of the forearm

b) Clinical examination:

The following features are fairly constant findings;

(i) Is there a wound communicating with the fracture
(ii) Is there any impairement of circulation distal to the fracture
(iii) Is there any evidence of nerve injury
(iv) Is there any evidence of visceral injury

Some positive findings above may make a case a surgical emergency or influence the outcome of treatment.

NB: The presence of skin laceration does not necessarily mean that the fracture is open.

State of circulation:

The part of the limb distal to the fracture must be examined for evidence of circulatory impairement.

The examination should be repeated frequently in the 1st 48 hrs after a fresh fracture that has been immobilized in a p.o.p or that has been operated on.

Severe pain within a P.O.P or markedswelling of the digits should arouse suspicion and therefore the following tests should be done;

1. Colour:

Pink colour is reassuring

A blue, grey or white colour should arouse suspicion.

2. Warmth:

Warm digits suggest circulatory flow

Cold digits do not necessarily cause alarm especially if the limb is encased in a fresh P.O.P that is still damp.

3. Arterial pulses:

It is a reliable guide to the state of circulation

Where necessary the pop should be trimmed sufficientlyto allow access to pulse

In compartment syndrome in which tension from oedema and haematoma within a closed fascial compartment in the forearm/leg builds up to the extent that the viability of contained tissues become impaired, the pulse may be present. In such case severe and unremitting pain is an important clue to the the compartment syndrome.

When digital bulb or a nailbed is compressed with a finger nail, and blanching can be seen around the point of pressure. If on release bld flows back briskly into the blanched area in a pink flush means that circulation is adequade.

4. Nerve conductivity:

An ischaemic nerve quickly loses ability to transmit impulses

Loss of sensibility in the digits in the absence of physical injury to the nerve suggests ischaemia

In deciding whether sensory impairement is caused by trauma to a nerve or by ischaemia it should be remembered that in ischaemic lesions all nerve trunks in the limb are likely to be affected. Whereas it is unusual for all nerve trunks to be involved in an injury.

Total sensibility of a hand or foot suggests ischaemia, whereas insensibility in the territory of a single nerve denotes mechanical injury.

NB: Motor tests are less valuable than tests of sensibility because the long extensors and flexors are innervated high up in the forearm and leg and will continue to move the digits despite complete ischaemia in the distal part of the limb.

Specific tests:

Incase of doubt the clinical tests about the intergrity arterial circulation;

1. Doppler ultrasound or arteriography

State of the spinal cord and peripheral nerves: Simple tests of sensibility, motor function, sweating is sufficient to indicate whether or not there has been an injury to the nervous system or cauda equine.

State of the viscera: - Investgate the state of bladder/ urethra in every fracture involving the anterior part of the pelvis.

MANAGEMENT OF FRACTURES:

At the hospital;

Assessment is required to determine

Whether there is a wound communicating with the fracture

Whether there is evidence of nerve injury

Whether there is evidence of vascular injury

Whether there is evidence of visceral injury

Resuscitation;

Many pts with severe or multiple fractures are shocked on arrival at hospital

Time must be spent on resuscitating the pt before definitive treatment of the fracture is begun

The mainstay of antishock treatment is immediate replenishment of the circulating blood to restore a normal blood volume and electrolyte balance, clear airway and pulmonary ventilation.

Treatment of uncomplicated closed fractures;

The 3 fundamental principles of fracture management are:

1. Reduction
2. Immobilization
3. Preservation of fuction (rehabilitation)

A. **REDUCTION**:

This 1st principle must be qualified with the words

if necessary because in many fractures reduction is unnecessary

If judged that perfect function can be restored in some of the uncorrected displacement of fracture fragments, there is no object in striving for perfect anatomical reduction for example broken fragments of a child's clavicle and the same applies to most fractures of the clavicle in adults.

In general imperfect apposition of fragments may be acceptable than imperfect alignment e.g. in the shaft of the femura loss of ½ a diameter may be acceptable whereas an angular deformity of 20degrees would demand reduction.

Methods of reduction:

1. By closed manipulation
2. By mechanical traction with or without manipulation
3. By open reduction

Manipulative reduction:

Closed reduction is usually done under GA but local or general anaesthesia is appropriate.

The technique is to grasp fragments through soft tissues to disimpact them if necessary and then to adjust them as nearly as possible to correct position.

Reduction by mechanical traction:

The contraction of large muscles exerts a strong displacing force, mechanical aid is necessary to draw the fragments out to the normal length of the bone especially fractures of the shaft of the femur and to certain types of fracture displacement of cervical spine.

Traction may be applied by wt (or by screw devices) (fixed traction)

The aim is to gain full reduction GA or gradual reduction by prolonged traction without anaesthesia. The wt applied is 10% of the body wt.

Operative reduction:

This is indicated when other methods failand occasionally as a method of choice , the fragments are reduced under direct vision at open operation.

When operative reduction is restored the fragments are fixed internally to ensure their position is maintained.

B. IMMOBILIZATION:

Like reduction this second great principle must be qualified by the words " if necessary"

Whereas some fractures must be splinted rigidly there are many that do not require immobilization to ensure union and there are some in which ecxcessive immobilization is harmful.

Indications for immobilization;

1. To prevent displacement or angulation of fragments
2. To prevent movement that might interfere with union
3. To relief pain

Methods of immobilization:

1. By P.O.P or other external splints
2. Continous traction
3. By external fixation

a. Immobilization by pop, splint or brace

In most fractures the standard method of immobilization is by pop cast

For some fractures a splint made from metal, wood or plastic is more appropriate e.g Thomas splint for fractures of the shaft of femur or plastic collar for certain injuries of the cervical spine.

Caution – When pop is aaplied on a fresh fracture or after operation on alimb, always monitor circulatory impairement or undue swelling within the pop.

Arterial supply to the distal part of the limb

The period of greatestdanger is between 12-36hrs after the injury or operation.

Severe pain within the plaster and marked swelling are warning signs calling for a careful reassessment of peripheral circulation

Take note hat after the operation upon the limbs a coagulated blood soaked dressing may act in exactly the same way as a tight plater and may seriously obstruct the circulation

Cast bracing (functional bracing):

A brace is a supportive device that allows continued function of the part, in this case a fractured long bone (e.g. # of the femoral shaft or tibia)is supported externally by pop or by mouldable plastic material in such a way that the function of adjacent joints are preserved and use of the limb for its normal purpose can be resumed. The technique entails snug fitting of the plater or plastic material over the appropriate limb segment incorporation of hinges at the level of adjacent joint. This procedure is carried out the fracture is already becoming "sticky" usually 5-6/52.

b. Immobilization by continous traction:

In some fractures especially those of femoral shaft, tibia, lower humerus may be difficulty o r impossible to hold the fragments in position by pop or external splintsalone.

In such case the pull of muscles must be balanced by continous traction upon the distal fragment either by a wt or other mechanical device.

Continous traction is often combined with some form splintage to give support to the limb against deformity – usually a Thomas splint or modified Thomas splint in case of a femoral shaft fracture, Braun's splint in case of tibia fracture.

The Gallow's or Bryant method of traction for fracture femoral shaft in young children embloy principle of immobilization by traction without any additional splintage (children up to the age of 3yrs)

Also in this category is traction upon the skull for cervical spine injuries.

c. **Immobilization by external fixation**:

This is rigid anchorage of bone fragments to an external device such as a metal bar through a medium of pins inserted into the proximal and distal fragments of a long bone # (external fixator).

External fixator finds its main application in the management of open fractures or infected # where the use of internal fixation devices such as plaster or nails is undesirable because of the risk of promoting or exacerbating infection.

It may be used in the treatment of certain closed #s of long bones as an alternative to internal fixation

Immobilization of internal fixation:

May be adviced in the following circumstances;

1. When it impossible in a closed # to maintain an acceptable position by splintage alone or in combination with traction.
2. When it becomes necessary to operate upon a # to secure adequate reduction
3. As a method of choice in certain #s to secure rigid immobilization and allow earlier mobility of the pt.

Methods used;

a. Plates held by srews
b. Bone graft held by screws
c. Intramedullary nail
d. Compression screw plate
e. Nail-plate (combined nail and plate)
f. Transfixion screws
g. Circumfrential wires and bands
h. Suture through attached soft tissues

TEST OF UNION OF FRACTURES:

A. Clinical test;

There are 3 clinical tests of union, i.e, absence of

i) Mobility between # fragments
ii) Tenderness on firm palpation over the site of #
iii) Pain when angulation stress is applied at the site of the #

These tests together are reliable but should always be confirmed by radiological studies.

B. Radiological criteria of union

They are two, i.e,

1. Visible callus bridging the # and blending with both fragments
2. Continuity of bone trabeculaeacross the fracture

Visible callus in generally early and more reliable

Trabecular continuity across the # is evidence of mature union.

Principles of fracture treatment:

Initial management (first aid) at the scene of the accident;

1. Ensure clear airway
2. Cover any wound with clean dressing
3. Provide some form of immobilization
4. Make the pt comfortable while awaiting for hospital help.

Complications of fractures:

They are considered in two groups thus;

1. Those involving the fracture itself (intrinsic group)
2. Those attributable to associated injury involving other tissues (extrinsic group)

Intrinsic group:

i. Infection
ii. Delayed union
iii. Non union
iv. Avascular necrosis
v. Mal – union
vi. Shortening

Extrinsic group:

i. Injury to major blood vessels
ii. Injury to nerves injury to viscera
iii. Injury to tendons
iv. Injury and post traumatic affection of the joints
v. Fat embolism

A. Infection;

Usually confined to open #s, the wound is contaminated by organisms carried in fromm outside the body

Closed fractures may become infected when it is converted into an open fracture by operative intervention

More often the infection extends to the bone giving rise to osteomyelitis. This is a serious complication because infection by pyogenic organisms tends to become chronic.

Part of the bone may die due to impairement of blood supply forming a sequestra.

Infection is a potent factor in delaying or preventing union

Treatment:

In acute recent infection;

Provide adequate drainage

Antibacterial indication

The wound is left open to eliminate potential pockets of pus by appropriate incision or excision of tissue

Between dressings the limb is immobilized in POP with external fixation

When the infection is overcome and wound becomes lined by healthy granulations, wound closure may be attempted by secondary suture or skin graft

Antibiotics e.g flucloxallin + fusidin acid, erythromycin + fusidic acid. The choice antibiotics depends on c/s

In chronic osteomyelitis, pus continous to discharge and fragments of bone may die and separate as sequestra. All sequestra must be removed and bone that is honey comb with small pus containing with muscle flap or in case of a superficial bone such as the tibia by lining the saucerized cavity with skin graft direct to the raw bone by a cancellous bone graft from the iliac bone.

B. Delayed union

There is no absolute time beyond which a # in a delayes union are freely mobilized 3-4 months after the injury. If a state of delayed union persists for many months, it eventually passes into a state of mal- union. The distinction between delayed in the condition of the bone to indicate that union will fail altogether. In non union characteristic changes are observed, radiologically which suggest union will never occur.

Causes of delayed union: the causes are like those of non union but acting in a less degree

Treatment:

Treatment is expectantant at 1^{st,} for one hopes will eventually without surgical intervention

6 months or more union doesn't appear to be progressing surgical, treatment must be considered, usually bone grafting operation offers the best prospect of promotinunion.

C. Non union:

When fracture remains ununited for many months distinctive radiological changes take place which indicate apermanent state of mal- union, the bone ends at the site of the fracture become dense and rounded and the rounded # line is a clear cast. Pathologically the healing process appears to have come to an end , there is no attempt to bridge the # with callus, the gap between the bone fragments ius filled with fibrous tissue, in some cases a cavity may form in the fibrous bridge , Suggesting an attempt to for a false joint (pseudo arthrosis)

Causes of non union;

1. Infection of bone
2. Inadequate bld supply to one or both fragments
3. Excessive shearing movement between the fragments.
4. Interposition of soft tissues between the fragments
5. Loss of apposition between the fragments (including over distraction by traction apparat us)
6. Dissolution of fracture haematoma by synovial fluid (in fractures within joints)
7. Presence of corroding metal in the immediate viscinity of the fracture
8. Pathological fractures (destruction of bone as by tumour)

NB sometimes two or more of these factors may act together. Acting in slight degree, some of these factors may be responsible for delayed union.

Treatment of delayed union;

Depends on the site of the fracture and the degree of the disability

If the disability is slight, it is best left untreated e.g. fracture of scaphoid bone.

When surgical treatment is desirable most ununited #s of long bones tend to respond well to treatment by bone graft

In certain #s within or near a joint excision of one of the fragments or its replacement by a prosthesis is appropriate.

D. Avascular necrosis

This is death of bone from deficient bld supply. It may cause intractable non union, disabling osteoarthtritis, total disorganization of a joint.

Pathology:

AVN occurs when bld supply to a bone or part of abone is interrupted by injury (or rarely by disease). It may occur as a complication of a fracture articular surface where terminal fragment is devoid of vascular tissue attachment and depends for its nutrition almost entirely upon the interosseous vsls which may be torn at the time of injury, dislocation if vital bld vsls supplying the bone are torn or occluded.

The immediate consequence of ischaemia is that the bone cells die and if the part affected is within a joint cavity, there is little chance that it can be revascularized from surrounding tissues before irreversible changes occur.

The avascular bone loses its rigid trabecular structure and becomes granular; in this state the bone crumbles easily and under stress imposed by muscle tone or body wt and eventually collapses into an amorphous mass.

In most cases a joint whose surface suffers AVN is doomed to crippling osteoarthritis whether or not fractured eventually unites.

Sites:

1. Head of femur after the # of the femoral neck or dislocation of the hip.
2. Proximal ½ of the scaphoid bone after # through the waist of the bone.
3. Body of the Talus after # through the neck of the Talus
4. Lunate bone

Diagnosis:

Radiographs after 1-3 months after injury

Radioisotope scanning with 99cm technetium. The affected part is devoid of bld vsls, the isotope is not taken up and the avascular bone may be shown as a void area.

Treatment:

AVN demands early operation because of the likehood of disorganization of adjacent joints; excision of the fragment is undertaken.

If necessary reconstruction of the joint by some form of arthroplasty or stabilize it by arthrodes.

E. Mal- union:

This is imperfect position of union of the fragments.

The union may be by angulation, rotation, loss of end to end apposition, overlap and consequent shortening.

Treatment:

Each case is considered on its merit

Slight deformity may be acceptable without treatment in others correction of deformity by refracturing or dividing the bone and after correction, fixing the fragments by appropriate means.

F. **Shortening:** Arises from 3 causes;

a) Malunion- union of fragments with overlap or marked angulation
b) Crushing or actual loss of bone as in severely comminuted compression #s or in gun shot wounds when a piece od bone is shot away.
c) In children, interfering with growing epiphyseal plate. As a rule epiphyseal growth plate will be impaired by crushing injury than by avulsion injury with a fracture separation of the epiphysis.

Shortening is important only in lower limbs. Shortening of up to 2cm is not significant and may not be noticed. If more than 2cm it should be correcte by appropriate raising of the shoe or by a leg shortening operation on the opposite side.

Uncorrected shortening may cause aching in the back from tilting of the pelvis and consequent scoliosis.

G. Injury to major bld vsls:

A fracture can result to injury of adjacent tissues e.g. muscles, fair and minor bld vsls, but results to spontaneous healing of the #

Sometimes an important artery may be damaged by either the agent causing the fracture or by the sharp edge of the bone fragment.

Serious complications that may lead to loss of the limb

The vsls may be torn, occluded by thrombosis, contused or may merely be temporarily sealed by aneurysm.

The effect may be;

1. Traumatic aneurysm
2. Impaitred bld supply leading to gangrene; ischaemic paralysis of the nerves or contractures of muscles (volkman's ischaemic contracture)

NB: vascular occlusion may be caused by tissue edema within closed fascial compartment or by overtight plaster or bandage especially 2/7 after injury or operationwhen swelling reaches its peak.

Examples;

Axillary artery- # dislocation of the shoulder

Brachial artery- # supracondylar of the humerus and dislocation of elbow

Politeal artery- dislocation of the knee and displaced # of upper end of tibia.

Rupture of medial meningeal artery from # of temporo-parietal region of the skull also comes in this category.

Clinical features:

1. Peripheral circulation (after # of a long bone)
- Ischaemia – pain on attempted extension of toes and fingers
- Numbness and sensation of digits

Treatment:

Should be immediate before ischaemia which is irreversible

Occlusion can be primary or secondary(after reduction or immobilization).

1st step:

Remove the bandage. Gross displacement of fragment, if not reduced, gentle manipulation is made.

If adequate circulation is not achieved within ½ hr the next step is taken.

2nd:

Damaged artery is exposed and nature of injury determined

If the occlusion is kinking or spasm of artery an attempt is made to free the vsls, by applying papaveritne (an arterial relaxant)

Heparinized saline may be injected between the clamps

If the vsl is divided restoration of patency using freshened end sutures is made

If the occlusion of a thrombi following damage to the intima then end arterectomy with repair using a vein can be made.

H. Compartment syndrome:

Miscles are enclosed within a fascial compartment. This is swelling occurring within a compartment as aconsequence of a injury and a viscious circle isset up;

The swelling occludes smaller arteries or veins supplying the muscle

Muscle becomes ischaemic and thus promotes swelling

Within a few hrs irreversible changes occur; muscle becomes necrotic and nerves in the compartment lose their conductivity because of ischaemia.

The muscles are eventually replaced by fibrous tissue threatens to produce contractures (volkman's ischaemic contracture) seen in most flexor muscles of the fore arm and leg.

Compartment syndrome shows that peripheral pulses may be present and this may deflect the unwary from the true diagnosis.

Treatment:

It demands immediate operation to decompress the whole length of the affected compartment by fasciotomy.

I. Injury to nerves:

Peripheral nerves are injured moreoften than major arteries

Common sites are;

- Brachial plexus (forcible depression of the shoulder)
- Circumflex nerve (shoulder nerve)
- Humerus (radial nerve)
- Ulnar nerve (medial epicondyle)
- Spinalcord (cervical/ thoracic spine)
- Cauda equine (lumbar spine)
- Sciatic nerve (hip dislocation with #ed acetabulum)
- Common peronial nerve (knee especially the lateral ligament torn)

Nerve injuries were classified by seddon into 3 types;

i. Neuropraxia

Damage is slight and causes only transient physiological block

Recovers spontaneously within a few wks

ii. Axonotmesis

Internal architecture of the nerve is preserved

Axons are badly damaged that peripheral degeneration occurs

Recovery occurs but depends upon regeneration of axons and may take months (2-3cm per month.

iii. Neurotmesis

Structure of the nerve is destroyed by actual division or severe scarring

Recovery is possible after excision of the damaged section; with end to end suture of stumps orbridging by nerve graft.

Treatment:

- Expectation especially in closed #s but recovery is not observed within the expected time exploration is made; so that repair can be made.

- In open #s with suspect that the wound is severed then the ends are tackled together and definitive repair made. In most cases a nerve repair is postponed until a wound healed. Best time is 3-4 wks after injury.

J. Injury to viscera:

Caused by the agent causing the injury or implement upon a sharp fragment of bone

Examples are;

- Laceration of pleura
- Lung complicating # of the ribs
- Rupture of the bladder or urethra
- Penetrationof colon or rectum
- Complicating # of the pelvis
- Rupture by direct trauma to the trunk without #

Treatment:

Follow general surgical principles

Inury to tendons:

In open fractures tendons may be severed by the agent causing the fracture

Injury to the joints:

Acute joint injuries such as dislocation, sublaxation or ligamentous strain are common complications of fractures.

Post traumatic ossification:

Also known as myositis ossificans

It is a rare cause of joint stiffness after # or dislocation

Occurs in case of severe injury of a joint especially when the capsule and periosteum have been stripped soft tissues, forming a large haematoma about the joint

Instead of being absorbed it is invaded by osteoblasts and becomes ossified

If a large mass is formed, then restriction of joint movement is severe

It is a complication encountered mostly in the elbow after # dislocation, also in the hip dislocation

There is risk of its occurance in children than adults because children the periosteum is only loosely attached to the long bones and easily stripped from them.

High tendencies is also in the lowerlimbs in pts with prolonged or permanent brain damage from head injury and in pts with paraplegia from spinal injuries

Treatment:

Minimize the formation of haematoma by enforcing complete rest from the joint, preferably in plaster for 3-4 wks

Treatment should consist of active excercises with avoidance of strains or stretching that might provoke further bleeding beneath the soft tissues

After several months it may be necessary to excise a mass of bone that is blocking movement but the operation is not successful but must be done with caution.

Osteoarthritis (degenerative arthritis):

Any roughening or irregularity of a joint surface is liable to precipitate the wearing and tear changes that form the basis of osteoarthritis

It involves an articular surface unless the fragments are replaced so perfectly in that thee smooth contour of joint surface is unimpaired

Even in slight step between fragments may lead to serious subsequent disability from arthritis especially in wt bearing joint

Avascular necrosis is a cause of severe osteoarthritis or of total disorganization of a joint.

If fragments unite with angular deformity, throwing the joint out of its correct alignment, because of mal-alignment of joint surface causes excessive stress at one part of the joint and consequently accelerates wear and tear changes thus creating a high risk of osteoarthritis.

Examples of circumstances above include knee after mal-union of # of the femoral shaft.

The risk of osteoarthritis is higher in wt bearing joint of the lower limb.

After severe damage in a joint, osteoarthritis may become clinically evident within 6-9 months of the injury whereas in slight damage or mal-alignment it may become apparent for 15-20 yrs.

FAT EMBOLISM:

It is uncommon

A serious complication of #

The essential feature is the occlusion of small bld vsls by fat globule

Pathology:

More significantly affects the lungs and the brain

In the lungs there is oedema and haemorrhages in the alveoli, so that transfer of oxygen from alveoli to arterioles is impaired

This thus leads to hypoxaemia which may be severe

In the brain there may be multiple petechial haemorrhages which occur in other organs and the skin

Clinical features:

Occurs after severe fractures in the lower limbs particulary the tibia and femur

Onset is within 2 days of injury but note that there is a symptom free period between injury and onset an important part of distinction of cerebral contusion

The presenting feature of breathlessness, usually associated with cerebral disturbance in the form of marked restlessness, contusion, drowsiness and coma.

The above syndromes may be caused partly by petechial haemorrhage in the brain but in large measure they are secondary to hypoxia from occlusion of small vsls in the lungs. Associated features are tarchypnoea and dyspnoea

Another clinical manifestation include petechial rash usually infront of the neck, anterior axillary fold or chest or in the conjunctiva

The finding of such a rash strongly supports a diagnosis of fat embolism syndrome.

Diagnosis:

Arterial bld gas analysis which may show reduction in partial pressures of oxygen in the bld vsls (often below the critical level of 60mmhg at which respiratoy failure is likely).

Treatment:

Spontaneously ,reversible if the pt can be handled over the dangerous period of hypoxia which may be corrected by administration if necessary with positive pressure respirations

The oxygen requirement should be controlled by repeated bld gas analysis

The administration of methylpredinisolone in pts with severe multiple injury may help toprevent and correct adverse effects of fat embolism by maintaining bld oxygen tension and stabilizing the free fatty acids

Heparin or dextran 40 may also be administered intravenously to improve capillary flow.

AMPUTATION:

Definition:

Indications: Ablation of a limb is an extreme step and an irreversible operation and every care should be

Amputation should be considered only if the limb is;

Dead (gangrenous),

Dying (grossly ischemic),

Dangerous (due to malignancy) or

Dud (useless limb)

Common indications for amputation are;

Traumatic conditions: Crash injuries to the limb

Vascular conditions: Ischemic conditions of limbs;

Thromboangitis obliterans (buerger's Disease)

Arteriosclerosis: Senile and diabetic

Gangrene: Dry and Moist

Neoplastic conditions: Malignant tumors of bones or soft tissues (Osteosarcoma, Synoviosarcoma)

Infective conditions:

Leprosy: When the leg is totally useless and grossly destroyed

Actinomycosis of the foot or hand

Filarial elephantiasis

Congenital conditions: when the limb is grossly deformed and useless.

Types of amputations:

Guillotine amputation: This is an emergency amputation done as a life saving measure. It is done in cases of gross crush injuries of the limb. It is also indicated in cases of gas gangrene, when a rapid removal of the dangerously infected part is a life saving procedure. In Guillotine amputation the incision is circular around the limb at the site of bone section and all the wound is cut at the same level and the wound is left open to provide free drainage.

Classical amputation: These are planned amputations where regular skin flaps are raised and the wound is closed after ablation of the limb.

Revision amputation: They are done;

As a second stage in guillotine amputation

To those with very unsatisfactory stumps following a previous amputation.

Selection of level of amputation: The classical sites of amputation of limbs are determined on the basis of the following considerations;

The disease process for which the amputation was done to eradicate the pathology.

The vascular supply to the skin flaps.

The requirement of limb fitting procedures and techniques available at that time.

Radiography of the part is done to see the extent of the malignant disease. In some cases arteriography may be used to assess the vascularity of the limb and level of viability.

Levels of amputation:

Lower limb;

Hind quarter amputation

Hip disarticulation

Above knee amputation

Through knee amputation

Below knee amputation

Syme's amputation

Fore foot amputation

Toe amputation

Upper limb;

Foerquartet amputation

Shoulder disarticulation

Above elbow amputation

Elbow disarticulation

Below elbow amputation

Wrist disarticulation

Finger amputation

Basic principles in amputation;

The following steps are applicable to all levels of amputation on the limbs;

Mark out and raise appropriate skin flaps.

Cut muscles and soft tissues 1/2@" proximal to skin section.

Cut bone 1" proximal to skin section.

Identify blood vessels and ligate.

Cut nerve ends and allow them to retract.

Remove tourniquet and obtain hemostasis

Suture the muscles and cover the bone end

Place a drainage within the wound

Suture deep fascia and skin.

Apply dressing and compression bandage

Provide a splint to rest the stump

Stump and its management:

The stump is the residual part of the limb left after the amputation. It should be just an anatomical residue but should be an active motor organ to move the prosthesis and also give some sensory

feedback. In the lower limb amputation, the function to be restored are wt bearing, locomotion and sensory feedback. The wt bearing could be (a) end bearing, (b) side bearing or (c) proximal bearing.

A good stump should be neither too long nor too short. It should have good muscle power with full movement in the proximal joint and a healthy non adherent scar. It should a fleshy end with no bony spurs. The care of the stump consists of;

Stump bandaging with crepe bandage to improve its shape for limb fitting.

Stump exercises to improve its motor power and movements in the proximal joint.

Stump hygiene to maintain the skin and scar in good condition.

Complications of amputation;

Immediate:

Infection

Secondary hemorrhage

Skin sloughing.

These are preventable by control of infection and proper technique in suturing.

Late:

Stump neuroma

Phantom limb

Contractures

Neuroma: This is the development of a bulbous swelling at the cut nerve end. It is tender and causes pain on wt bearing. Pain may be relieved by local hydrocortisone injection or in some cases by ultrasonic therapy.

Phantom limb: In this condition the pt feels that the limb is still present and he may feel even pain in some areas in the non existent foot or toes. It usually clears up with reassurance, analgesics, stump exercise and regularity in the use of prosthesis.

ORTHOPAEDICS:

Unit 1: conditions of the soft tissues, bones and joints.

Congenital deformities:

1. Talipes equino varus/valgus (club foot)

Definition: a congenital deformity that presents as a club foot i.e inward twisting (inversion), adduction of the forefoot and plantar flexion (equino). It is common in boys than girl

Causes;

It appears to be a combination of geneticand environmental factors (intrauterine positioning)

Is associated with hypohydromnious or polyhydromnious

Clinical features:

n) Common in boys trhan girls

-Shortening of tendo-Achilles

Calcaneous is smaller and points downwards

The heel doesn't step down and there is inversion of fore foot

Internal rotation of the tibia

Wasting of the cuff muscles due to disuse

Shortening and thickening of soft tissues especially the Talo-Navicular capsule which will give riise to Navicular dislocating medially

Diagnosis:

At deliverly, hold foot of baby in neutral position and evert and dorsiflex it. Normally the foot can touch the shin bone. If there is antagonism in this movement then a diagnosis is made.

Treatment:

Should be started as early as possible to avoid later complications.

In mild cases the correction should be made by 6/12.

Passive manipulations under nothing

In moderate cases, manage ment is by manipulation and splinting under local anaesthesia and splint in Denis Browne splint or P.O.P. leava it for 1/52, remove P.O.P, manipulate and reapply the p.o.p. later continue at every 2/52 up to 6/52.

Metatarsal varus is corrected by stabilizing the talus and abducting and everting the foot.

The equinous is corrected by pulling down the calcaneumand pushing the metatarsal bases upwards.

If the deformity persists,

Manipulation under anaesthesia, then the deformity is corrected and held in desired position in long leg plaster with the knee joint in a flexed position at 90 degrees. The pop will be left for 1/12 and removed at the age of 9 monctomyths when the child starts to step on the ground on his soles. Orthopaedic shoes are advised.

Surgical:

1. E.T.A (elongation of tendo Achilles)
2. Muscle slide (tendons of tibialis posterior) should take about 3/12.
3. Arthrodesis
4. Wedge tarsectomy.

2. Genu varus/ valgus Recaurvatum

Genu varus: a deformity where one presents with bow legs.

Genu valgus: where one presents with knock knee

Recurvatum:

pt presents with hyperextension of the knee joint.

In children this deformities correct spontaneously with or without treatment.

Causes:

1. Fracture of lower femur or fracture upper tibia
2. Rarefying of bones due to some diseases e.g. rickets, osteomalacia causing postural defects due to tear and wear, leaving some debri which are deposited at the articulating surfaces.
3. Uneven growth of epiphyseal plates due to diseases like osteoarthritis

Diagnosis:

It is not easy because of underlying diseases. Patient lies supine, puts legs together and check on the posture.

Management:

Treatment is generally conservative.

Orthopaedic boots

If in genu valgus, inner raise is put.

If in genu varus, outer raise is put

If there is any underlying cause like rickets, chemotherapy is given.

In severe cases there are three types of operations done,

i) Epiphysiodesis:- fusing the epiphysis so that they don't grow any more
ii) Supracondylar femoral osteotomy
iii) Tibial osteotomy.

As a clinician refer all these pts.

3. Pes cavus:

Definition:- refers to a hollow foot where the longitudinal arch is raised or exaggerated.

Causes

i. Congenital abnormality where the muscles of the dorsum are stronger than those of the plantar aspect (heredity)
ii. Could be due to any form of neurological disorders (e.g. polio, spina bifida) with resultant paralysis giving rise to muscle imbalance.

Pathology:

1. Metatarsal heads are lowered in relation to the hind part of the foot with increased longitudinal arch
2. Shortening of the soft tissue leading to
3. Clawing of toes due to defective action of intrinsic muscles leading to
4. Functionless toes (wt bearing is lost)
5. Excessive wt borne on the metatarsals head
6. Malalignment of the structures of bones (joints) complicating development of osteoarthritis due to tear and wear.

Clinical features:

It may present in one or both feet

Symptoms:

1. Painful callosities beneath the metatarsal heads
2. Tenderness over deformed toes due to pressure against the shoes

Signs:

1. Longitudinal arch is raised

2. Thick fore foot
3. Clawed toes
4. Metatarsal heads are prominent on sole

Management:

1. Conservative

Sponge rubber pad

Surgical boots

2. Surgical treatment

a) Arthrodesis – making an artificial fracture then the bone fused together at all interphalangeal joints.
b) Long flexor tendon transplantation into the extensor expansion (muscle slidig operation), p.o.p immobilization and removed later and pt trained by physiotherapist.

PES PLANUS:

Definition: it is characterized by reduced longitudinal arch of foot. The medial borderis close to or in contact with the ground.

Cause:

1. Congenital
2. Neurological disorders resulting in muscle imbalance

Pathology

The tarsals when articulating take a form of a straight line rather thanan arch.

Clinical features:

i. Symptomless in children
ii. Symptomless in adults except foot strains could be common which may develop to osteoarthritis later.

Treatment:

a) Conservative:- (i) inner (medial) raise (ii) fitting an arch support in the shoes
b) Surgical – arthrodesis of the talocalcaneal joint

GENU RECARVATUM

Definition: the pt presents with hyperextension of the knee joint. In children with this deformity correct spontaneously with or without treatment.

Causes;

1. Fracture lower or upper tibia
2. Rarefying of bones due to some diseases e.g. rickets, osteomalacia causing postural defects due to tear and wear leaving some debris which are deposited at the articulating surfaces causing postural deformities.
3. Uneven growth of epiphyseal plates due to disease like osteoarthritis.

Diagnosis;

Usually not easy due to underlying cause (disease). Between 1-3 yrs they are known as false (benign) equino- varus.

Pt lays spine, puts legs together and check on the posture.

Management;

The treatment is generally conservative

Orthopaedic boots

- If in equinovalgus – inner raise is put
- If in equinovarus an outer raise is put
- If there is any underlying cause like rickets , chemotherapy is adviced.
- In severe cases there are three types of operation done;

1. epiphysiodesis – fusing the epiphysis so that they don't grow any more
2. supracondylar femoral osteotomy
3. Tibial osteotomy.

As a clinical officer just refer them.

CONGENITAL HIP DISLOCATION (CHD)

Definition : a condition in which a child is born with the dislocation of the hip (one or both hips). It is common in girl ifants. The causes are unknown

Points of diagnosis (ostolan's test)

Shortening of the limb

Broad perineum

Skin creases

Limited abduction

NB x-rays are misleading

If with the above features and is;

0-5 yr = septic arthritis

5-10 yrs = perthes disease

10- 15 =slipped epiphysis

5. 15yr = osteoarthritis

Treatment:

Conservative

At birth the baby is put on an abduction splint for 3 months

In older children it can be reduced under G.A and then put on P.O.P for abduction for 3months

TORTICOLLIS:

Def. it is the commonest form of " wry neck"(amusing) characterized by tilted head as a result of the contracted sternomastoid muscle.

Causes

Uncertain but a possible assumption (cause) is the interfearance of bld suppl to the sternomastoid.

Pathology: formation of fibrous tissue in mid1/3 of the muscle. Tumour around an infarcted segment of the tumour due to trauma.

Clinical features:

Commonest age is between 6 months and 3yrs

Head is tilted to one side

Contracted muscle feels as a tight cord

Ear of the affected side is approximated to the corresponding shoulder

Retarded development of the face on the affected side

Diagnosis:

History of the above feature in a period of <.> 6/12- 3yrs

There should be a tight contracted sternocleidomastoid

Underdevelopment of the face in the affected side (facial asymmetry)

DDX:

- Trauma
- Lesions in the cervical nerve roots
- Abnormal development e.g. cervical wing
- Hemivertebra
- Infection of the cervical gland
- Psychogenic disorders.

TREATMENT:

In early stages;

Passive stretching of the muscle

Heat treatment

Manipulation

In late stages;

Surgical- the affected muscle is split at its lower attachment to release the contractures.

KYPHOSIS:

This is a general term used to define excessive posterior curvature of the spinal column

Presentation:

The deformity can take a form of long rounded curve

There might be a sharp posterior angulation "hump back"

In the cervical and lumbar region there is lodosis

When localized at the thoracic spine it is called thoracic kyphosis

Reversing of cervical and lumbar lodosis can lead to cervical and lumbar kyphous.

Causes:

1. Tb spine
2. Unreduced vertebral compression fracture

3. Osteochondritis
4. Ankylosing spondylitis
5. Senile (old age) osteoporosis
6. Tumours of spinal column

Treatment

It depends on the cause;

Treat the cause

For example if due to TB streptomycin3 months)+ thiazina(18 months)= note that this tregimen has changed

Fracture spine must be corrected

SCOLIOSIS:

Def. lateral curvature of the spine (S shaped)

It could be postural or structural

It can be primary or secondary.

Primary is due to causal pathology while secondary is due to a compensatoyr mechanism.

Types:

1. **Postural:-**

Caused by muscle spasms associated with PID

Compensatory mechanism (due to leg shortening)

2. **Structural**: Caused by;

K. Osteogenic (congenital) - children born with hemivertebra
3. Neurogenic paralysis. Common in; anterior poliomyelitis, severe spina bifida, neurofibromatosis, syringomyelia, cerebral palsy and spinal muscular atrophy

4. Myogenic: - Generalized muscle weakness as seen in cases of muscular dystrophy(they never survive beyond the age of 17yrs.

5. Thoracogenic: - It penetrates to the thoracic spine as in Tb spine, ca lung, and intrathoracic drainage tube.
6. Idiopathic: examples here include, rotated vertebra. Rib are prominent posteriorly “rib hump”.

When it occurs up to the age of 3yrs it is called infantile scoliosis and males show more than females (2/1000 in population).

4-10yrs it is called juvenile scoliosis and females show more than males

Above 10yrs it is called adolescent scoliosis.

Investigations:

a. Conservative

Constant assessment of degree of curvature

Measuring of the cardiopulmonary functions

Excercises coupled with spinal braces

b. Surgical: fusion of the facet joints stabilized by Harringtout’s distraction rods.

PROLAPSED INTERVERTEBRAL DISC (P.I.D):

Intervertebral discs are interposed between the vertebral discs.

Functions:

1. Serve as shock absorbers for the spinal column.
2. Provide the normal mobility between the adjacent vertebrae.

Each disc consists of a soft central portion of spongy material nucleus (polposus) which is surrounded by tough fibrous ring(annulus fibrosus) which is attached to the adjacent vertebral bodies, the whole being enclosed between fibrocartilaginous plates above and below. During normal flexion of the spine, the disc is deformed and the annulus fibrosus and nucleus bulge backwards slightly into the neural canal. Intervertebral disc protrution is produced by the effect of flexion forces acting upon the most mobile portions of the spine a sudden strain with the spine in an unguarded position will rupture the tough annulus, allowing portions of the torn annulus and soft nucleus to escape into the spinal canal and form either a central protrusion in the midline under the posterior common ligament of the vertebrae, or a lateral protrusion at the side of the posterior common ligament adjacent to the intervertebral foramen.

In 80% of the cases, the protrusion is traumatic in origin and there is either a history of sudden severe stain or the pt’s occupation is one in which flexion strain must be resisted, such as a packer, porter, fireman, etc. the condition is therefore more common in men.

In 20% of the cases, the condition is degenerative in origin. There is no history of injury. A small portion of the nucleus polposus herniates through it. The mechanism of prolapse demands a combination of stress and mobility. Therefore it is common in mobile portions of the spine which are subject to greatest of stress.

19% of the cases occur in the cervical spine at C5/6, C6/7

1-2 % occur in the immobile thoracic spine

80% occur in the lumbar region at level L4/5, L5/S1

Escape of material leads to;

1. Narrowing of the intervertebraljoint space which is visible in 50% of the cases
2. Slackening of the anterior common ligamentous vertebrae producing abnormal mobility betweenthe vertebrae of with local joint pain and ultimately development of intervertebral arthritis.

Osteophytes form on the anterior aspect of the major cervical injuries.

Alternatively, when spinal ligaments are softened at the end of pregnancy tha strain of labour may force out a massive protrution in the lumbar giving rise to the form of obstetric paralysis.

LUMBAR DISC PROLAPSE:

It usually occurs above or below L5. Rarely the disc between 3rd and 4th may be affected. There is low back ache with evidence of compression of the 5th lumbar nerve. If the 1st sacral nerve root is compressed there is pain and loss of sensation in the back of the leg, sole, side of the foot.

If the 5th lumbar nerve root is affected there is pain and loss of sensation at the back of the thigh, lateral aspect of the leg, dorsum of the foot.

Acute prolapsed causes severe pain often following lifting of heavy wts. The spinal muscles go into spasms, the pt is in agony, unable to walk or bend. The lumbar spine is flattened and sometimes sclerotic.

A prolapsed in the midline presses the cord and causes back ache alone.

A lateral prolapsed may press on the adjacent nerve roots leading to numbness of foot and diminished ankle reflex.prolapse of the disc between L5 and S1 typically produces pain which radiates down the leg, soleof foot and big toe and is called SCIATICA.

Prolapsed of disc between L1 and L2 is less common but may cause pain in the flanks with radiating pain to the groin which may mimic renal colic or billiary colic.

Prolapsed is often accompanied with scoliosis. This scoliosis may change from one side to the other as the pt bends and extends and is calle ALTERNATING SCOLOSIS.

Characteristically a pain of prolapsed disc is aggravated by cough, sneezing etc.

In the early phase it is often worsened when the pt is resting in bed. An important feature of disc prolapsed that distinguishes it from an inflammatory disease e.g. Tb, spondylitis is that in this prolapsed some joints are very mobile and painless unless symptoms are very acute whereas in Tb all movements cause pain. In addition there are no signs of a systemic disease in prolapsed.

Clinical features relating to level lambosacral disc compression

N.root affected	pain and sensory loss	motor weakness	reflex change
1st sacral	back of leg, sole and side Of foot	Gastrocnemius, weak plantar reflex	absent Aj
5th lumbar	back of thigh, most of Lateral aspect of leg Dorsumof foot to big toe	anterior tibialis, weak dorsiflexion	Nil
4th lumbar	side of thigh, front of Inner aspect of leg	quadriceps and anterior tibial, weak dorsiflexion and extension of knee	diminished knee jerk
3rd lumbar	front of lower thigh	quadriceps	diminished kne e jerk
2nd lumbar	front of mid thigh	quadriceps	diminished knee jerk
1st lumbar	Groin	Nil	Nil

Treatment:

1. Confinement to bed until symptoms abate, usually 2-4 wks. Majority of cases are cured by this method
2. Surgery is indicated when;

o) Symptoms persist
p) If severe pain reccurs
q) If weakness, sensory loss or sphincter disturbance develop.

Operation approaches available are;

i) Laminectomy
ii) Hemilaminectomy

iii) Fenestration(interlaminar)
iv) Microdisectomy

Complications:

Even after satisfactory removal of PID, symptoms or physical signs are not always alleviated.

a) Prolonged compression of the nerve may have resulted in interstitial neuritis resulting in sensory loss or motor weakness or
b) The intervertebral joint may be unstable or become affected by osteoarthritis.

PERTHES DISEASE:

Def. this is the infarction of the proximal femoral epiphysis and is known as Legg-calve- perthes disease.

The epiphyses of other bones are not spared, for example, involvement of the distal epiphysis of the 2nd metatarsal it is known as Freiberg's disease

Navicular bone – Kohler's disease

Lunate bone – Kienbock's disease

Of these, perthes disease is the most common and has the most serious consequences for the pt.

Pathology:

The presence of an epiphyseal plate results ina peculiarity of bld supply to the boe of the epiphysis, for bld vsls don't cross the epiphyseal plate. The bld supply to the epiphysis is derived entirely from vsls passing around the periphery of the plate and along the ligamentum teres. Since the epiphysis is entirely intraarticular all epiphyseal vsls run beneath the synovial membrane before entering the epiphysis itselfand thus the epiphysis could be infracted if, for example, fluid collected in the joint under sufficient pressure to occludethe vsls. This is the mechanismof infarction in perthes disease.

Clinical features:

Boys are affected more than girls in the ratio of 4:1 (reason is unknown)

Condition usually present between 3 and 10 yrs

18% of the cases are bilateral

Pt presents with pain poften in the anterior thigh and knee

Pt limbs particularly in the evening and after exercise

There may be history of injury in the past but there is commonly a silent period between this incident and the onset of symptoms

Clinically:

Shortening of the limb

Wasting of the quadriceps

Restriction of hip movement by muscle spasm with pain at the extremes of movement. This muscle spasm will commonly resolve in a few days of bed rest with skin traction.

DDX:

Inguinal hernia

Genitor-urinary abnormalities

Radiological features:

Joint space becomes greater than the opposite side. This is due to effusion or continuing growth of the cartilaginous portion of the femoral head.

In the 2nd stage, part or all bony epiphyses become crushed or fragmented, protruding a broad and flattened femoral head in many cases.

In the 3rd phase the crushed area is reabsorbed and finally replaced bone.

Prognosis:

The longterm prognosis of this disease is ostoarhritis of the hip.

The symptoms may be delayed for 30-40 yrs. The more deformed the femoral head at the time of healing the greater the chances of early symptoms.

Girls have a worse prognosis than boys

Treatment:

No specific treatment for the underlying pathology. The objective of treatment must be to prevent severe flattening of the femoral head. The principles of treatment are divided as;

1. Restoration of movement by;
a) Containing the femoral head within the acetabulum
b) Mobilization of the reduced hip
2. Prevention f further ischaemia by;
a) Relief of further stress to the hip
b) Prevention of injury

Containment of the femoral head within the acetabulum may beachieved by holding the legs apart in abduction and internal rotation (broomstick plaster). Active movement is encouraged while the legs are in the plaster, to restore the normal range of movement to the hip.

BURSITIS:

Def. Comes from the word bursa which is a fibrous sac lined with synovial membrane containing a small quantit of synovial fluid.

Sites: It occurs in three main sites;

Between tendons and bone

Between muscles and muscles

Between skin and bone

Functions: To facilitate movement without frictionbetween the surfaces involved

Simple bursitis: (acute traumatic bursitis)----It follows injury or unaccustomed exercise e.g. inflammation of the bursa from anterior to the tendo-Achilles may occur following cross country.

Chronic bursitis: occurs as a result of repeated pressure or slight injuries to bursae e.g.

i) Involvement of the pre-patela bursa givingrise to " housemaid" knee
ii) Olecranon bursa giving rise to student or miner's elbow
iii) Satorreal bursitis involves the satorrius muscle of the thigh which flexes one leg over another.

The above three are anatomical bursae.

Semi-membranosus, which occur in children posterior to the knee joint. The cyst is aspirated if it causes disability but most cysts disappear spontaneously. If semi-membranosus communicate with the knee joint they enlarge when there is an effusion in the joint and form one variety of " Baker's" cyst. In general synocial cysts in the popliteal fossa which are known as Baker's cysts may arise from a semimembranosus bursa or rheumatoid arthritis of the knee as a result of posterior rupture of the joint (another form of Baker's cyst).

ACUTE SUPPURATIVE BURSITIS:

Is due to acute infection of the bursae by penetrating wound or spread of local cellulitis. The most commonly involved is the pre-patela bursa. It should not be confused with infective arthritis because in infective arthritis any attempt to move the joint is painful and the pain is elicited by pressure on the popliteal fossa. Asympathetic effusion in the knee joint sometimes follows.

Treatment:

1. The infection usually responds to chemotherapy but
2. If pus is already present incision and drainage will be necessary.

ADVENTITIOUS BURSA:

It forms as a result of prolonged pressure over bony eminencies. It means that no anatomical bursa where a cyst has formed and that it was generated in connective tissue as a result of repeated motion in the tissue. The commonest is that over the medial aspect of the 1st metatarsal bone which is found in a condition called Hallus Valgus, a condition inititated by too narrow foot wear. It is characterized by deviation of the big toe. It can be come infected.

Excision of the bursae:

Persistent trouble with a subcutaneous bursa is solved by total excision of the whole of the endothelial lining under G.A.

TENOSYNOVITIS:

Simple tenosynovitis: It follows excessive or unaccustomed use and is commonly seen in connection with the extensor tendons of the hand and the Achilles tendon.

Clinical features:

Pain and local oedema are present and a characteristic soft crepitus is sometimes palpable when the fingers are moved.

Treatment:

Resting of the tendon involved which in the case of the extensors of the hand must involve the fingers

A minimum of 3/52 absolute rest in a splint is usually required, followed by a period of gentle but progressive activity.

In appropriate cases, steroid injections into the tendon sheath can be very effective.

Suppurative tenosynovitis:

This condition affects particularly the flexor tendons of the hand since these sheaths are far more commonly injured than those elsewhere in the body. Infection is by bacteria introduced by the point of needle or other sharp object penetrating the tendon sheath. The sheath may be infected by extension from its terminal, pulp space or in some cases from the scalpel transgressing the hallowed ground of the septum that closes the proximal end of the space. This is the most feared of the infections in the hand. pus within the tendon sheath destroys the gliding mechanism, creates adhesions and leads to loss of tendon function and reduced movement.

Acute fulminating tenosynovitis involves the whole sheath rapidly and nearly always the infecting organism is staphylococcus aureus or streptococcus pyogens. The classical local signs are;

Symmetrical swelling of the entire finger

Flexion of fingers (hook sign) with severe pain on extension

Tenderness over the sheath

Treatment:

This condition must be treated aggressively. There is no time for conservative management.

1. Transverse incisions at opposite end of the tendon sheath are required. The tendon sheath should be thoroughly irrigated at the time of surgery with isotonic saline using a ureteric catheter. There is no need to leave a catheter in situ for post operative irrigation. In late cases that require excision of necrotic tissue exposure is best obtained by mid lateral incision but the outcome is likely to be a stiff joint.
2. Antibiotics (sensitive to the organisms that have been cultured).
3. Analgesics.

Complications of suppurative tenosynovitis:

a) Involvement of the forearm from the hand (spread)
b) Continuation of suppuration chronicity should suppuration continue for more than 14 days, the hand should be x-rayed to rule out bone involvement (osteomyelitis).
c) Suppurative arthritis in a related joint . in these circumstances timely amputation of any digit except the thumb will reduce the period of disability.
d) Stiff digit – Total amputation of digit is less of a handicap than a stiff finger but amputation should be done when the infection has subsided.
e) Paralysis of the median nerve.

OSTEOGENIC IMPERFECTA:

Def. A congenital and inheritable condition in which the bones are abnormally soft and brittle (fragile)

Clinical features:

1. In severe cases;

The child may present with multiple fractures

Survival is very limited

2. In mild cases;

Fractures occur at birth or after a slight violence

Later on present with very bad deformities due to mal - union

Later on there is a marked bluecolouration of the sclerotic bone

Deafness due to the osteosclerosis

Laxity of ligaments

Treatment:

- Immobilization

- P.O.P application

- in severe cases intramedullary nailing of long bones and immobilize either by P.O.P (for children about 6/52)

- Protective/supportive appliances e.g walking calipers, clutches so that there is no direct wt transmission to the fractured area.

BENIGN TUMOURS OF SOFT TISSUES:

a) NEUROFIBROMA:

A benign tumour arising from the connective tissue of the nerve sheath.

Varieties:

i) **Local neurofibroma** – this tumour is usually found in the subcutaneous tissue. It is a painful subcutaneous nodule which forms a smooth firm swelling which may be moved in a lateral direction, but otherwise fixed by the nerve from which it arise. Paraesthesia or pain is like ly to occur from the pressure of the tumour on the nerve fibres which are spread over its surface cystic degeneration or sarcomatous changes occur occasionally. As the nerve fibres are part and parcel of the tumour they are difficult to remove without removal of the itself. In major nerve recurrence is known, also malignanant (sarcomatous) change.

ii) **Generalized neurofibromatosis:** This is an inherited (autosomal dorminant) disease. Any cranial, spinal, or peripheral nerve may be diffusely or nodulary thickened. Overgrowth occurs in connection with the endoneurium. Associated pigmentation of the skin is common. Sarcomatous change may occur.

b) FIBROMA:

A true fibroma contains only fibrous connective tissue and is rare. Most fibromas are combined with other mesodermal tissues such as muscle (fibromyoma), fat (fibrolipoma), nerve sheaths (neurofibroma) etc. Multiple tumours are not uncommonas in neurofibromatosis. Fibromas are

either soft or hard depending on the proportion of fibrous to the other cellular tissue. Soft fibromas are common in the subcutaneous tissue of the face, and appear as a soft, brown swelling.

Keloid: this overgrowth of fibrous tissue commonly occurs in scars especially of black people.

c) LIPOMA:

This is a slowly growing tumour composed of fat cells of adult type. They may be encapsulated or diffuse. They occur anywhere int the body where fat is found and earn the tittle of the "universal tumour" or "ubiquitous tumour". Common areas include, head, neck, abdominal wall, and the thighs.

Encapsulated lipoma:

Are among the commonest tumours.

-The chractaristic features are the presence of a definite edge and lobulation

May be fluctuant

Deeply seated (situated) lipomas may be miatsken for other swellings

Most lipomas are painless, but some give rise to an aching sensation which may radiate.

Multiple lipomas:

They are not uncommon

They remain small or moderate in size

Are sometimes painful

Should a lipoma contain an excessive fibrous tissue it is termed as fibrolipoma

If considerably vascular and often with considerable telangiectasis of the overlying skin the tumour is then called naevolipoma.

Large lipomas of the thigh, the shoulder and the retroperitoneum occasionally undergo sarcomatous changes.

Clinical classification of lipomas (according to situation)

I) Subcutaneous:

Commonly found on the shoulder or back

May be present over the site of a spina bifida

Occasionally become penduculated

i) Subfascial:

Occur under the plantar and palmar fascia

Liable to be mistaken for Tb tenosynovitis as the tough overlying fascia masks the definite edge and lobulation of the tumourdifficulty is encountered in complete removal as pressure encourages the tumour ramify(move).

ii) Sub-synovial:

From the faty padding around joints especially the knee

May be mistaken for Baker's cyst ut easily distinguished as in distinction toa cyst or bursa, their consistency is constant whether the joint is in extension or flexion.

iii) Intraarticular
iv) Intermuscular
v) Periostel –occasionally occur under the periosteum of the bone.
vi) Subserous –sometimes beneath the pleura, where they constitute onevariety of inocent thoracic tumours.
viii) Sub mucous
ix) Extradural – a lipoma is a rare variety of spinal tumour. Owing to the absence of fat within the skull, intracranial lipomas do not occur.
x) Intraglandular – lipomas have been found occasionally in the pancrease, under the renal capsule and in the breast.

Treatment:

If it causes trouble on account of its size, site, appearance, or the presence of pain removal is indicated. During operation any fingerlike projections of the tumourinto the surroundingtissue should also be removed. Although the tumour is relatively avascular, care is needed to obtain complete haemostasis in the resulting cavity; otherwise a haematoma is common which may be followed with infectionand delay in wound healing. Drainage is often necessary.

d) Hamaegioma:

This is a developmental malformation rather than a true tumour. It is an example of a Harmatoma. It may occur in any tissue of the body but most common in the skin and subcutaneous tissue. A haemangioma is either capillary, venous (carvenous) or arterial in type.

Capillary haemangioma:

(i) Salmon patch (stork bites) is present at birth over the forehead in the midline, and over the occiput. It disappears by the age of 1yr.

(ii) Port wine stain (naevus flameus) it presents at birth, it changes very little throughout life,although the colour may alter a little and it may become nodular in some areas. Treatment is for cosmesis skin texture is quite normal.

(iii) Strawberry angioma:

It is common

Has a typical history i.e. the baby is normal at birth and at the age of 1-3 wks is noted to have a red mark. It rapidly increases for some wks or even up to 3 months, until the typical straw berry - like swelling is present. The lesion is composed of immature vasoformative tissue. The subcutaneous tissue as well as the skin is often involved, and in severe cases the mm may be affected. Submucous naevi are prone to haemorrhage, which is sometimes alarming. From the age of 3 months to 1yr the naevus grows with the child and then it caeses to grow. Eventually the colour fades and flattening occurs so that at the age of 7-8 yrs involution is complete.

Venous angi oma (carvenous):

It is relatively uncommon. It presents at birthand consists of multiple venous channels of varying clibre. Usually shows no tendency to involution and may become larger and more troublesome later. Sometimes the whole of one limb and the adjacent part of the trunk is affected. It is occasionally associated with a lipoma (naevolipoma). In some cases, arteriovenous communications are present. The skin overlying the naevus may be atrophic, and may be in danger of developing severe haemorrhage from trauma. The patient mat suffer from septicaemia if organisms gain entry, in which case the energetic use of antibiotics is an urgent matter.treatment is generally conservative. Injection of sclerosing agents as for varicose veins is followed by variable results.

Arterial (plexiform) angioma:

It is a type of arterivenous fistula. The pulsating swelling of arteries and arterialized veins is often called a crisoid aneurysm.

Spider naevus: may be associated with liver diseases and may present in the skin over the manubrium sterni, but may occur quite innocently. It shows the characteristic sign of emptying.

Naevus tardes: are small angiomas occurring in adults, often around the mouth. They may be associated with vasospastic and scleroderma.

Lymphangioma: they affect the lymphatic channels. Sometimes associated with haemangioma (haemolymphangioma).

Tumours

Table 9.1 A classification of the less rare primary bone tumours

Cell type	Benign	Malignant
Bone	Osteoid osteoma	Osteosarcoma
Cartilage	Chondroma Osteochondroma	Chondrosarcoma
Fibrous tissue	Fibroma	Fibrosarcoma
Marrow	Haemangioma	Angiosarcoma
Uncertain	Giant cell tumour	Malignant giant cell tumour

TUMOURS OF THE BONE:

BENIGN TUMOURS:

Benign tumours of the musculoskeletal system are common and frequent.

They appear to have been present from birth and they grow with the child.

They may be multiple

If truly benign they stop growing when the child stops growing.

They are incidentally found on an X-ray taken for other reasons.

Most benign tumours of bone occur in children and young adults and often stop growing with the cessation of skeletal growth.

Such lesions might more probably be regarded as localized dysplasias rather than neoplasia.

Nomenclature and classification

These tumours are named after dorminant type of tissue from which they have arisen. They can be identified from their shape and their staining. The benign tumours take the suffix "OMA". Where possible benign tumours should be left alone. But there are three indications for surgical intervention in benign tumours.

1. When there is doubt as whether benign or malignant and a biopsy is required to determine the type of tumour. When removing a biopsy in this case, it should be treated as a malignant and therefore excisional biopsy is adviced to avoid another surgery later.

2. When the tumour has weakened a bone and a pathological fracture has occurred or is likely to occur.
3. If the bony protruberance is so prominent that it creates a cosmetic deformity or interferes with muscles or joints and the function of the limb is interfered with.

Difference between benign and malignant tumours:

It is usually not easy in a growing child.

1. History: - Benign tumours are usually painless while in the malignant tumours pain is noticeable at rest particulary at night (very characteristic)
2. Examination: - A swelling which has been present for a long time is likely to be benign. However some benign tumours can turn malignant.if this kind of lumps suddenly change in size or increase in tenderness then malignancy should be suspected.
3. X-rays: - Benign tumours have a well defined margin unlike the malignant ones which have irregular margins. Periosteal lifting over the site of lesion indicates inflammation and can be a clear sign of malignancy. Periosteal elevation may also occur over a stress fracture or if there is underlying infection, so rule them out.

a) Osteochondroma:

A common benign tumour.

Usually multiple,

They are overgrowths of bone, which may look much smaller on x-ray than they feel on clinical examination. This is because the overgrowth of bone has a large cartilage cap over it.

They are commonly on a bony pedicle which gros away from the epiphyseal plate and which is covered in a large cartilage cap.

The common site is the femur or the tibia around the knee. If large enough they can interfere with the function of the knee.

Occasionally they become malignant.

Therefore if they become painful malignancy should be suspected.

Usually it ceases to grow when skeletal growth is complete.

The lesion may be solitary or multiple

Compact osteoma (ivory exotosis): It consists of a small knob of extremely hard, dense, but otherwise normal bone usually arising on the inner or outer table of the skull.

b) Osteoid osteoma:

They occur in children and adults.

They are commonest in the femur and tibia but can occur elsewhere (even in the spine)

They differ from other benign tumours because they produce constant aching pain commonly at night unrelieved by rest but

The pain is specifically relieved by salicylates.

They are difficulty to see in an x-raywhere they look like an area of slight sclerosis.

Tomography shows a characteristic radioluscent center.

This tumour consists of a center of cellular, highly vascular tissue with randomly arranged osteoid trabeculae.

It is usually less than 1 cm in size and provides dense new bone formation around it. It is most commonly in pts between 5-25yrs old.

It is slightly more common in males and usually occurs in the long bones.

The only abnormal physical sign is bone tenderness; and even this is often absent so that pt may sometimes be regarded as hysterical.

Surgical excision relieves the pain but they can be difficult to find at surgery.

c) Chondroma:

They are mainly made of up of cartilage and are common in the hands and feet'

The medulla of the bone may be scalloped out (eaten out) and this is known as enchondroma. There may be thinning of the cortex causing pathological fracture. When there are multiple enchondromas the condition is known as Oilier's disease.

Malignant change is not common but when many chondromata are present this risk is increased.It consists of a lobulated mass of cartilage. It may arise in any bone, but is more frequent in the metacarpals, phalanges or metatarsals. Occasionally arise in synovial membrane. if situated in medulla (endochondroma), the bone is thinned and expanded by the tumour causing pain and deformity.pathological fracture is common. Tumours on the surface of the bone are known as ecchondroma. The matrix of the tumour may calcify and sometimes ossify. They may be solitary or multiple. Malignancy is rare in solitary tumours but may occur when the tumours are multiple.

d) Osteoclastoma(giant cell tumour)

They are benign tumour filled with undifferentitated spindle cells and multinucleated giant cells. Commonly found in the epiphysis of a bone, lying close to the epiphyseal plate.the cortex over the tumour may be destroyed and there may be periosteal elevation. They can be treated block excision but, unfortunately, they are closely associated with a joint. When rapidly growing or recur after excision they may be malignant and require more aggressive treatment.

Mechanisms of spread

- Local
- Haematogenous
- Lymphatic
- Intramedullary
- *Others e.g.* injections, transfer (iatrogenic)

Clinical Diagnosis of tumours

a. **Age of patient;**
    - **< 5yrs old - Malignant**
    - **5-30yrs old - Benign; Also Ewing's sarcoma & Osteosarcoma**
    - **30-60yrs - Mixed - Chondrosarcoma, Fibrosarcoma**
    - **> 60yrs - Secondaries (malignant); Multiple myeloma**
b. **Symptoms;**
    - **Benign - No pain**
    - **Malignant - Vascular pain - *Worse at night & throbbing due to ischemia due to increased blood demand;* Also 2° to *pressure effect on bone & soft tissue***
c. **Duration of symptoms;**
    - **3 months - Malignant**
    - **6 months - Borderline**
    - **1 year - Benign**
d. **Imaging;**
    - **Benign - *well defined transitional zone* with regular/Scalloped margins e.g. Osteochondroma which are mostly metaphyseal especially around the knee & may be pediculated or sessile**
    - **Malignant - *Diffuse transitional zone***

DDx

- Soft-tissue haematoma
- Myositis ossificans
- Stress fracture
- Tendon avulsion injuries
- Bone infection
- Gout
- Other bone lesions e.g. *fibrous cortical defects, medullary infarcts & 'bone islands'*

Enneking System of Classifying Musculoskeletal Tumours

I. All **low-grade** sarcomas with **<25%** chance of metastasis e.g. *2° chondrosarcoma, Parosteal osteosarcoma*

II. Histologically **high-grade** lesions with **>25%** chance of metastasis e.g. *osteosarcoma & fibrosarcoma*

III. Sarcomas which have **metastasized**

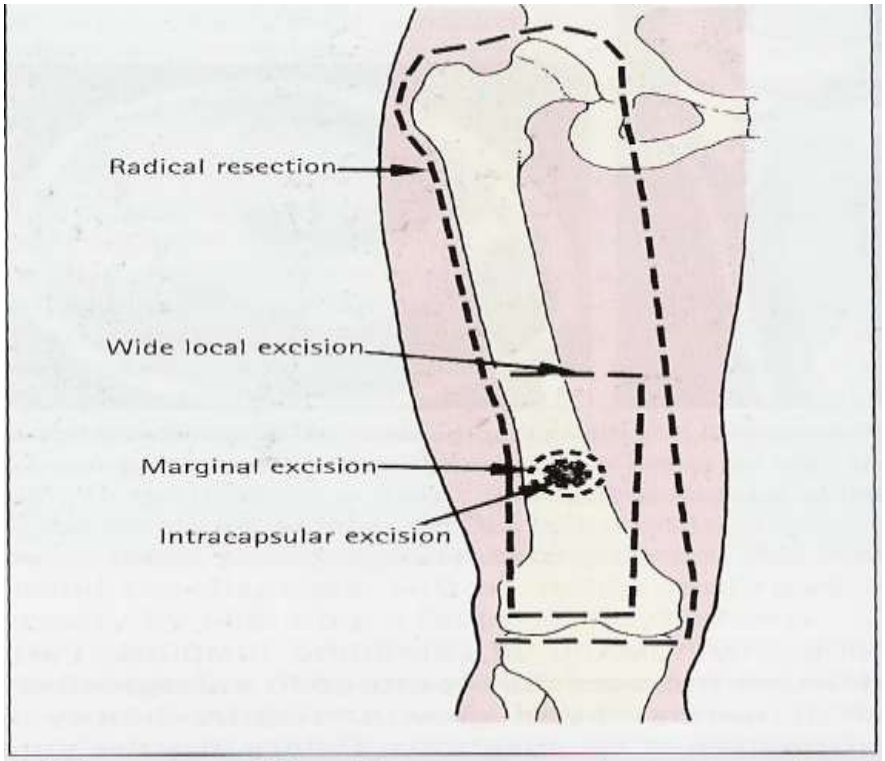
+

A. **Intracompartmental** e.g. A lesion contained in a single muscle belly or a bone lesion that has not broken out into the surrounding soft tissue

B. **Extracompartmental** e.g. A lesion in the *popliteal space, axilla, pelvis, or midportion of the hand or foot*.

MANAGEMENT

C.



9.4 Tumour excision The more aggressive a tumour is, and the wider it has spread, the more widely it needs to be excised. Local excision is suitable only for low-grade tumours that are confined to a single compartment. Radical resection may be needed for high-grade tumours and this often means amputation at a level above the compartment involved.

Osteosarcoma

In its classic (**intramedullary**) form, osteosarcoma is a ***high grade malignant tumour*** arising within the bone & spreading rapidly outwards to the periosteum & surrounding soft tissues.

Epidemiology

- Children > Adults - **5-19yrs; 22-26yrs** - *This has been attributed to* ***increased bone growth***
- Adults - **≥60yrs** - *History of* ***exposure to radiation*** *when young &/or* ***bone infection***
- **M:F - 2:1**

Pathogenesis

The tumour is usually situated in the **metaphysis of a long bone,** especially **around the knee** & at the **proximal end of the humerus,** where it destroys & replaces normal bone. Areas of *bone loss & cavitation* alternate with *dense patches of abnormal new bone*. The tumour extends within the medulla & *across physeal plate*. There may be obvious spread into the soft tissues with ossification at the periosteal margins & streaks of new bone extending into the extra-osseous mass. The tumour spreads mostly ***haematogenously*** > *intramedullary* > *local spread*. Rarely lymphatic.

Classification

a. **Primary;**
    - **Central (classical)**
        - **High grade - *Distal femur, proximal tibia & proximal humerus***
    - **Juxtacortical;**
        - **Low grade (Parosteal) - *Distal femur***
        - **Intermediate grade (Periosteal) - *Shaft of long bones***
        - **High grade (surface sarcoma) - *Shaft of long bones***
    - **Haemorrhagic or Telangiectatic;**
        - **High grade - *Epiphysis***
b. **Secondary;**
    - **Paget's disease**
    - **Radiation**
    - **Fibrous dysplasia**

C/P

- Pain - *constant, worse at night & gradually increases in severity*
- Swelling
- Local tenderness

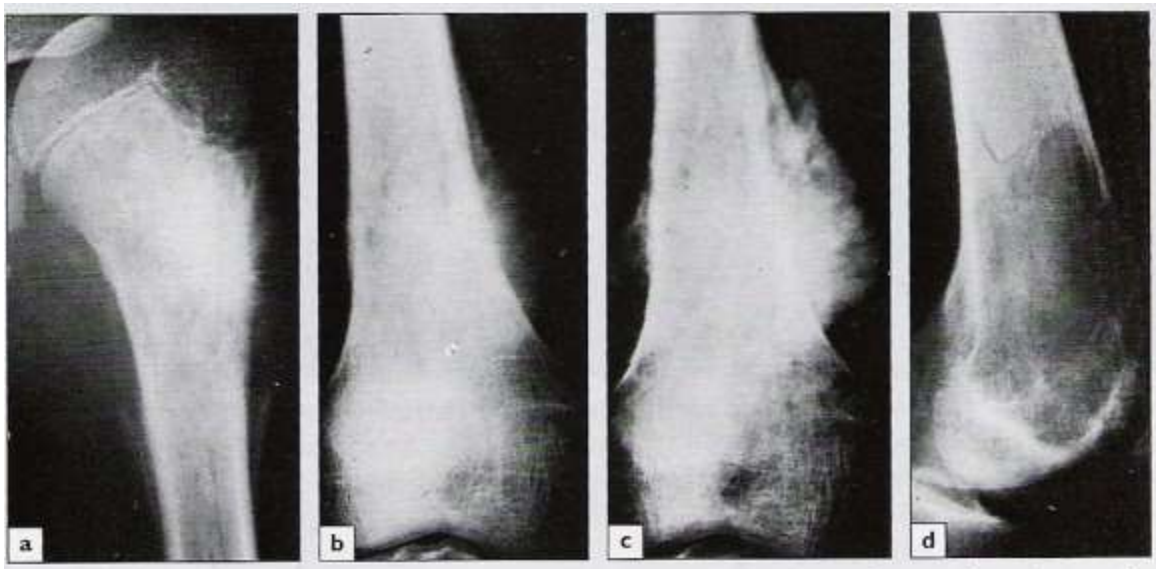
** *Pathological fracture is* ***rare***

Ix

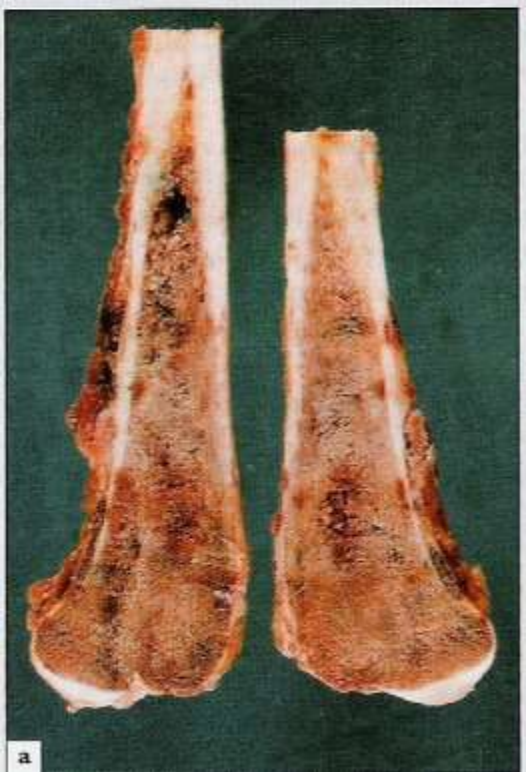
X-Ray;

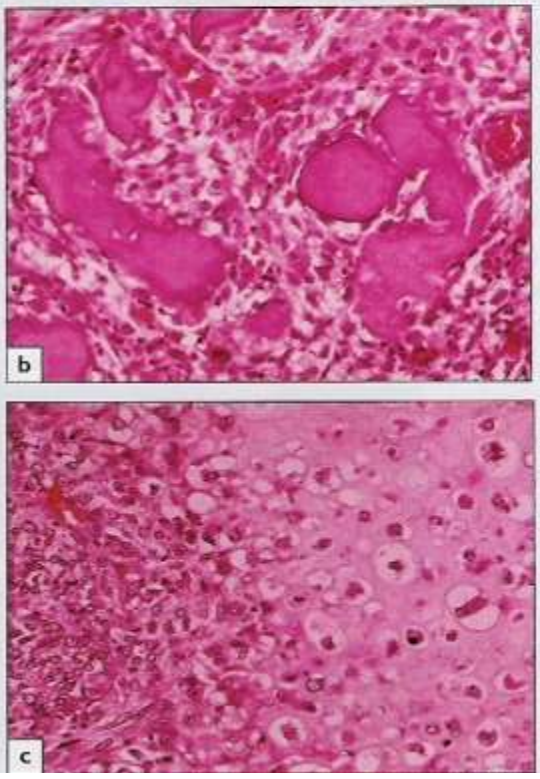
i. ***Hazy osteolytic*** lesions alternating with unusually ***dense osteoblastic*** areas
ii. The *endosteal margin is poorly defined*
iii. The *cortex is breached* & the tumour extends into the adjacent tissues; when this happens, *streaks of new bone appear radiating outwards from the cortex* - **Sunburst effect**
iv. Where the tumour emerges from the cortex, *reactive new bone forms at the angles of periosteal elevation* - **Codman's triangle**

e



9.27 Osteosarcoma (a) The metaphyseal site, increased density, cortical erosion and periosteal reaction are characteristic. (b) Sunra spicules and Codman's triangle; (c) the same patient after radiotherapy. (d) A predominantly osteolytic tumour.





9.28 Osteosarcoma – pathology (a) After resection this lesion was cut in half; pale tumour tissue is seen occupying th distal third of the femur and extending through the cortex. (b) The dominant features in the histological sections were malignant stromal tissue showing osteoid formation (pink masses). (×480) (c) The same tumour showed area of chondroblastic differentiation. (×480)

- Blood;
  - FHG + ESR
  - ↑ ALP + LDH

- CT for staging - **Pulmonary CT** is a much more sensitive detector of **lung metastases** which are present in about **10%** of patients at presentation.
- Liver ultrasound
- Scintigraphy using Methyldiphosphonate PC^{99} (MDP)- *For skip lesions*
- Biopsy;
    - FNAC
    - Incisional biopsy - *All layers from skin to bone*
    - Excisional biopsy - *Wide margin (at least 2mm)*
- ***MRI is NOT very useful***

DDx

- Stress fracture
- Infection - *Acute osteomyelitis*
- Post-traumatic swelling
- 'Cystic' lesions
- Other tumours

Mx

Supportive

Specific

- **Multi-agent neo-adjuvant chemotherapy is given for 8-12wks;**
    - **Eliminates micrometastasis**
    - **Reduces size of tumour reactive zone**
    - **Causes tumour necrosis**

  Then provided the tumour is resectable & there are no skip lesions, a **wide resection** is carried out. The tumour responds well to **Methotrexate, Adriamycin, Cisplatin, Ifafosphamide (MAC-i)**
- The segment of bone is replaced with either a large **bone graft** or a **custom made implant;** in some cases, amputation may be more appropriate.
- The tumour specimen is examined to asses response to pre-op chemotherapy & **if tumour necrosis is marked**, chemotherapy is continued for another **6-12months;** if response is poor, a different chemotherapeutic agent is substituted.
- Pulmonary metastases, especially if they are small & peripherally situated, may be completely resected with a wedge of lung tissue.

Prognosis

Long-term survival after wide resection & chemotherapy - **50-60%** if treated early & **<10%** if late presentation.

MALIGNANT TUMOURS OF THE BONE:

Majority bone tumours are secondaries and their management is significantly palliative. A pathological fracture through a metastatic tumour may be the first clue that a pt has malignant disease. The primary lesion may never be found. In some cases metastasis occurs sometimes after treatment of the primary tumour. The commonest source of metastasis in bone is tumours of the

breat, prostate, and kidney. Malignant tumours take the suffix “SARCOMA”. Tumours forming a cartilaginous matrix are named chondrosarcoma, the fibrous series 0 fibrosarcoma .

Primary malignant tumours:

They are very rare. The commonest is osteosarcoma. They have very variable histology and occur either in adolescents or in the elderly secondary to paget’s disease.

Giant cell tumour (osteoclastoma):

It is uncertain from which cell the tumour arises.

It is composed of undifferentiated spindle cells and multinucleated giant cells in a vascular stroma.

Microscopically it is distinguished from the so called brown tumour of hyperparathyroidism and this condition must be excluded by biochemical and radiological investigations.

It occurs between the age of 30-40yrs.

It is common in women

The epiphyseal regions of the long bones, especially around the knee, are the most common sites of origin but humerus, radius and ulna are occasionally affected. A tumour arising from the metaphysic is uslikely to be a giant cell tumour.

The tumour is osteolytic

It expands the bone with thinning, often perforation, of the cortex to cause a pathological in some cases.

The bone is destroyed irregulary so that the tumour is traversed by remnants of the original bone and comes to lie in the cavity with heavyily trabeculated walls.

As a consequence, it has a typical “soap bubble” radiologica appearance.

Rarely invades soft tissues but may do so when fracture occurs.

Locally the tumour is only of low grade malignancy.

Metastasis is rare but occurs via the blood stream to the lungs.

Reccurence following local removal is common and such reccurences are more likely to be frankly malignant than the original tumour.

OSTEOSARCOMA:

This is the commonest among the rare malignant tumours.

It has a variable history

It is common in adolescents or in the elderly secondary to paget's disease

It is usually metaphyseal (commonly the tibia or femur)

It is to confuse it with a stress frature or an infected haematoma.

If suspected the tumour must not be biopsied

Imaging:

Characteristically there is periosteal lifting (codman's triangle) and a sun ray specules of new bone within the tumour. MRI and CT scan may be helpful in determining the true extent of the tumour within the medulla.

Treatment:

Choice of treatment depends on the histological grading of the tumour, age of the pt, the presence or absence of secondaries and the wishes of the pt.

If no evidence of secondaries, an amputation is the treatment of choice with chemotherapy to destroy micrometastasses.

Alternatively, RT and chemotherapy to shrink the lesion followed by a massive joint replacement(there is high risk of recurring but more acceptable cosmetically and functionally

Prognosis: Survival depends on the histological grading and the degree of spread. Cure rates of about 50% can be achieved.

CHONDROSARCOMA:

Commonly occurs in the pelvis, ribs, or proximal large bones in middle aged people. The tumour is lytic with ill defined boundaries and has speckled (coloured or spots) calcification within its substance. The grade of malignancy is very variable and closely linked to the prognosis. Low ,grade tumours mhave a survival rate of 75% at 5yrs. High grade tumours have a survival rate of less than 10% at 5yrs

FIBROSARCOMA:

They occur most commonly in the metaphysic or diaphysis of the tibia or femur. They may also arise in the soft tissues. They metastase to the lungs and the 5 yr survival rate is around 30%

SYNOVIAL SARCOMA:

A highly malignant tumour. Metastases through the blood stream and lymphatics. Five yr survival rate is low regardless of the treatment offered.

EWING'S TUMOUR:

Common in children

Arises in the mid shaft or metaphysis of long bones.

Child may present with pyrexia

They usually present with high sedimentation rate and can be confused with osteomyelitis

X-ray may show multiple layers of subperiosteal new bone producing an "onion"arrangement. The tumour is highly malignant but aggressive treatment with chemotherapy and radiotherapy has produced some survivors.

NB:

Bone tumours are rare. Most of them are secondary tumours, which to be primary bone tumours should only be investigated and treated at specialized centers. The quality of life of pts with bonne secondaries can be improved dramatically with internal fixation followed by radiography. Pain will be reduced and mobility maintained.

OSTEOMYELITIS:

Definition: infection of the bone and bone marrow.

Types:

Occurs in two forms;

a) Acute osteomyelitis
b) Chronic osteomyelitis

Aetiology:

Acute haematogenous osteomyelitis: The bacteria reach the bone through the blood stream.a primary focus may be obvious in the form of a bil or infected wound. However sometimes may occur without obvious source of infection. May rarely occur secondary to se pticaemia.

Causative organisms include;

Staph. Aureus (the commonest)

Streptococcus

Pneumococcus

Haemophilus influenza

Staph. Albus

Salmonella

Pathology:

The disease nearly always begins at the metaphysic. The infective processs progresses through the thickness of the cortex via the Harversian canals. This leads to thrombosis of the blood vessels in the bone leading to infarction of the cortex. In the first 24-048 hrs of infection, an inflammatory exudates forms deep to the periosteum, elevating the membrane from the bone. Periosteal elevation is paiful and, since the eriosteum is ineslastic, the inflammatory exudates deep to it is under tension. This leads to rapid development of toxic signs. Farnk pus develops subperiostially after 48hrs. the inflammatory process progresses along the length of the medulla causing venous and arterial thrombosis. Subperiostially, pus tracks both longitudinally and circumfrentially around the bone sripping the periosteum and interuupting the periosteal vessels consequently, larger areas of the cortex become infracted and involved in the inflammatory process. Without treatment pus finally bursts through the periosteum and tracks through the muscles to present subcutaneously and eventually through the skin. The pus is then discharged through a sinus which connects bone with skin surface.

Clinical features:

- Pain of sudden onset
- Fever
- Localized tenderness
- Irritable pt that resists examination
- Adjacent joint may contain an effusion (but not tender)
- Swelling
- Inability
- Inability to use the affected limb
- Discharging sinus.

Investigations:

1. Blood cultures: Thye should be done before commencement of antibiotic treatment. Three separate venepunctures should be made and cultured to provide maximum positive. Others include ,(but of no diagnostic value)
2. ESR which shows raised WBc (non specific)

3. X-ray

Is normal in the first few days of infectiont

Involucrum deposited by the elevated periosteum but is seen after the 10th day of infection.

Periosteal elevation.

Treatment:

1. Exercabations - Immobilzation and antibiotics (signs subside but only to recur again in life)
2. Surgical intervention whose objective is;
- To remove the dead bone (sequestrectomy)
- To eliminate the dead space

The soft tissues are stripped from the bone and the involucrum is removed to reach the sequestrum. If cavity is present, the overhanging walls are removed with an osteome. The wound is drained and closed in such a way as to eliminate dead space as far as possible.

Modern approaches;

- Debridement of affected area
- Insertion of gentamycin impregnated beads for 14/7
- Dead space obliterated by packing the cavitywith cancellous bone chips or filling it with a local muscle flplap.

NB:

- Operative intervention may not cure osteomyelitis if large volume of bone is involved.
- The most feared complication is amyloid disease

Amputation is considered when;

- There is frequent or pprolonged exacarbations to rid pt of repeated painful disability and prevent amyloid disease.
- A brodie's abscess should be treated by surgical evacuation and curettage of the cavity under antibiotic cover followed by packing with cancellous bone chips if cavity is big.

Complications:

General

1. Septicaemia
2. Pyaema

Local

3. Secondary involvement of adjacent joints
4. Spontaneous pathological fractures
5. Deformity
6. Chronic osteomyelitis

DDX:

1. Acut suppurative arthritis – sepsis is intraarticular
2. Acute rheumatic arthritis – Polyarticular, fleeting, history of sore throat and cardiovascular signs are present
3. Haemathrosis – may be due to haemophilia
4. Scurvy
5. Acute exanthemous and typhoid fever
6. Ewing's tumour

ACUTE TRAUMATIC OSTEOMYELITIS:

It occurs as a result of infected wounds like compound fractures and operaton on bones. General disturbance are less severe than in acute infective osteomyelitis because the causative wound provides some drainage.

Treatment:

- More extensive opening of the wound
- Removal of dead bone
- Antibiotics

Prevention:

1. Adequade initial treatment of compound fractures
2. Sterile operating conditions

CHRONIC OSTEOMYELITIS:

Pathology:

Acute haematogenous osteomyelitis may complicate to chronic osteomyelitis if treatment is not adequate or not available. The bone dies to form involucrum.

The pathology may take two forms;

a) A large volume of bone may be involved which has been explained under acute osteomyelitis. However this has reduced greatly due to the advent of modern antibiotics
b) The infection may closely contain to create a chronic abscess within the bone. This abscess is known as Brodie's abscess. It contains pus or jelly like granulationtissue surrounded by sclerotic bone. This abscess could be as a result of pyogenic septicaemia from which the pt has removed leaving a bone abscess which may remain dormant for yrs. alternatively it may be found in apt who has had osteomyelitis affecting a bone other than the one in which the Brodie's abscess is discovered.

Risk factors (conditions which predispose to bone infarcts)

- Open fractures
- Local trauma
- Presence of prosthetic orthopaedic implant
- Vacular insufficiency
- Neuropathy
- Sickle cell disease
- - Diabetes mellitus
- I.v drug use
- Haemodialysis

Clinical features:

It may remain quiescent for months or yrs. But acutor sub acute exercabations occur from time to time.

Constitutional (general) features;

- Generally sick looking
- Weak
- Febrile etc

Local features;

- Inflammation
- Chronic abscess with discharging sinus; changing in scar – hyperpigmentation, adherent to the underlying tissues.
- Chronic ulcers – non haeling with exposed bone
- **Brodie's abscess** –this is a special form of chronic osteomyelitis which arises insidiously, without a preceding acute attack. There is a localized abscess within bone, often near the site of the metaphysic. Its signs and symptoms include a deep "boring" pain is a predominant symptom. X-ray shows a circular or oval cavity surrounded by a zone of sclerosis. The treatment is surgery to

deroof and pus evacuated and if possible the cavity filled with a flap of muscle to obliterate the dead space.

X-ray shows a sequestrum which has separated from the space of the bone or lies a cavity

- Tomographs may show a sequestrum
- Sonograms may delineate an abscess cavity in the bone.

Management of chronic osteomyelitis:

- *Antibiotic therapy;*

– Fusidin acid, clindamycin and cephalosporins, to stop the spread of infection to healthy bone and to ontro acute flares.

- *Local treatment;*

- A sinus may be painless and need dressing simply to protect the clothing.
- Colostomy paste may be used o stop excoriation of the skin.
- An acute abscess may need urgent incision and drainage as a temporary measure.

Surgical operation;

1. The periosteum is incised to release any subperiosteal abscess. If none is found, the bone should be drilled to decompress the marrow and drain any intraosseus abscess.
2. Removals of dead bone – sequestrectomy and non-viable necrotic tisse (not bleeding or moving on touch) - are of utmost importance to effect cure. Pack the medullary cavity with local antibiotics e.g. gentamycin beads.
3. In pts with vascular insufficiency or severe gangrenous infection, amputation may be the only effective treatment.
4. If a large part is involved, wait for the involucrum to become strong and bigger than the sequestrum (3-6 months) then remove sequestrum otherwise you end up with septic non-union.

ARTHRITIS

TUBERCULOUS ARTHRITIS (TB ARTHRITIS) AND TB OSTEOMYELITIS:

PATHOLOGY;

Bone and joint tuberculosis is haematogenous in origin. The primary focus is from;

i) G astrointestinal tract – if the disease is acquired by ingestion of bovine tubercle or
ii) Lungs – if acquired by inhalation of the human strain

The disease starts in either in the synovial membrane or in the intra-articular bone. Tb may develop in any synovial joint especially those with extensive membranes like hip or knee joints. It may alsoaffect tendon synovial sheaths especially those of finger flexors or bursae like that overlying the greater trochanter.

The spine is not spared and here it is described as "potts disease". The vertebral bodies of the neighbouring vertebrae are almost always involved first. Typical tubercles develop in the synovial membrane. It becomes bulky and inflamed with an infected effusion collecting in the synovial cavity . If diagnosed and cured at this stage then full function of the joint may be restored. But if the pathology progresses, articular cartilage and adjacent bone is involved or destroyed. Loss of function is therefore certain due to healing by fibrous ankylosis by fibrous tissue. The articulating surfaces are replaced by fibrous tissue. Always assume involvement of both the synovial membrane and bone if a diagnosis of TB arthritis is made. This is because the synovial membrane is rapidly involved if the diseas e starts in the articular bone.

Tb spine is rarely diagnosed until significant involvement of neighbouring two vertebrae. Should ttreatment for Tb spine be delayed an abscess is formed and the vertebral bodies collapse. The pus from the abscess may track along the psoas muscle to present in the groin. Kyphosis and abscess formation compresses and may damage the spinal cord. This may produce paraplegia (pootts paraplegia).

Clinical features:

a) Symptoms;

They could be from the diseased joint, primary focus or systemic effects of the disease.

Locally:

- Aching joint initially mild in nature, but worse on exertion or at night
- Joint swelling if superficial and is more obvious when associated muscles are wasted.
- Joint stiffness as the disease progresses (can also be due to pain)
- Joint dislocation
- Local deformity may be obvious
- In Tb spine a mild ache may be a symptom of a potentially crippling disease.

Systematically:

- Pt feels unwell
- Listless
- Febrile especially at night
- Night sweats

b) Physical signs;

- Synovial thickening and effusion if joint is superficial
- Wasting of muscles associated with affected joint

- Joint held at its position of ease
- Moderate tenderness over affected joint
- Reddening of skin overlying the joint
- Slightly warm a feature of a cold abscess

Septic Arthritis

Mechanisms of infection

- Direct invasion through a *penetrating wound, intra-articular injection or arthroscopy*
- Direct spread from an *adjacent bone abscess*
- Blood spread from a *distant site*

Causal organisms

- Staph. Aureus - ***Most common***
- **Infants** - Haemophilus influenza
- **Neiserria gonorrhoea - *commonest*** *cause of septic arthritis in* ***adults***
- Others - Streptococcus, E.Coli, Proteus

Predisposing Conditions

- Rheumatoid arthritis
- IV drug abuse
- Immunosuppression - *Chronic debilitating disorders; Immunosuppressive drug therapy; AIDS*

Pathology

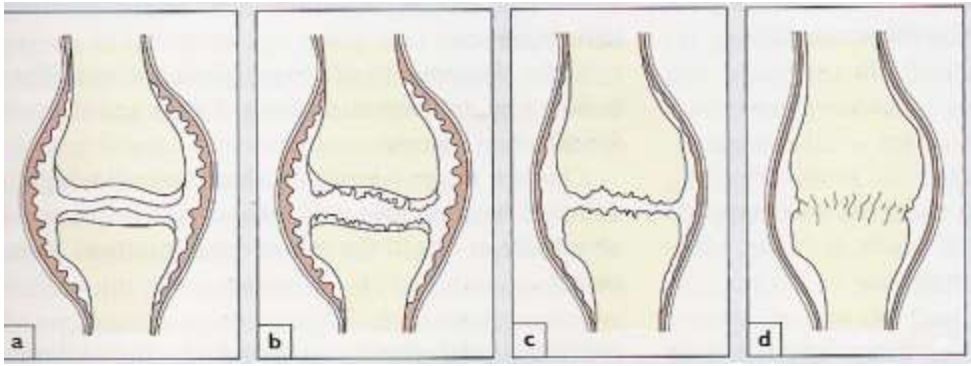
The usual trigger is a **haematogenous infection** which settles in the **synovial membrane**; there is an acute inflammatory reaction **- acute synovitis,** with a **serous or seropurulent exudate** & an **increase in synovial fluid**. As pus appears in the joint, **articular cartilage is eroded** & destroyed partly by **enzymes released from synovium, inflammatory cells & pus.**

In **infants**, the entire epiphysis, which is still largely cartilaginous, may be severely damaged; in **older children**, **vascular occlusion** may lead to **necrosis of the epiphyseal bone**. In **adults**, the effects are **usually confined to the articular cartilage**, but in the late cases, there may be extensive erosion due to **synovial proliferation & growth.**

If the infection goes **untreated**, it will **spread to the underlying bone** or **burst out of the joint to form abscesses & sinuses.**

With healing there may be;

- Complete resolution & a return to normal
- Partial loss of articular cartilage & fibrosis of the joint
- Loss of articular cartilage & bony ankylosis
- Bone destruction & permanent deformity of the joint



2.9 Acute suppurative arthritis In the early stage (**a**) there is an acute synovitis with a purulent joint effusion. (**b**) Soon the articular cartilage is attacked by bacterial and cellular enzymes. If the infection is not arrested, the cartilage may be completely destroyed (**c**); healing then leads to bony ankylosis (**d**).

C/P

- **Children** - Usually a large joint, commonly the **Hip**
- **Adults** - Superficial joints - **Knee, Wrist or Ankle**
- Acute pain
- Swinging fever
- Rapid pulse
- The overlying skin looks red
- Local warmth & marked tenderness
- Reluctance to move the limb (*'pseudoparesis'*) - *All movements are restricted, & often completely abolished, by* ***pain & spasm***

DDx

- Acute osteomyelitis
- Gout & pseudogout
- Trauma - *Traumatic synovitis or haemarthrosis*
- Irritable joint
- Haemophilic bleed
- Rheumatic fever - *typically pain flits from joint to joint*
- Gaucher's disease - *Presents as acute joint pain & fever without any organism being found ('pseudo-osteitis')*
- Bursitis
- SCD in crises

Ix

- X-ray - *Normal*
- FHG - ↑WBC & ESR
- Blood culture - *May be positive*
- Ultrasound;
    - Joint effusion
    - In **children** the joint 'space' may ***seem to be widened*** (because of the fluid in the joint) & there may be slight sublaxation of the joint.
    - With **E. coli** infections there is sometimes **gas** in the joint.
    - ***Narrowing or irregularity*** of the joint space are ***late features.***
- **Joint aspiration m/c/s** - Leukocyte counts **>50,000/ml**
    - Normal synovial fluid leukocyte count - <300/ml

- Non-infective inflammatory disorders - >10,000/ml

Mx

- **Aspirate** joint
- Give **analgesics** for pain & **IV fluids** for dehydration
- **Rest** the joint on a *splint or in a widely split plaster*; with **hip infection**, the joint should be **abducted & 30° flexed, on traction;**
    - To manage pain
    - To prevent dislocation
    - To keep the synovial cavity open to allow circulation
    - In children;
        - To prevent slipping of the upper femoral epiphysis
        - To strengthen the perichondral ring
- **Antibiotics;**
    - **<4years -** *Ampicillin or 3rd generation cephalosporins*
    - **Older children & Adults -** *Flucloxacillin & Fusidic acid* **IV** for **2-7days** & then **orally** for another **3wks**
- In children, give **cod-liver oil** which reduces inflammation by supplying Omega 3 reducing the formation of arachidonic acid necessary for the formation of prostaglandins that mediate inflammation.
- **Surgical eradication;**
    a. Under anaesthesia the joint is opened through a small incision, drained & washed out with physiological saline. *A small catheter is left in place* & the wound is closed; **suction-irrigation** is continued for another **2-3days**. This is advisable;
        - **In very young infants**
        - **When the hip is involved (*Joint is opened from behind*)**
        - **If the aspirated pus is very thick**

    b. For **knee**, ***arthroscopic debridement*** from the *lateral aspect* & copious irrigation may be equally effective

    c. Older children with **early septic arthritis** (symptoms for **<3days**) involving any joint **except the hip** - ***Repeated closed aspiration*** of the joint; however, *if there is no improvement within **48hrs, open** drainage will be necessary.*

Post-op;

- **Intact** articular cartilage - ***Physiotherapy***
- **Destroyed** articular cartilage - The joint is ***splinted*** in the optimum position awaiting *ankylosis (stiffness or fixation of a joint by disease or surgery)*

Complications

- Bone destruction
- In adults, *partial destruction of the joint* will result in ***2° Osteoarthritis***
- Cartilage destruction -may lead to either ***fibrous or bony ankylosis***
- ***Growth disturbance*** - presents either as a localized deformity or as shortening of the bone
- Dislocation of the ***hip***
- ***Osteomyelitis***

Osteoarthritis

Is a chronic joint disorder of **post middle age** in which there is **progressive softening & disintegration of *articular cartilage*** accompanied by ***new growth of cartilage & bone*** *at joint margins (osteophytes)* & ***capsular fibrosis.***

Epidemiology

Most patients are **past middle-age (50yrs); M:F - 3:1**

When it occurs in younger patients, it is usually **2°** & develops if *articular cartilage* is **damaged** or *subjected to* **abnormal stress.**

>80% of persons 55 years old show radiological evidence of osteoarthritis but only **25%** have clinically significant symptoms.

Causes

1. **Primary**

   Develops without any obvious underlying cause & is best characterized by ***1° generalized nodal osteoarthritis***, a disorder affecting many joint groups, including;

   - Hips
   - Knees
   - Zygapophyseal joints of the spine

   Also;

   - Elbow/Ankle
   - IP joints of the fingers & toes

   Studies have shown that there is a significant **increase in bone density** in people with osteoarthritis which is determined by a variety of **genetic, hormonal & metabolic** factors which may also ***influence cartilage metabolism*** independently of any effect due to bone density. Women with osteoporosis **seldom** have osteoarthritis.

2. **Secondary -** This is as a result of ***increased stress, weakened cartilage or abnormal support of cartilage*** e.g. avascular necrosis

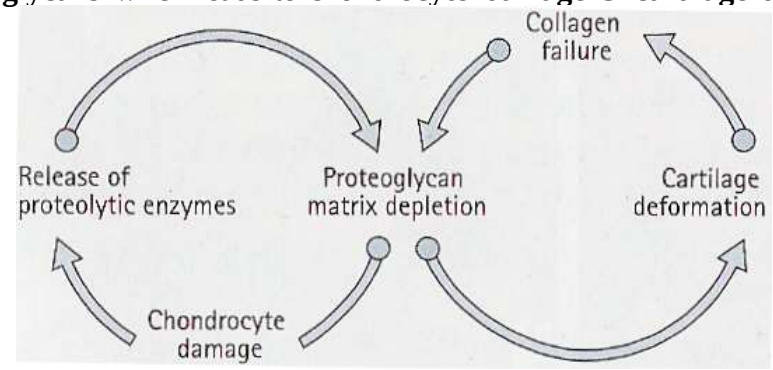
   - **Genetic or developmental**
     - **Congenital hip dislocation**
     - **Slipped upper femoral epiphysis**
     - **Chondrodysplasia**
     - **Perthe's disease**
     - **Genu valgum or varum**
     - **Haemophilia**
   - **Metabolic**
     - **Hyperuricaemia**
     - **CPPD arthropathy**
     - **Alkaptonuria**
     - **Gaucher's disease**
   - **Endocrine**
     - **Diabetes mellitus**
     - **Hypo/Hyperthyroidism**
     - **Acromegaly**
   - **2° to Inflammatory Disorders**
     - **Septic arthritis**
     - **Rheumatoid Arthritis**

- **Ankylosing spondylitis**
- **Psoriatic arthritis**

- **Trauma**
  - **Fractures (*particularly osteochondral fractures*)**
  - **Joint instability (e.g. *cruciate ligament injury, joint hypermobility syndromes*)**
  - **Post meniscectomy**
  - **Osteochondritis dissecans**
  - **Neuropathic joints (*Charcot joints*)**
  - **Mechanical causes including *leg length discrepancy, instability, repetitive (occupational) injuries***

Pathogenesis

This is thought to be as a result of ***intrinsic disturbances in the metabolism of cartilage*** which leads to **increase in water content** of the cartilage & **easier extractability of the matrix proteoglycans** which leads to **chondrocyte damage & cartilage deformation.**



Cardinal features;

i. **Inflammation** leads to **progressive cartilage destruction** forming an area of **fibrillation**, which is a hair-like patch where the cartilage matrix components are lost, leaving only a skeleton of disrupted collagen fibres attached to the bone below.

ii. **Subarticular cyst formation** in the marrow below the subchondral bone from extrusion of joint fluid through the hyaline cartilage clefts into the marrow, with a *fibroblastic and osteoblastic cellular* reaction leading to ***granulation tissue*** formation in the cyst.

iii. **Sclerosis of the surrounding bone** due to *increased synthesis of bone by subchondral osteoblasts,* presumably prompted by intercellular communication by cytokines between chondrocytes and osteoblasts. With increased bone formation in the subchondral area, physical properties change; the *bone becomes stiffer with decreased compliance, and microfractures occur*, followed by callus formation, more stiffness, and more microfractures. The term **eburnation** applies to the glistening appearance of the polished sclerotic bone surface.

iv. *Metaplasia of the **peripheral synovial cells*** results in ***peri-articular*** formation of **osteophytes** (or, more correctly, *osteochondrophytes*, consisting of bone and a mixture of connective tissues with a coating of fibrocartilage and sometimes islands of hyaline cartilage within the osteophyte) and in ***subchondral bone,*** especially in areas denuded of cartilage.

v. **Capsular fibrosis -** There is **NO primary change** in the capsule or synovial membrane, but the recurrent strains to which an osteoarthritic joint is subjected to often leads to slight ***thickening & fibrosis*** *of the capsule or synovial membrane*

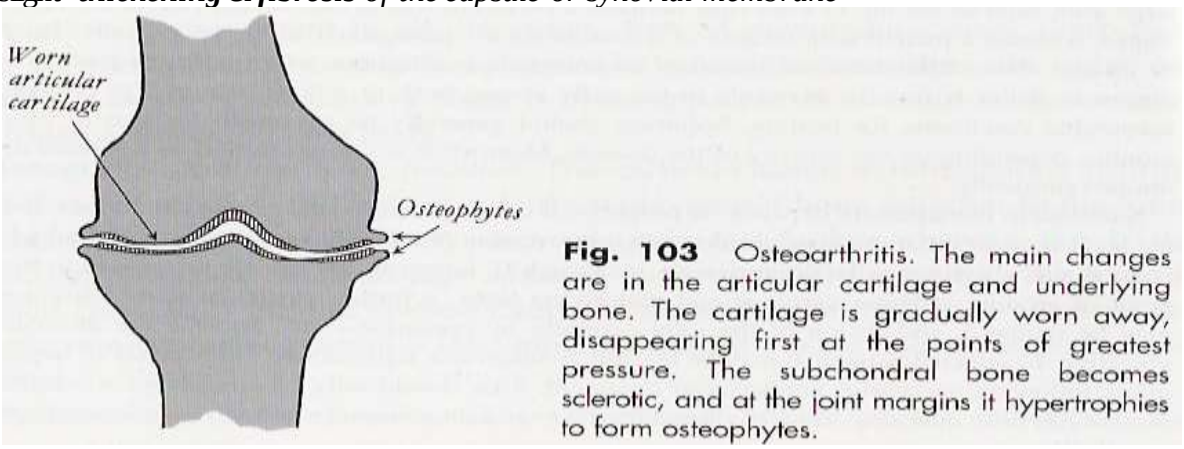


Fig. 103 Osteoarthritis. The main changes are in the articular cartilage and underlying bone. The cartilage is gradually worn away, disappearing first at the points of greatest pressure. The subchondral bone becomes sclerotic, and at the joint margins it hypertrophies to form osteophytes.

S/S

Symptoms characteristically wax & wane, & pain may subside spontaneously for long periods.

Commonly affected joints

- DIP
- Thumb MCP
- Cervical & lumbar spine
- Knee

Early Osteoarthritis

- ***Pain/Tenderness*** *(worse at the end of the day; background pain at rest*) due to;
  - Exposure of nerve endings 2° to bone erosion
  - Capsular fibrosis → shrinking, with pain on stretching
  - Bone pressure due to vascular congestion
  - Muscular fatigue

Moderate Osteoarthritis

- ***Stiffness*** **-** In larger joints, movement is accompanied by *palpable or audible coarse crepitations.*
- ***Swelling*** due to;
  - Intermittent - *Effusion*
  - Continuous;
    - *Capsular thickening*
    - *Large osteophytes*
      - Gives the appearance of nodes in the *PIP* - **Bouchard's nodes**

▪ "

Severe osteoarthritis

- ***Deformity*** due to;
  - Capsular contracture
  - Joint instability

Fixed deformity (inability of the joint to assume the neutral anatomical position) is often found in the *Hip*, & sometimes at the *Knee & in other joints.*

- ***Loss of function***

Ix

- Cardinal features on **X-Ray;**
  - **i.** **Asymmetric *narrowing*** *of joint space*
  - **ii.** *Sclerosis of subchondral bone* under the area of cartilage loss
  - **iii.** *Subchondral cysts*
  - **iv.** *Osteophytes* at margins of joints

  Also *features of previous disorders.*
- ↑ **CRP**
- **Radionuclide scanning** (^{99m}Tc) - shows increased activity during the *bone phase in the subchondral regions* of the affected joints. This is due to *increased vascularity & new bone formation.*

Mx

a. Early treatment principles;
- **Relieve pain** - ***Analgesics & Anti-inflammatory therapy***
- **Reduce rate of degeneration** - ***Proteoglycan matrix supplements*** **e.g. Glucosamine sulphate, Chondroitin sulphate**
- **Protect the joint from 'overload'**
  - **Reduce weight**
  - **Supportive footwear**
  - **Walking aids**
- **Modify daily activities**
- **Exercises**

b. Intermediate treatment
- Maintain movement & muscle strength – ***Physiotherapy***
- Injection of ***depot*** *intra-articular* ***long acting steroids*** **6monthly** (S/E - *Osteoporosis*)

c. Late treatment;

Indications;
- Must be demanded by the patient
- Keeps patient awake at night
- Reduced walking distance to **<100m**
- Confinement of activity

i. Minimally invasive procedures - ***Arthrotomy, Arthroscopy***

ii. ***Intertochanteric Realignment Osteotomy*** **-** It must be done while the joint is still stable & mobile (usually in patients **<50yrs old**) & x-rays show that a major part of the articular surface (the radiographic 'joint space') is preserved. Objectives;
- To change the orientation of the femoral head in the socket so as to *reduce mechanical stress in a damaged segment*
- By realigning the proximal femur, to *improve joint congruity*
- By transecting the bone, to *reduce intraosseous hypertension & relieve pain*

- An unintentional & poorly understood consequence is *fibrocartilaginous repair of the articular surface*.

iii. ***Arthrodesis*** (surgical immobilisation of a joint so that the bones grow solidly together) - is indicated if the *stiffness is acceptable & neighbouring joints are not likely to be prejudiced* e.g. Lumbosacral tilting & rotation. This is a practical solution for young adults with marked destruction of a single joint.

iv. ***Total joint replacement*** *for hip & knee* - because of the *tendency for implants to loosen with time*, joint replacement is usually reserved for patients aged ≥**65yrs**

Mechanical considerations;

- The prosthetic implants must be durable
- They must permit slippery movement at articulation
- They must be firmly fixed to the skeleton
- They must be inert & not provoke unwanted reaction in the tissues

The usual combination is a **metal femoral component** (*stainless steel, titanium, cobalt-chrome alloy*) articulating with a **polyethylene socket**

Complications;

Intra-op;

- Perforation/fracture of the femur or acetabulum
- Sciatic nerve palsy (usually due to traction but occasionally caused by direct injury)

Early;

- Infection
- DVT
- Dislocation
- Myositis ossificans

Late;

- ***Aseptic*** *loosening* due to granuloma formation of either the acetabular socket or the femoral stem is the ***commonest*** *cause of long-term failure*
- *Stress shielding* - Aggressive osteolysis with or without implant loosening
- Infection
- Dislocation

SPINA BIFIDA

Def. a congenital condition inwhichthe elements

Pathology:

There is failure of total closure of the embryonal neural tube or the mesodermal tissue.

Closed lesion which denotes an intact skin

Open lesion which denotes deficiency in the skin hence exposure of nerve tissue posing a danger of infection leading to primary or secondary paralysis.

1. Primary paralysis will present at birth due to failure of part of the spinal cord to develop and this is called myelodysplasia.

Meningomyelocele – it is a meningeal sac containing both the meninges and the nerve root. If the nerve tissue remains in the primitive state then it is called a myelocele giving rise to leakage of CSF leading to hydrocephalous. It may lead to functional impairement or complete paralysis of the muscles of the lower limbs.

2. Secondary paralysis due to infection

Clinical featurd more and es;

It is difficulty to assess in infants

c) Motor paralysis: happens in the lower limbs and the trunk. It can be very severe or mild.

Severe- flexors of the hip are affected more and adductors, quadriceps and tibialis anterior are spared.

d) Sensory paralysis
e) Pressure sores even at birth

Visceral paralysis mainly the bladder and the bowels leading to Incontinence of urine and stool

f) Hydrocephalus due to malformations of the ventricles in the cerebellum affecting the flow of CSF mainly the 4th ventricles.

Principles of treatment:

1. Correction of the deformity
2. Maintainance of the correction
3. Promotion of possible function in the affected limb.

GLANDS:

THE BREAST:

Comparative and surgical anatomy:

This is the protruberant part of the human breast which overlies the 2nd to the 6th ribs. It extends from the lateral border of the sternum to the anterior axillary line. In actual fact a thin layer of mammary tissue extends considerably further from the clavicle above to the 7th to 8th rib below and from the midline to the edge ot lattismus dorsi posteriorly. This fact is important when performing a mastectomy, the aim of which is to remove the whole breast.

The breasts are modified sweat glands in that they are embryologically derived from a downward growth of ectoderm into the underlying messemchyme. The first stage of development occurs at 6th or 8th wk of gestation, when two strips of thickened ectoderm, the mammary ridges grow in aline extending from the embryonal axilla to the inguinal region. In many animals breasts develop along a whole length of the ridge, but in humans true breast tissue occurs only in the pectoral region. The breast overlies the pectoralis major, seratus anterior, and external oblique muscles. Medially, the breasts reach the sterna edge and laterally the midclavicular line. The pyramidal axillary tail extends into the axilla. The tail is of surgical importance. In some cases it is palpable, and in a few it can be seen premenstrually or during lactation. A well developed axillary tail is sometimes mistaken for a mass of enlarged lymph nodes or lipoma.

The lobule is the basic unit of the mammary gland. The number and size vary enormously. They are most numerous in young women. From 10-100 lobules empty via ductules into a lactiferous duct of which there are from 15-20. Each lactiferous duct is lined by a spiral arrangement of contractile myoepithelial cells and is provided with a terminal ampulla- a reservoir for milk or abnormal discharge.

The ligaments of cooper are hollow conical projections of fibrous tissue filled with reast tissue, the apices of the cones being attached firmly to the superficial fascia and thereby to the skin overlying the breast. These ligaments account for the dimpling of the skin overlying a carcinoma. The areola contains involuntary muscles arranged in concentric rings as well as radially in the subcutaneous tissue. The areola epithelium contains numerous sweat glands and sebaceous glands enlarge during pregnancy and serve to lubricate the nipple during lactation (Montegmery's gland)

The nipple is covered by thick skin with corrugations. Near its apex lies the orifice of the lactiferous ducts.the nipple contains smooth muscle fibres arranged concetricallyand longitudinally, thus is an erectile structure which points outwards. Lymphatics of the breast drain predominantly into the axillary and internal mammary lymph nodes receive approximately 75% of the drainage.

Blood supply:

Internal mammary artery

Branches of the axillary artery i.e.

The thoracoacromial

Subcapsular

Lateral thoracic.

Innervations:

It is primarily by sensory and sympathetic nerves. The nipple has a rich sensory supplty while most of the sympathetic innervations are to the breast parenchyma. Nerves of the breast are principally derived from the 4^{th}, 5^{th} and 6^{th} intercostals nerves.

Cyclic changes during the menstrual cycle:

Volume varies during the menstrual cycle. The volume is greatest in the second half of the cycle, after a premenstrual increase in size, nodularity, density and sensitivity. Progesterone may stimulate glandular growth in the lateral phase than the follicular phase.

In pregnancy:

There is dramatic increase in secretion and release of circulatingovarian and placental estrogen and progesterone. The gland enlarges. The areola skin darkens. The areola glands become prominent as ducts and lobules proliferate. The proliferating glandular epithelium replaces connective tissue and the components of adipose tissue.

Postmenopausal breast:

There is concomitant decrease in ovarian secretion of estrogen and progesterone. There is involution of ductular and glandular components.there is loss of fat content and supporting stroma, therby initiating loss of lobular structure, density, form and contour.

Gynaecomastia:

Presence of female type mammary gland in the male. In most cases it should not be considered a disease because male breast enlargement is common. Physiologic gyneacomastia occurs mostly during three phases of life;

i) Neonatal period
ii) Adolescence
iii) Senescence (growing old)

It is usually due to excess estrogen in relation to circulating testerone.

Neonatal type is due to the action of placental estrogen on neonatal breast parenchyma

In adolescence there is an excess of estradial relatve to testerone

With aging the plasma testerone levels fall with a relative hyperestrinigism.

Drugs with estrogens or estrogenic activity (digitalis, estrogens, anabolic steroids, marijuana) may cause gynaecomastia.

Drugs that inhibit the action or synthesis of testeroe (cimitidine, ketoconazole, phenyntoin, spirinolactone, antineoplastic agents, and diazepam) may also be implicated.

Clinical breast problems:

Most breast complaints that cause a woman to seek medical attention are benign. The problems may be divided into four general categories of;

a) Breast pain
b) Nipple discharge
c) Breast masses
d) Breast infections.

a) Breast pain (mastalgia):

It is a common problem which is rarely a presenting sign of breast carcinoma. It may originate from the breast itself or reffered from extramammary structures like ribs, vertebrae, or occasionally the teeth. The pain can be cyclic or non cyclic on the basis of its relationship to the menstrual cycle. The cyclic pain wanes with the menstrual cycle and is frequently bilateral. It usually involves the upper outer quadrant of the breast and radiates to the axillae down the arms. It tends to be more severe immediately before the menses. Non cyclic pain occurs in postmenopausal women or when seen in premenopausal women, bears no relationship to the menstrual cycle. It is more commonly unilateral, localized and described as sharp and stabbing or burning. Mondor's disease(thrombophlebitis of the lateral thoracic or superior thoracoepigastric vein)may cause breast pain.

b) Nipple discharge:

This is a common complaint but an uncommon sign of carcinoma of the breast. Only 3-11% of women with ca breast have an associated nipple discharge. 99% of women presenting with nipple discharge, the cause is benign. It is good to determine whether they are physiologic or pathologic.

Discharges are classified as if they are spontaneous and localized to one duct. Pathologic diseases may be;

Bloody or

Serous and

Are almost always unilateral.

Physiologic discharges occur only with nipple compression

Frequently originate from multiple ducts, and

Usually bilateral

Fluid can be expressed from the nipples of approximately 80% premenopausal women.

5. Pts with physiologic discharge should be advised against squeezing their nipples and there is no therapy required.
6. Pathologic discharges should include testing the fluid for occult blood and identifying the quadrant of the breast from which the discharge originates.
7. 70-80% of discharges associated with ca contain blood.

A non bloody discharge that meets the other criteria of pathologic discharge is an indication for breast biopsy.

Cytology is not usually useful in the evaluation of nipple discharge because the absence of malignant cells does not reliably exclude.

Galactorrhea: This is a non puerperial discharge of milky fluid from both nipples. It differs significantly from that of other forms of nipple discharge. It is not a sign of primary breast pathology and should prompt a work up to exclude an underlying endocrine disorder. It is normal if intermittently secreted up to 2yrs after breast feeding has stopped. Galactorrhoea may be secondary to;

Hypothyroidism

Pituitary adenoma

Chest trauma (including thoracotomy)

A variety of medications- including oral contraceptives, phenothiazines, tricyclic antidepressants, metoclopromide, and reserpine, also cause galactorrhoea.

Causes of nipple discharge:

Solitary papilloma is the most common cause

Ductal ectasia is classically thick and cheesy "tooth paste" and common in the aging women above 50yrs.

Carcinoma but not common

c) Breast mass:

It may be cystic or solid and are characterized by their persistency throughout the menstrual cycle.

They may descrete or poorly defined but they differ in character from the surrounding breast tissue. Often what the patient perceives as a breast mass is actually a normal variant of breast tissue.

d) Cysts:

They are a common cause of dorminant breast mass.

Peak incidence in women in their 40s and perimenopauseal yrs.

They are;

Well demarcated from the surrounding breast

Mobile and

Firm.

Difficult to distinguish from solid lesions on physical examination although yheu fluctuate with the menstrual cycle, but solid lesions do not. Cystic lesions in postmenopausal women who are not on hormone replacement therapy are uncommon and should be regarded with a high degree of suspicion than those in premenopausal women because they may be secondary to ductal obstruction by a malignant lesion. It should be evaluated by aspiration. If fluid is not groos ly bloody and the mass restores completely, no further therapy than follow-up examination.

SOLID MASSES OF THE BREAST:

FIBROADENOMA:

It presents most frequently in pts between the age of 20 and 50 yrs characteristic clinical presentation.

It is a well defined palpable mass that is rubbery in texture and mobile.

It is usually solitary but may be multiple in 10-15% of cases.

They are thoyght to be the result of a minor aberration in the process of lobular development.

Hormonal factors appear to be important in their growth, because they involute after menopause and they dramatically increase in size during pregnancy. In postmenopausal women receiving only estrogen fibroadenomas may increase in size relative to the surrounding breast parenchyma.

Fibroadenomas typically stop growing when they reach 2-3 cm in diameter.

Blacks are more affected than whites and in early age in rare circumstances, fibroadenomas have been associated with carcinoma.

FIBROCYSTIC DISEASE:

This is a common term used to describe a variety of benign breast disorders. It is not a clinical entity because it encompasses a heterogenous group of processes some pathologic and some physiologic, with a wide varying cancer risks. The term fibrocystic change is not a synonym for lu mpy breast. It should be reserved for women in whom a breast biopsy has demonstrated one of the histologic components of fibrocystic change. When a breast biopsy is performed for vague areas of nodularity that lack mammographic or ultrasonographic correlates a fibrocystic process is the diagnosis.

BREAST CANCER:

It is the most commoncancer in American women and the second most common cause of death. It a major cause of cancer death in most industrialized nations.

Risk factors:

1. **Age;**

Age is the most common risk factor

Half of women's lifetime risk for breast cancer development occurs after age 65yrs.

Between the age of 35-55yrsthe risk for breast cancer development is only 2.5%

Breast cancer at a young age is more common in black women than in white women.

2. **Gender;**

e) Breastcancer is 100 times more common in women than in men. Female sex is therefore a major risk factor.

3. **Family history;**

Approximately 20-30% of women with breast cancer have a family history of the desease. But only only 5-10% have an inherited mutation in a breast cancer susceptibility.

4. **Hormonal factors;**

Studies have linked breast cancer risk to age at menarch, menopause and pregnancy.

The increased number of ovulatory cycles associated with early menarch, nulliparity, and late menopause appears to be the common mechanism of risk. Women who undergo bilateral oophorectomy before menopause are at decreased risk. Generally hormornal risk factors are associated with relative risks in the range of 1.5- 2.0. Long duration of lactation appears to reduce risk in menopausal women. Post menopausal obesity has also been shown to increase risk perhaps through an increase in peripheral estrogen production.

5. **Dietary:**

There is a relationship between dietary mammal fat and the incidence of breast cancer. Fried high fat foods can increase the risk of developing breast cancer approximately twofold.

6. **Breast feeding and menopause:**

Women in whom menopause occurs after the age of 55yrs the risk is twice as those women whose menopause started below the age of 45yrs. Artificial induced menopause appears to be protective for breast cancer and the protection is lifelong. Breast feeding of long duration (< 36 months in a lifetime) was thought to reduce the risk of breast cancer.

7. Child bearing and fertility:

Infertility and nulliparity are associated with a high probability (30-70%) for ca btreast. Women impregnate before 18yrs of age whom have a full term pregnancy have a breast cancer risk. Women who have their first full term pregnancy after age 30yrs have an even more risk for breast cancer than nulliparous.

8. Multiple primary neoplasms:

Women with a history of primary breast cancer have a risk three – four times higher for primary cancer in the contralateral breast.

9. Irradiation:

Exposure to ionizing radiation, whether from nuclear or medical procedures increases the risk for breast cancer. The level of risk varies with age at exposure being greatest for exposure in childhood and adolescence, and minimal foe exposures in after 40yrs. Pts who were treated with irradiation for Hodgkin's lymphoma in their adolescent or childhood yrs are the group at risk on the basis of radiation exposure most commonly encountered today.

10. Benign breast disease:

Benign breast lesions are classified as non proliferative, proliferative, or proliferative with atypia. Proliferative without atypia are associated with a small increase in breast cancer risk. Proliferative lesions with atypia are uncommon.

HOST TUMOUR RELATIONSHIP:

The growth rate of ca breast and the ability to metastize are determined bythe balance between the biological behavior of the tumour and the immune response of the host. The growth potential of the tumour and resistance of the host vary over a widevrange from pt to pt and may be altered during the course of the disease.

In arapidly growing tumour, its cells double to about 23days from one cell to two cells.

In the slow growing tumour the cells double in 309 days. If the range of doubling is constant and then the ca cellsrise from one cell of origin for the tumour to grow 1cm in diameter and clinically be detectable it will take about 8yrs. The slow growing tumours will take even between 15-30yrs and sometimes the the tumour can change its course, i.e. remain a benign breastlump for life. The normal periods of 5yr survival rate for untreated ca is 3yr but some take 4-5yrs and survive longer than that.

METASTASIS:

It .will determine the method andchoice of treatment

1. Regional lymph nodes:

Axillary and internal mammary nodes are the major routes of spread. Supraclavicular nodes are involved secondarily because they are in continuity with the axillary nodes. If the supraclavicular nodes are involved, it shows that there is distant metastasis and surgery here becomes a waste. The internal and axillary glands may be involved differently or both. When the axillary nodes are involved the ca in this case is operable.

2. Distant metastasis

This is mostly by haematogenous spread. The following sites are common;

The bones especially in the spine, pelvis, ribs, femur, skull, humerus,

Lungs (most frequently involved)

Liver " " "

The haematogenous spread affects the 5yr survival rate because it is fast in growing of the tumour.

CLINICAL FINDINGS:

Always start with history ecause it will give you 80% of the diagnosis. Combined with physical examination, characteristics of the lesion and if there is any metastasis will help you in spot diagnosis.

There is always a lump in the breast.sometimes the patient may come very late when an ulcer has already developed, fungating with axillary lympadenopathy or oedema of the arm confirms the diagnosis.

It can be confirmed more by histopathology in addition to other investigations.

All these will help determine the method or choice of treatment after stagin;

a) Symptoms: Note the following apart from the history of the patient.

Menarch

Pregnancy (parity)

Artificial or natural menarch

Date of last normal, menstrual period

Any previous lesions

Family history of ca breast

Pains of metastasis especially systemic complaints e.g cough, haemoptysis shows diatant metastasis. About 80% of the patient will complain of a painless breast lump and 90% will diagnose themselves.

Less frequent symptoms:

Nipple discharge

Redness

Generalized hardness of the breast

Shrinking of the breast

Rare symptoms:

Axillary mass

Swelling of the arm or

Bone involvement may be the 1st symptom/ sign.

Examination of the breast: refer to your clinical methods but note the following;

Variation in size –compare with the normal one and colour of the breast

Minimal nipple retraction because with ca are retracted upwards because of the ca fixation to the skin.

Palpate the lymph nodes –size, mobility, fixed?, tender.

Mass – size, mobility, fixed, tender, firm, fixed to the chest wall or the skin.

Breast ca usually presents with the following;

Non tender mass

Firm as tip of your nose or

Hard lump with poorly defined margins because of local infiltration

Skin or nipple retraction

Breast may be asymmetrical

Waterly,serous or bloody discharge from the nipple

In advanced cases it may present with the following;

Oedema of the arm

Peau de orange

Redness

Nodularity with laceration of the skin

Presence of a large fungating tumour

Fixation to the chest wall

Marked axillary lymphadenopathy

Supraclavicular lymphadenopathy and distant metastasis

The breast is divided into four quadrants i.e

Lateral upper – 45%

Lateral lower – 10%

Medial upper – 15%

Medial lower – 5%

Around the nipple – 25%

The commonest site is the lateral upper quadrant.

OTHER CLINICAL FORMS OF Ca BREAST:

a) Paget's disease;

Incidence -3% of all breast ca

It is an intraductal ca well differentiated, mid-centric in the nipple and ducts

The nipple epithelium is also infiltrared

Symptoms:

Itching or burning of the nipple

Erosion and ulceration but superficial and is confirmed by biopsy.

DDX

Dermatitis artefacta

Bacterial infection of the nipple

b) **Inflammatory ca**:
clinical findings

Rapidly growing tumour

Painful +/- enlargement of breast

The skin becomes erythematous, oedematous and warm

Incidence – is 3% of all breast Cas

Diagnosis:

When the redness is more than1/3 of the underlying skin of the breast

DDX:

g) Any infective process of the skin like cellulitis.

Treatment: difficulty as metastases are rapid and very early hence it is rarely curable but you can do radical mastectomy but the cure rate is very low and therefore;

Irradiation

Cytotoxic drugs or

Hormonal therapy

c) Mastitis carcinomatosis:

This is ca occurring during pregnancy

Incidence – 1-2% of all breast Cas

Usually occurs in women at the age of < 35yrs, concurrent with pregnancy

Diagnosis:

Is delayed because of physiological changes (in the breast) due to pregnancy

DDX:

Physiological changes of breast in pregnancy.

Laboratory findings in ca breast:

If confined to the breast and axillary lymph nodes it causes no abnormalities. However if the blood findings are abnormal e.g. ^ ESR and low Hb it shows disseminated ca.

Liver metastasis may be associated with increased alkaline phosphayases

Hypercalcaemia is another finding in advanced ca. i.e L.F.Ts will be abnormal.

X-ray findings:

Lungs, spinal column and long bones will be affected due to metastasis.

Scanning of the bones:

Ct scan detects early bone metastasis to the liver, lungs and brain

Mammography:

Soft tissue x-ray will detect the ca before the mass is palpable as early as 2yrs before the ca becomes palpable. It is also good for screening.

Indications for screening:

1. Evaluate the opposite breast when the diagnosis is confirmed in the other breast.
2. To evaluate ill defined masses, nipple discharges, erosions or retractions of the nipple or skin, skin dimpling or breast pain.
3. To search for occult breast (mass screen) for all child bearing groups (14-45yrs).

Dangers of mammography:

a) Repeated ionization may cause ca of the breast as radiationsare carcinogenic.
b) Dangers of false positives may subject the woman to unnecessary breast operations
c) Dangers of false negatives as in lobular ca mammography should be done to both breasts i.e the normal and diseased.

Biopsy:

For histopathology is the gooa method to any ca of the body

Specific indications for breast biopsy:

i) Persistant mass
ii) Bloody nipple discharge
iii) Eczematoid nipple

Methods of biopsy:

It depends on the operation;

a) Needle biopsy (Vim Silverman needle)
b) Open biopsy

Frozen biopsy takes about ½ hr(not reliable)

Paraffin wax biopsy (reliable)

Cytology:

Examination of the nipple discharge or cystic fluid,but must be confirmed with open biopsy of paraffin wax.

DDX OF CA BREAST:

In order of frequency;

1. Mammary dysplasia (cystic disease of the breast)
2. Fibroadenoma
3. Intraductal papilloma
4. Fat necrosis (following trauma)
5. Breast abscess

Management of ca breast:

Tou should start with staging of the ca. it will help in the managementbecause mxn varies with every stage.

Stage I:

Tumour is less than 5cm in its greatest diameter

Skin fixation is absent or incomplete

No fixation to the underlying muscles

Axillary nodes not palpable

Stage II:

As in stage I above but with palpable mobile nodes in the homolateral.

Stage III:

Tumour more than 5cm in its greatest diameter or

Skin fixation complete or

Skin involvement + wide fixation

Palpable mobile nodes

Peau de orange

Fixation to underlying muscle

Palpable axillary and supra clavicular nodes or

Oedema of the arm because of the lymphatic drainage.

Stage IV:

Distant metastasis regardless of condition of the primary and the regional nodes

Treatment may be curative or palliative.

1. **Stage I and II cases**
3. Treatment is curative. Excision offers the best method of cure. Halsted radical mastectomy or local simple mastectomy combined with RT in all cases. Local simple mastectomy is also called standard radical mastectomy where lymphnodes are also removed especi ally the intermammary l'nodes.
4. Can also do simple mastectomy which is best for the ca which is confined to the breast without spreading to adjacent organs (not beyond stage I)
5. Local excision is known as lumpectomy.

Stage III:

Usually palliative but this stage is sometimesborderline. Radical mastectomy is contraindicated here since we are most likely not going to eradicate the tumour because of the metastasis. Instead the lymphatic channels are opened up and this will end up spreading the tumour cells to th e rest of the body. What is done here is simple mastectomy followed with RT post operatively. Some centers do pre-opRT then post- op RT then followed by Halsted method.

Stage IV:

This is only palliative. Do the following;

Raise the nutritional status of the pt i.e. HPD, BT, Haematenics

Simple mastectomy(in fungatingpart) + RT

Other metastasis like pleural effusion may be removed by RT

Dress the wound with antiseptics

Cover with antibiotics to treat all infectios promptly

Progress of the disease may be controlled by

8. Hormonal therapy
9. Endocrine surgery
10. Cytotoxic drugs

Hormonal therapy and endocrine surgery:

Hormonal therapy is applied because 40% of the breast cas are hormone dependent. So the course of the disease may be slowed down or regressed by altering the pt's hormonal balance. Then the pt will be relieved of pain.

Nutrition of the pt will improve

The tumour will regress locally

Osteolytic lesion will calcify

Pathological fractures will heal

Unfortunately the disease always procedds sometimes very fast after the tumour cells have lost their dependency on the hormones about 4-6 months.

Two types of hormones are used;

a) **Pre-menopausal women**:

Here surgry is used by removing the ovaries. This will always delay the progress of the disease. Advise the pt to admit to have oophorectomy. The ovaries can be removed by minilaparatomy or by radiation which is good for the weak pts

b) **Postmenopausal women**:

Oestrogen is used in this pts. Before its usage make sure that there is no estrogen activity or else you will aggravate the condition. You must make several vaginal smears to ascertain the estrogen activity has stopped. Tumour remission rate from estrogen (also to androgens as well) tend to increase with increasing number of yrs past the menopause. If the therapy is given for 6 months and there is no response, stop the procedure and peoceed with other methods or sometimes you can combine with adrenolectomy or hypophysectomy.

c) Corticosteroids:

Are also of help e.g. cortisone about 150mg or predinsolone 30mg bd or tid. They reduce pain. Other symptoms disappear.

d) Chemotherapy:

Cytotoxic drugs which are only used for palliation in advanced metastasis and also when the hormonal therapy has failed. The drugs include the following;

Durorubicin (adriamycin) i.v 40-50 % good response although it is short. Combinations also show good results e.. dororubicin + cyclophophamide.

Others: cyclophosphamide + vincristine or methotrexate or flurouracil.

Prognosis:

Usually poor in males because of the tumour attaches directly to the chestwall even if inearly stage. The crude 5yr survival rate is about 36- 17 %. In women when the ca is confined to the breast the 5yr cure rate by radical mastectomy is 75-90%. Involvement of the axillary nodes is between 40-66% at 5yrs. The course by anatomical site for the breast ca is the medial portion of the inner lower quadrant. Lymphatic drainage or capillaries of the breast form anastomosing networkwhich is continous across the midline with that of opposite site below with that of the abdominalwall. They all correspond to the arteries supplying the glands. Hence this part of the breast/ chestwall have got extensive supply of lymphatic vsls as compared to the other quadrants. Breast ca is more malignant in the old (440% are hormone dependent cancers).