



3. Obstetrics and Gynaecology

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1. NORMAL PREGNANCY

Antenatal period is the most crucial period as the services provided during this period can have positive impact on health of both the mother and her child.

Essential obstetric care to every pregnant woman

1. Registration before 12 weeks

2. Detailed history at first visit

- Menstrual history: Regularity of cycles, date of LMP, calculate EDD and record on Mother Child Protection (MCP) card
- Obstetric history: Number of prior pregnancies and outcome of each pregnancy (full term birth, preterm birth, abortion), place and mode of delivery, weight of baby, Live birth/stillbirth,

complications after delivery (PPH, Retained Placenta, Infection)

- Past history: Hypertension, Diabetes Mellitus, Tuberculosis, Asthma, Heart disease, any other surgical procedures undergone, medications taken during peri conceptional period, history of bleeding.
- H/O current symptoms, perception of fetal movements if pregnancy > 16 weeks.

3. Schedule of Examination

Ideally all the ANCs should be examined monthly after registration. If it is not possible to attend ANC clinic monthly, then checkup should be carried out at least five times during the pregnancy. First 8-12 weeks, second between 14-20 weeks, third at 22-26 weeks, fourth at 28-32 weeks and fifth at 36-40 weeks.

*Height	*Breast examination
Weight: Compare with previous visit (calculate BMI in first trimester)	*Systemic examination: Auscultate chest
Pallor, icterus	P/A: Fundal height & its correlation with period of amenorrhea (POA)
Edema over feet, hands, face	Fetal presentation and position after 32 weeks
Blood pressure	Fetal heart rate

Table 1: Examination at Antenatal Clinic

* During first check up

- The last two visits are important as many of the pregnancy complications are detected during last trimester. For 'high risk' mothers more frequent examinations will be required.
- Medical officer should perform at least one checkup during the third trimester and auscultate her chest to rule out any systemic abnormality.

4. Investigations

Table 2

Hemoglobin estimation	VDRL
6	
Urine Analysis: Protein and sugar	Blood sugar testing
Test for sickling in selected tribal area	Malarial parasite testing in endemic area.
Blood grouping. Rh typing	Hepatitis B surface antigen (HBsAg)
Voluntary HIV testing	Ultrasonography (around 18 weeks)

5. Examination and action to be taken during ANC check up

Table 3

Examination	Action		
Edema	• Examine whether edema is on one leg or both legs and is it pitting. Look for edema over face, hands, and abdomen. Check for proteinuria and hypertension.		
	• High blood pressure and albuminuria present, refer to specialist as she has pre- eclampsia.		
	• History of kidney disease, if yes refer to specialist.		
	• If edema is on one leg, refer to specialist.		
	• If bilateral pedal edema without albuminuria and normal BP: Reassure mother, check for anemia and give IFA tablets as required		
Weight gain	• Record monthly weight on MCP card of mother, calculate weight gain since the previous visit		
	• Weight gain more than 3 kg. in a month: Suspect preeclampsia		
	• Weight gain less than 1 kg. in a month: Suspect fetal growth retardation.		
Blood Pressure	• If 140 / 90 mm Hg or more, advise mother to rest for half hour and then repeat the BP recording. Check for proteinuria		
	• If systolic between 140-160 and /or diastolic 90 or above: advise extra rest and refer to MO PHC		
	• If systolic 160 or more or diastolic > 100: refer to specialist		
Fundal Height	Examine fundal height in weeks and compare with calculated duration of pregnancy as per LMP. If it is greater or lesser refer to specialist. Causes of fundal height less or more than expected are given in table below.		
Fetal Presentation	Non-cephalic at 34-36 weeks. Refer to Obs/Gyn specialist (correction can be attempted at 36 weeks in suitable cases).		
Fetal Heart Rate	FHR < 120 or > 160 /minute: Refer to specialist.		
Hemoglobin %	Hb 11 gm% or more: IFA 1 tablet daily for 180 days		
	Hb between 7-11 gram%: Start IFA double dose* and re-examine after one month - If improvement of Hb by more than 1gm%, continue IFA. Give tab Albendazole		

Examination	Action
	(during second trimester)
	Hb< 7 gm % - Refer anemia treatment guidelines
Proteinuria	If proteinuria present suspect pre-eclampsia and refer to specialist.
Risk factors	All high-risk pregnancies should be checked by MO and then referred to specialist if necessary for further checkup or during delivery depending upon the risk factor.

* For better absorption, IFA tablets should be taken 1.30 hours before meals

Table	4
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Fundal height < Period of Amenorrhea	Fundal height > Period of Amenorrhea
Wrong dates	Wrong dates
Infrequent periods prior to conception	Polyhydramnios
Intrauterine Growth retardation(IUGR)	Twins
Oligohydramnios	Big baby
Intrauterine fetal death.	Hydatidiform mole
	Uterine fibroids

6. Education and counseling regarding care during pregnancy

6.1. First and second trimester:

- Diet: Two meals, breakfast and evening snacks. Rich in proteins, iron, calcium, vitamins, inclusion of sprouted legumes, pulses, green leafy and other vegetables, seasonal fruits, milkmilk products. Consumption of Iodized salt
- Consumption of Folic acid tablets in first 12 weeks, iron folic acid tablets after 12 weeks for 180 days for anemia prophylaxis.
- Tetanus Toxoid 2 doses/booster dose.
- Consumption of Calcium carbonate (500mg) + Vitamin D tablets 1 tab twice a day.
- Rest: 2 hours in afternoon and 8 hours at night in lateral position.
- Exercise: Walking for 30 minutes daily.
- Habits: Avoid tobacco in any form, avoid alcohol.

- Practicing safe sex.
- Self-reporting of danger signals, e.g. Abdominal pain, severe headache, giddiness, palpitations, easy fatigability, breathlessness, fever, generalized edema, vaginal bleeding, watery discharge per vaginum, blurred vision, excessive vomiting, reduced fetal movements.

6.2. Third trimester:

- Avoid heavy work and jerky travel on bad roads.
- Importance of institutional delivery, safe delivery, inform Toll Free No.102 and 108 for free ambulance service, JSY, JSSK and other benefits, Plan for place of delivery, preparation for delivery.
- Importance of early initiation of colostrum feeding within half an hour of birth & exclusive breast-feeding for 6 months, child immunization and contraception especially PPIUCD.
- Identify birth companion.

7. Identify high-risk mothers:

Risk factors detectable during	Abnormalities developing during	
first check up	Current pregnancy	
 Age: Teenage/ elderly primi Para 4 and above Short stature, limping gait, vertebral spine abnormalities Bad obstetric history: H/O stillbirth, neonatal death, LBW baby, recurrent abortions Previous Caesarean delivery H/O Preeclampsia/eclampsia, PPH, retained placenta during previous pregnancies Preexisting medical conditions: Heart disease, Diabetes Mellitus, renal disease HIV /VDRL positive gravida Rh negative gravida 	 Anemia Hypertension, proteinuria Vaginal bleeding during pregnancy Premature rupture of membranes (PROM) Gestational Diabetes Mellitus (GDM) Fundal height < POA or > POA Uterine over distension: Twins, Polyhydramnios Fetal malpresentation persisting near term Pregnancy > 41 weeks Reduced fetal movements 	

Table 5:

7.1. Actions suggested for some high-risk indicators

RISKS	ACTION
1. Elderly Primi	
 Hypertension during pregnancy Gestational Diabetes Difficult labour - Chances of caesarean section are higher Fetal abnormalities. 	 Refer to specialist soon after registration for evaluations to exclude fetal anomalies (biochemical markers and ultrasonography) Regular ANC: B.P, urine analysis every month Pelvic assessment at or after 36 weeks. Institutional delivery under care of specialist.
2. Teenage primi	
 Hypertension during pregnancy Anemia Pre-term labour Fetal growth retardation. Difficult labour 	 Regular antenatal care Hb%, BP, urine analysis more frequently Adequate rest IFA tablets, nutrition guidance Pelvic assessment at 36 weeks Hospital delivery.

Table 6:

RISKS	ACTION
3. Primi: Height less than 145 cms	Regular ANC checkup.
Difficult labour	Assessment of place of delivery by specialist. Observe progress of labour partographically.
4.Primi having vertebral /limb deformity	Regular checkup at PHC,
Difficult labour	Assessment by specialist for place of delivery.
5. Grand multipara (para 4 and more)	
Anemia	• Supplement IFA, nutrition guidance
Malpresentation	• At 34 and 36 weeks look for fetal malpresentation & refer
Atonic PPH	• Hospital delivery - Active management of 3rd stage of
Uterine rupture	labour - Keep IV line ready
	Avoid injudicious use of oxytocics for augmenting labour

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Figure 1.1: Fundal Height Measurement and Obstetric Examination

2. NORMAL LABOUR

Every delivery should be conducted by a skilled birth attendant. At a PHC, cases admitted for delivery need to be assessed for detection of any abnormality and complications. It is necessary to give initial care to the complicated cases and execute appropriate referrals.

When a woman is admitted for delivery, review ANC card (Lab test results, weight gain, risk factors, complication if any) and perform complete examination.

1.General examination: Pulse, temperature, blood pressure, pallor, icterus, edema

2. Obstetric examination per abdomen:

Uterine contractions: Frequency(No/10minutes), duration in seconds and intensity

Fundal height: Proportionate to POA or greater or lesser than POA

Presentation: Cephalic or non-cephalic

Engagement of head: 2/5th or less fetal head palpable per abdomen

Fetal heart rate: Normal FHR 120-160/min, regular.

3.Vaginal Examination:

Take adequate aseptic precautions and note:

Cervical dilatation in cm and effacement as percentage

Membranes intact or ruptured. If ruptured, since how many hours?

Color of liquor: Clear, meconium stained or blood stained.

Presenting part: Vertex or other than vertex.

Position: Occiput anterior or posterior

Head is well flexed (posterior fontanel felt easily) or deflexed (anterior fontanel also felt easily)

Station of presenting part in relation to ischial spines

Presence of caput or molding

Assessment of pelvis and test for CPD

3.1. Pelvic Assessment

Tab	le	1

Parameter	Adequate	Suggestive of abnormality
Sacral promontory figure 1	Not felt	Felt easily
Diagonal Conjugate*	>11.5 cms	< 11.5 cms
Sacral curvature	Well curved	Flat
Lateral Pelvic walls	Parallel	Converging
Ischial spines	Both cannot be palpated simultaneously	Both can be palpated simultaneously
Subpubic Angle	Accommodates two fingers (85 ⁰)	Acute
Inter tuberous diameter	Accommodates closed fist (4 Knuckles)	Cannot accommodate 4 knuckles

* If sacral promontory is felt, distance between sacral promontory and lower border of pubic symphysis is measured.



Fig 2.1. Palpating Sacral Promontory & Measuring Diagonal Conjugate



Figure 2.2. Assessing transverse diameter of Outlet



Figure 2.3. Assessing Subpubic Angle

3.2. Clinical Examination for CPD

- Place the woman in dorsal position.
- Hold the fetal head by left hand.
- Place two fingers of gloved right hand into the vagina at the level of ischial spines.
- Place the thumb of right hand on the pubic symphysis.
- Push the head into the pelvic inlet and note whether it descends into the pelvis (felt by fingers in vagina) or overhangs on the pubic symphysis. If head descends with no overlap at pubic symphysis: No inlet CPD.
- If the head is engaged, it indicates that the pelvic inlet is adequate.

4. Monitoring during active phase

Table 2

Examination / Observation	Periodicity	
Maternal pulse and FHR	Every 30 minutes	
Uterine contractions	Every 30 minutes	
Maternal temperature, BP, urine volume	Every four hours	
Vaginal Examination	Every four hours	

Note the time of rupture of membranes and perform vaginal examination immediately

- To rule out cord prolapse and see the color of liquor.
- To assess cervical dilatation, station and position of presenting part.

Assess and monitor the progress of labour by using 'Simplified Partograph' adopted by GOI to record FHR, liquor, cervical dilatation, uterine contractions, medications administered during labour, maternal pulse, blood pressure and temperature.

Points to Remember

- This graph is used only during active phase of labour.
- The complications requiring immediate intervention need to be excluded first.

4.1. Partograph

On this graph, one square represents one hour on horizontal axis. On vertical axis, one square represents cervical dilation of one centimeter.

4.1.1 Phases of first stage and alert and action line:

- Latent phase: Cervix dilates very slowly up to 4 centimeters. It may take up to eight hours for this.
- Active phase: 4 cm onwards; cervix dilates rapidly at the rate of about 1 centimeter per hour.
- Alert Line: An oblique line on the graph from 4 to 10 centimeters progressing at the rate of 1 centimeter per hour.
- Action Line: Parallel line four hours to the right of alert line

4.1.2 Recording on partograph:

- Initiate recording when cervix is 4 cm dilated and immediately if the cervix is > 4 cm dilated on admission to the labour ward.
- Record the dilatation in cm (symbol x) on the alert line and record the time at which observation has been made on the horizontal axis below this point.
- Perform vaginal examination every 4 hours and note the cervical dilatation and mark it on the graph.
- Join the points and interpret the observations.

4.1.3 Interpretation and Action:

- Satisfactory progress: Cervical dilatation lies on left side of alert line, active phase progression > 1 cm/ hour, Fetal and maternal health parameters are normal.
- Slow progress: Alert line crossed, careful monitoring to note the progress. At sub centre and PHC the arrangements for referral to FRU.
- Action line reached or crossed: Careful review to find the cause for delay followed by appropriate interventions.

5. Management of Labour

5.1 First stage of labour showing satisfactory progress

Continue observation till second stage starts.

5.2 Second stage of Labour

- Cervix is fully dilated.
- Signs of imminent delivery: Mother starts pushing (bearing down). Fetal scalp seen at vulva, perineum bulges, anus gapes. Mother gets sensation of defecation.
- Encourage the mother to push only during contraction.
- Monitor progress and fetal wellbeing during second stage.
- Note FHR every 15 minutes.
- Assess descent of presenting part (fetal station) every 15 min.
- Look for caput, moulding, meconium staining of liquor.
- Support the perineum and deliver the head gently
- Deliver the shoulders
- Keep the baby on mother's abdomen on a prewarmed towel and give immediate care.
- Follow universal bio safety precautions.
- Delivery of baby takes place usually within 30 minutes in a multi and 60 minutes in a primi.
- Give mediolateral episiotomy under local infiltration anesthesia by injecting 1% lignocaine, if mother is unable to push or if there is undue delay.

5.3 Third stage of Labour

Deliver the placenta by active management of third stage of labour (AMTSL).

Active management of 3^{rd} stage of labour reduces the amount of blood loss due to uterine atony, reduces the chances of having atonic PPH and requirement of blood transfusion significantly thereby helping to reduce maternal mortality due to severe PPH.

It is an integral part of skilled attendance at birth and is mandatory for all deliveries. The steps are as follows:

- After the childbirth, exclude presence of another baby by abdominal palpation
- Give Inj. Oxytocin 10 IU IM. immediately after birth (Oxytocin should be kept in refrigerator). When Oxytocin is unavailable, tablet

Misoprostol 600 mcg (3 tablets) may be given orally.

- Check for uterine contraction.
- Clamp and cut the cord (1-3 min after birth)
- Deliver the placenta by controlled cord traction (CCT). When the uterus is well contracted give gentle downward traction on cord while giving counter traction by other hand pushing the

uterus upwards towards umbilicus. Repeat this during contraction as required. Only trained SBA should give CCT figure 4.

• After delivery of placenta, give uterine massage to keep the uterus contracted.



Figure 2.4: Controlled Cord Traction

6. Care after delivery

- Examine the perineum, vulva, lower vagina for tears.
- Examine the placenta and membranes carefully for completeness and any abnormality.
- Observe the mother every 15 minutes for two hours for general condition, pulse, vaginal bleeding, pallor, uterine contraction.
- Repeat uterine massage every 15 minutes for 2 hours.
- Encourage mother to take the baby to breast within ½ hour of delivery.

7. Augmentation of Labour

7.1. Slow progress of Labour

- Alert line crossed or the action line reached
- Causes: Hypotonic uterine action, occipitoposterior position, deflexed head, CPD etc. Refer the case to higher level of care.

Labour augmentation is done only when there is unsatisfactory progress of labour. Amniotomy (artificial rupture of membranes) and Oxytocin infusion are offered as per the clinical situation. Labour augmentation should be done in places where clinical expertise and facilities for operative delivery are available.

- If vertex is engaged and cervix is well applied to the vertex, then controlled ARM can be performed to hasten the progress.
- After ARM review progress after two hours. If progress is still slow, assess uterine contractions. If uterine contractions are infrequent and weak then consider Oxytocin augmentation after excluding contraindications.
- If there is no progress after augmentation or if signs of fetal distress develop, an emergency Caesarean Section might be required. Hence such a woman should be referred to a well-equipped maternity unit.

7.2. Oxytocin Augmentation: (only where specialist is available)

Exclude contraindications before starting Oxytocin

- CPD, Fetal distress
- Grand Multiparity, Malpresentation
- Scar on the uterus

7.2.1 Dose and Administration

- Add 2.5 IU Oxytocin to 500 ml of normal saline or ringer's lactate. Start infusion at the rate of 10 drops/min. Observe uterine contractions.
- If contractions are mild increase the drip rate by 10 drops /min every 30 min until she gets 3 contractions in 10 minutes, each contraction lasts for 40-45 seconds and there is good relaxation of uterus in between the contractions (optimum response).

Drip rate can be increased maximum up to 60 drops/minutes. If the contractions are inadequate at this rate, prepare another drip with 5 IU /500 ml and start it at the rate of 30 drops/min, increase by 8-10 drops every 30 minutes and observe the response.

7.2.2 Monitoring:

Monitor following parameters every 15 minutes,

- Drip rate, Uterine contractions frequency and intensity
- FHR and Maternal pulse
- Progress of labour

7.2.3 Complications:

- Hyperstimulation: > 5 uterine contractions / 10 minutes, each lasting for > 60 sec
- Uterus failing to relax between the contractions
- Fetal bradycardia (distress)
- Uterine rupture if drip is not supervised vigilantly.
- Water intoxication if too much of electrolyte free infusion is administered.
- Neonatal hyperbilirubinemia

7.2.4 Treatment of Hyperstimulation:

- Discontinue the drip
- Maternal repositioning (left lateral position), Oxygen therapy
- Terbutaline 250 mcg can be given IV slowly over 5 minutes for tocolysis if required

Caution:

- For augmentation of labour Oxytocin should never be given intramuscularly.
- Cautious use in multiparous women as the uterus tends to rupture.
- Misoprostol should **not** be used for labour augmentation.

7.2.5 When to discontinue the drip:

- If six hours of strong stimulated uterine activity is unable to bring progress of labour, discontinue the drip and review the case.
- If there are signs suggestive of fetal distress.
- If after initial satisfactory progress, cervical dilatation does not progress for two hours or more in established active phase of labour then review her. This could be due to cephalo-pelvic disproportion or large head with deflexion. Such patient may need operative intervention.

8 Prolonged second stage

If second stage is prolonged for more than 2 hours in primi and for > 1 hour in multi, then

- Assess size of baby and pelvis again.
- See whether fetal head is still palpable in suprapubic region.

Refer the case to FRU/DH if:

- Failure of fetal head to descend (station fails to advance)
- Increasing caput or molding

- Suspected CPD including mid-pelvic and outlet contraction
- Occipito-posterior/Occipito-transverse position with arrest at mid-pelvis
- Maternal exhaustion
- Fetal distress

Cases of prolonged second stage may require assisted instrumental delivery or Caesarean Section.

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3. CLINICAL CARE OF HIGH RISK PREGNANCY

Common Risk Indicators:

- Bad obstetric history (BOH): Recurrent pregnancy loss (RPL)
- Cervical incompetence
- Hypothyroidism
- Infections: Syphilis
- Rh negative pregnant woman, husband Rh +ve
- Post caesarean pregnancy.
- Diagnosed twins, polyhydramnios.
- Pregnancy beyond 41 weeks.
- Hypertension, Preeclampsia
- Vaginal bleeding during pregnancy, APH.
- Fetal malpresentation persisting at 36 weeks.
- Medical diseases: Anemia, Diabetes Mellitus, Cardiac disease, Chronic Hypertension, Kidney disease, Tuberculosis. (Subsequent chapters)
- Other high risk indicators (refer chapter 1).

1. Bad Obstetric History

Bad obstetric history is a term used in day to day practice for describing unsuccessful pregnancy outcome in previous pregnancies. A variety of unsuccessful outcomes are included in this such as previous H/O stillbirth, early neonatal death, preterm delivery, recurrent mid-trimester/early abortions.

1.1. Recurrent Pregnancy Loss

1.1.1 Definition: Three or more consecutive pregnancy losses

1.1.2 Causes:

- Genetic
- Environmental and occupational exposure to organic solvents, ionizing radiation, toxins, tobacco, alcohol
- Uterine anomalies, Cervical incompetence, Intrauterine adhesions, Uterine fibroids
- Infections
- Endocrine dysfunction: Polycystic ovary syndrome (PCOS), Luteal phase inadequacy (LPI), Diabetes Mellitus, Thyroid dysfunction, Prolactin disorders.
- Antiphospholipid antibody syndrome (APLAS)
- Unexplained: In some cases, no cause can be detected.

1.1.3 Risk Factors:

- Risk is highest among couples where the age of woman is ≥35 years.
- The risk increases after each successive pregnancy loss, reaching approximately 40% after three consecutive pregnancy losses.

1.1.4 History:

- Detailed obstetric history about each pregnancy. Duration of pregnancy at mishap. Whether USG documented cardiac activity was seen, fetus was live born/stillborn (fresh/macerated stillbirth), baby normal/abnormal.
- H/O Consanguinity, history of infertility, menstrual abnormality, infections, STD.
- Personal history: Tobacco, alcohol, caffeine, medications.
- Personal/familial H/O thrombosis, autoimmune disorder.

1.1.5 Physical examination:

- a) Look for obesity, hirsutism, acanthosis, thyroid enlargement, galactorrhea
- b) P/S & P/V examination: Look for uterine fibroids, double uterus, bicornuate uterus, genital infection, torn or short cervix
- c) Investigations: Carry out the relevant investigations as indicated and available depending on whether the woman has presented during pregnancy or after the mishap.
- d) During pregnancy:
 - Hematology, blood group and Rh typing, VDRL
 - Urine analysis
 - Rule out Diabetes
 - Antiphospholipid antibody testing
 - USG: For uterine abnormalities, Monitoring early pregnancy
 - Thyroid function, Prolactin

e) Non pregnant state:

- Pelvic USG for polycystic ovaries (PCOS),
- Hysterosalpingography (HSG), Hysteroscopy for uterine abnormalities
- Parental karyotype
- Mid luteal serum progesterone and thrombophilia profile if available
- f) Soon after miscarriage: Chromosomal studies of conceptus

1.1.6 General Measures:

- Quitting cigarette smoking, tobacco, alcohol and caffeine consumption
- Weight reduction in PCOS cases
- Therapies depending upon cause.
- Psychological counseling & support is extremely helpful.
- 50% Success even if no treatment is given.

2. Cervical incompetence

- Premature softening and dilatation of internal cervical os during pregnancy can lead to protrusion of membranes, rupture of membranes and fetal expulsion during second trimester or early third trimester
- Typical obstetric history is diagnostic: Repeated mid-trimester or early third trimester pregnancy terminations, relatively painless, rapid, often preceded by rupture of membranes followed by expulsion of fresh still birth or live fetus
- Symptoms: Low backache, mucoid vaginal discharge may precede
- P/S: Bag of membranes may be seen through the cervical os
- Vaginal examination: Internal cervical os dilated in absence of uterine contractions, cervix may be short.

2.1 Investigation:

USG may show short cervix, funneling of cervix, membranes dipping into the cervical canal.

USG assessment of cervical length (25mm or less) by transvaginal scan before 24 weeks in women having previous history of preterm labour is considered significant for prediction of preterm labour.

2.2Treatment:

At PHC: Refer the case to specialist soon after registration.

At RH/DH: Prophylactic cervical os tightening at 16 weeks by McDonald method under short general anesthesia.

- Follow up: Extra rest, avoid exertion.
- Stitch removal at 37 weeks or at the onset of labour whichever is earlier. Anticipate rapid delivery.

3. Infections

Generally, cause sporadic pregnancy loss. Toxoplasmosis, Rubella, Cytomegalovirus, Herpes and Listeria infections do not cause recurrent pregnancy loss and routine TORCH screening is not indicated. Active syphilis during pregnancy can lead to recurrent pregnancy loss.

3.1. Syphilis during pregnancy

- **3.1.1 Effects:** Late abortions, stillbirths, neonatal deaths, infant having active congenital syphilis.
- **3.1.2 Diagnosis is serological:** VDRL at booking. Treponemal test (TPHA) is confirmatory, however not mandatory.

3.1.3 Treatment:

Treatment of maternal disease can prevent fetal infection and also treat established fetal disease

- Benzathine penicillin single injection 2.4 Mega units deep IM **after sensitivity test** as Penicillin is the only antibiotic that can cross the placenta in adequate amounts to treat the fetus. Check emergency drug tray.
- For latent disease > 1-year duration, 3 Injections at weekly interval.
- Penicillin sensitive individuals require desensitization.
- Erythromycin 2 gms daily in divided doses for 14 days can be considered however Penicillin is recommended during pregnancy.
- Infant of a VDRL positive mother needs blood testing, penicillin treatment and care by specialist.
- Partner testing, Safe sex counseling, voluntary HIV testing



Fig 3.1. Congenital Syphilis: Blistering Skin rash

4. Hypothyroidism

- Hypothyroidism can be due to iodine deficiency or thyroid autoantibodies.
- Infertility, menstrual problems due to ovulation disturbances.

- Increased risk of miscarriage due to luteal phase insufficiency.
- Anemia, gestational hypertension, placental abruption and postpartum hemorrhage are common.
- Fetal/neonatal problems: Preterm birth, low birth weight, congenital malformations, neonatal respiratory distress, perinatal death. Cretinism.
- Diagnosis by testing serum TSH. If elevated, thyroid function tests and antibody testing.
- Treatment by Thyroxine can correct ovulatory dysfunction and help achieving pregnancy and successful outcome.
- Counsel pregnant women for consumption of iodized salt.
- Targeted case finding approach is followed. Universal TSH screening is currently not recommended.

5. Rh Negative Pregnant Woman

A major cause of hemolytic disease of newborn (HDN) is Rh blood group incompatibility between the mother and her fetus. When Rh negative mother bears Rh positive fetus, the Rh antigen from fetal red cells enters the maternal circulation and results in antibody formation in mother. These antibodies cross the placenta and cause destruction of fetal red cells resulting in fetal anemia. Prevention of such sensitization is possible by antenatal screening and appropriate care.

5.1. Pregnancy outcome

First child usually escapes. Subsequent babies suffer from mild hemolytic anemia, rapidly increasing hyperbilirubinemia within 24 hours of birth or hydrops fetalis resulting in intrauterine fetal death. The adverse perinatal outcome occurs earlier during pregnancy with each subsequent pregnancy.

5.2. Management

- Every pregnant woman should be tested for Rh typing at registration.
- Those testing negative should have their husband's Rh typing done.
- Take detailed history about every pregnancy and its outcome. H/O neonatal jaundice, hydrops, stillbirths etc.

- Note history of receiving anti D immunoglobulin, time, dose during/following every pregnancy.
- Perform Indirect Coombs test (ICT) on blood sample to detect maternal sensitization.

5.2.1 Nonsensitized woman:

- Negative ICT indicates that the woman does not have detectable antibodies against Rh antigens. Refer her to gynecologist at FRU. ICT is repeated at 28 weeks².
- Do not perform external cephalic version for malpresentation. Any episode of bleeding or manipulation, procedure such as amniocentesis should be followed by administration of anti D immunoglobulin.
- It is recommended to give 300 mcg anti D immunoglobulin during pregnancy at 28 weeks for reducing the risk of antepartum sensitization.
- At delivery, cord blood sample to be tested for: Infant's Rh, ABO grouping, hemoglobin, hematocrit. Direct Coombs test and direct and indirect bilirubin estimation.
- Administration of 300 mcg of anti D Ig IM to mother within 72 hours of delivery. 300 mcg neutralizes 15 ml of feto-placental hemorrhage (FMH). If it has not been given within 72 hours, can be given up to 28 days with some benefit.
- The baby is referred to neonatology specialist and is observed for icterus, anemia.

5.2.2 Sensitized woman:

- If ICT is positive, it indicates that the woman is sensitized and has got antibodies against Rh antigen in her blood. Note the titers reported. There is no use of giving Anti D Ig to sensitized mother.
- Sensitized woman must be referred to a tertiary care centre having equipment and expertise for Doppler middle cerebral artery peak systolic flow velocity studies, Cordocentesis and intravascular fetal transfusion as required.

6. Post Caesarean Pregnancy

Pregnancy with h/o previous Caesarean Section (CS) is a high risk condition as there is increased risk of complications during childbirth after each Caesarean Section (Scar rupture, placenta praevia, adherent placenta, PPH)

6.1 Risks

Maternal	Fetal/neonatal
Scar dehiscence during labour	Fetal distress
Scar rupture leading to severe haemorrhage & shock	Fetal death due to scar rupture
Chances of repeat CS are high	Neonatal asphyxia

Table 1: Risks to Pregnant Woman and Baby

All cases having Cesarean Section in previous delivery should be referred to specialist at FRU/district hospital during third trimester. At 36 weeks she should have a review by specialist regarding plan for delivery either by repeat elective Cesarean Section or trial for vaginal birth after Cesarean Section which depends on indication of prior Cesarean Section and assessment of scar integrity.

6.2 History

- Indication for Cesarean Section, elective or emergency, when in labour it was done.
- Place and person operating, performed at term or preterm.
- Type of Cesarean Section: Lower segment transverse incision or classical. (Vertical scar is very weak having high chances of scar rupture which is likely to happen during late pregnancy and during labour with increased risk to the mother and fetus hence elective caesarean section should be performed at term in such cases)
- Review operation notes regarding complications, inverted T extension of incision, or lateral extension of tears, post-operative infection, blood transfusion, prolonged hospital stays.
- Inter pregnancy interval < 24 months increases the risk of scar rupture.

6.3 Examination

- Anemia: Correction before term is important as the chances of requiring repeat Cesarean Section are high.
- Note the size of baby, presentation, presence of any other risk factor or complication in current pregnancy.

- Exclude pregnancy complication: Twins, polyhydramnios, APH.
- Look for scar tenderness.
- Perform clinical pelvic assessment at or after 36 weeks.

6.4 Investigations

USG assessment of fetal weight, localization of placenta. Look for morbidly adherent placenta if placenta praevia.

6.5 Selection for Trial of Labour after Caesarean Section

- Indication for previous Cesarean Section non recurrent (eg fetal distress, breech, placenta praevia.)
- Only one prior low transverse Cesarean Section delivery. No other uterine scars or previous rupture.
- Vertex presentation, clinically adequate pelvis.
- No other obstetric complication (eg APH, breech presentation)
- Surgeon, Anesthetist immediately available for emergency Cesarean Section.

All post caesarean pregnancies should only be managed in equipped hospitals with personnel readily available for emergency caesarean section when needed. Carefully selected women can be allowed to have trial of vaginal birth under expert supervision.

Repeat Cesarean Section: Women not eligible for vaginal trial will need an elective repeat Cesarean Section which should be performed at 39 weeks for the best neonatal outcome, if there is no maternal or fetal indication to perform it earlier.

6.5.1 Management of labour following Caesarean Section:

- Refer post Caesarean delivery to higher centers.
- Counseling and informed consent for trial of labour.
- Keep operation theatre ready for CS, arrange blood.
- Monitor progress of labour.
- Watch for early signs of fetal distress, scar dehiscence and perform immediate caesarean section if these signs appear.
- If satisfactory progress, cut short the second stage of labour and deliver.
- After vaginal delivery, watch for PPH/intra peritoneal hemorrhage.

6.5.2 Symptoms & Signs of impending rupture during labour:

- Suprapubic pain persisting in between uterine contractions.
- Slight fresh vaginal bleeding.
- Unexplained tachycardia, tenderness over uterine scar.
- Alteration in fetal heart rate, sudden signs of fetal distress.
- Hematuria.
- Falling BP with increasing pallor is a late sign of uterine rupture.

6.5.3 Contraception:

• Counseling for contraception for increasing inter pregnancy interval.

• Postpartum IUCD insertion at operation before closure of uterine incision is safe.

7. Twin Pregnancy

7.1 Antenatal Diagnosis and Management

The incidence of twin gestation is increasing as a result of infertility treatment. With modern assisted reproduction techniques higher order of multifetal gestation is common.

7.1.1 History:

- Ovulation induction by Clomiphene or Gonadotropin Injections for infertility.
- Maternal family history of twins, advanced age, high parity.

7.1.2 Signs:

- Suspect twins when the fundal height is greater than the period of amenorrhea and multiple fetal parts are felt.
- Palpation of 2 heads, 3 major poles
- May have associated polyhydramnios
- Two persons simultaneously hearing FHS at two different locations with a difference of > 10 beats/min.

7.1.3 Investigation:

Ultrasonography. Early sonography in second trimester helps in assessing chorionicity

7.2Risks

Pregnant Woman	Baby
Hyperemesis gravidarum	Prematurity
Anemia	IUGR
Pre-eclampsia	Fetal Malpresentation
Antepartum haemorrhage due to Placenta praevia and also abruptio placentae is likely	Fetal asphyxia
Preterm labour	Twin-to-twin transfusion
Hypotonic uterine action, prolonged labour	Fetal malformations
Postpartum haemorrhage	Fetal death
Difficult delivery	Perinatal mortality is high

Table 2: Risk to Pregnant Woman and Baby

7.3 Management during pregnancy

- Detection and correction of anemia.
- Nutrition counseling, extra nutrients to meet the need of two fetuses.
- Avoiding exertion, extra rest in left lateral position.
- Early diagnosis of preeclampsia.
- Refer to specialist for antenatal care and delivery at FRU.
- Explaining warning signals for preterm Labour.

8. Polyhydramnios

8.1. Definition

Excessive volume of amniotic fluid

8.2. Causes

- Fetal anomalies: Anencephaly, esophageal atresia.
- ٠ Maternal Diabetes, Multiple pregnancy.
- Idiopathic, Placental chorioangioma.

Can be mild, moderate or severe.

Development can be acute or chronic

8.3 Risks

Table 5. Risks to Freghant Woman and Daby			
Pregnant Woman	Baby		
Hypertension	Prematurity		
Risks due to associated Diabetes Mellitus	Malpresentation		
Risks due to associated twins	Fetal asphyxia due to cord prolapse or placental abruption		
APH: Placental abruption	Fetal malformations		
Preterm labour, PROM, cord prolapse Hypotonic uterine action, prolonged labour	Problems due to associated complications: twins/preeclampsia/APH		
Postpartum haemorrhage			
Difficult delivery			

Table 3. Risks to Pregnant Woman and Baby

8.4. Diagnosis

- Fundal height is > POA. Abdomen is over • distended.
- Fluid thrill can be demonstrated.
- Fetal parts are not well felt if the abdomen is tense. FHS may not be clearly heard.
- USG confirms the diagnosis by demonstrating • large amount of amniotic fluid. Amniotic fluid index (AFI) 24-25 cm
- Association with twins and fetal malformations need to be looked for.
- Severe hydramnios can lead to edema, severe breathlessness, inability to sleep and may require immediate attention by specialist.
- Maternal Diabetes Mellitus needs to be excluded.
- Hydramnios should be differentiated from ascites and large ovarian cyst.

8.5 Management

Mild cases: No intervention required. Extra rest in comfortable position.

Symptomatic women need to be hospitalized.

Specialist can offer following interventions depending upon the case.

- Tablet Indomethacin: 50-100 mg stat followed by maximum 200 mg in 24 hrs. for 1-2 days at <30-32 weeks of pregnancy (As there is risk of premature closure of fetal ductus arteriosus)
- Amniocentesis to relieve the maternal distress. The success is transient and repeated fluid removal is required. Complications such as preterm labour, rupture of membranes, chorioamnionitis, and placental abruption are likely.
- Delivery in a well-equipped institution under care of a specialist.
- Oxytocin augmentation if contractions are weak.

- Risk of placental abruption is high when the membranes rupture and a large volume of amniotic fluid is drained suddenly. Preserving membranes is important. When required, controlled ARM is done.
- Active management of third stage as there is a risk of atonic PPH.
- Examine the baby for congenital malformations (Esophageal atresia or trachea- esophageal fistula)

9. Prolonged Pregnancy

9.1. Definitions

Post term pregnancy: Pregnancy that has crossed 42 weeks.

9.2. Risks

- **Post maturity syndrome**: In about 20 % cases of prolonged pregnancy there is placental insufficiency which results in a pathologic syndrome in which there is fetal growth retardation associated with meconium stained amniotic fluid, oligohydramnios and fetal distress. The newborn is at risk of meconium aspiration syndrome. The baby shows loss of subcutaneous fat, wrinkled, dry, cracked skin (old man look). It has long thin body and long nails. Associated oligohydramnios increases the likelihood of post-maturity.
- In other cases, not complicated by placental insufficiency, there is continued growth of the fetus leading to macrosomia, with increased risk of abnormal labour, shoulder dystocia, and Injuries to baby.
- Increased perinatal mortality in pregnancies continuing > 42 weeks.
- Mother: Difficulties in labour due to fetal macrosomia, Shoulder dystocia.
- Increased chance of caesarean delivery.

9.3. Risk Factors

Primiparity, pervious history of post term pregnancy, sedentary life style, anencephalic fetus. Genetic predisposition

9.4 Prevention

• Accurate calculation of EDD. Ask whether menstrual cycles before conception had been regular.

- Instruct ASHA, ANM to refer a woman who has crossed her EDD to the medical officer.
- At 40 weeks: Check for any complications. In pregnancies complicated by hypertension, preeclampsia, IUGR the fetus is at greater risk of asphyxia. Hence these women should not be allowed to cross their EDD. They should be referred to specialist as they need to be delivered early.
- Uncomplicated pregnancy: Wait until 41 weeks. Instruct the woman to report if fetal movements are reduced.
- Vaginal examination: Assess whether the cervix is ripe or unripe (Bishop Score).
- Sweeping of membranes should be done during this examination as it decreases the chances of post term pregnancy.
- At 41 weeks: At PHC, once pregnancy has reached 41 weeks, refer her to FRU for further evaluation and interventions.

9.5 Management of post term pregnancy at FRU

There are two options of managing 41 weeks pregnancy: Immediate induction of labour or expectant management until 42 weeks, while monitoring the fetal wellbeing.

9.5.1 In low-risk pregnancies routine induction of labour at 41 weeks is associated with reduction in the risk of fetal distress and in perinatal mortality. Hence induction of labour is recommended.

9.5.2 Follow up: If expectant management option is chosen, the mothers should be advised to come for fetal surveillance test at 41 weeks and twice between 41 and 42 weeks.

- a) History: Accurate assessment of gestational age to avoid delivery of a preterm baby. Errors likely if prior irregular menstrual cycles or if she has conceived soon after cessation of oral contraceptive pills. Review the early USG reports and the date of detection of positive UPT. Ask H/O of diminished fetal movements.
- b) On examination: Less amount of liquor on abdominal palpation, baby may be of large size.
- c) USG: Look for oligohydramnios: Amniotic fluid index (AFI) 5 cm or less or there is no vertical pocket > 2 cm. This is a marker for fetal compromise. Note estimated fetal weight and check for macrosomia.
- d) Non-stress test: Reactive or nonreactive.

[Reactive: FHR increased by > 15 beats lasting for 15 seconds in response to fetal movement]

Fetal compromise is indicated by reduced AFI, nonreactive NST, reduced fetal movements when immediate delivery is indicated.

e) At 42 weeks: Labour is induced. If the baby is too large or severely compromised, caesarean section is performed.

9.6 Induction of labour

- Cervix unfavorable (Bishop's score < 6): Vaginal Misoprostol 25 mcg 6 hourly for induction of labour. Oral Misoprostol 25 mcg 2 hourly can be used.
- Pre induction cervical ripening can also be achieved by intra cervical PGE2 gel or by Foley catheter. Cervix is reassessed for improvement

in bishop score. Induction can then be carried out with Oxytocin infusion.

• Favorable cervix: Induction can be done with Oxytocin infusion and ARM.

9.7 During labour (Induced or spontaneous)

- Partographic monitoring of labour. Monitor FHS and color of liquor, (electronic monitoring if available). Watch for signs of fetal distress.
- CS is indicated for intra partum fetal distress and for large baby.
- Second stage: Anticipate risk of shoulder dystocia.
- Episiotomy, assisted delivery may be performed as required.

Parameters	0	1	2	3
Cervical dilatation in cm	Closed	1-2	3-4	5+
Cervical effacement in %	0-30	40-50	60-70	80+
Fetal station	-3	-2	0, +1	+2, +3
Cervical consistency	Firm	Medium	Soft	
Cervical position	Posterior	Middle	Anterior	

Table 4: Bishop Score

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4. OBSTETRIC COMPLICATIONS

1. Preeclampsia and Eclampsia

Pre-eclampsia is a condition specific to pregnancy, arising after the 20th week of gestation, characterized by hypertension and proteinuria.

Eclampsia is preeclampsia with convulsions.

1.1. Signs of preeclampsia

• Hypertension: BP 140/90 mmHg or more on at least 2 occasions 4 hours apart after 20 weeks of gestation.

• Proteinuria: One plus or more.

Oedema of hands, face, abdominal wall (generalized edema) may be present but it is not a diagnostic feature. Excessive weight gain. 1 kg or more in a week or 3 kg in a month could be a warning signal.

1.2 Classification of pre-eclampsia

Mild and severe.

Finding	Mild Pre-eclampsia	Severe Pre-eclampsia
Blood Pressure (BP)	$BP \ge 140/90 \text{ but } < 160/110$	BP 160/110 mm Hg or
	mmHg	more
Proteinuria	Present, 2+ or less	*3+ or greater
Generalised Oedema (including in the face & hand)	May or may not be	Present
	present	
Headache	Absent	Present
Visual Disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Fetal growth restriction (IUGR)	Absent	Present
Pulmonary Oedema	Absent	Present
Decreased foetal Movement	Absent	Present
Platelet count	Normal	Less than 100,000

Table-1: Classification of Preeclampsia

It is not necessary that all these signs are present in all cases

1.3. Diagnosis

There are no symptoms in mild preeclampsia. Look for signs

At each prenatal visit, check the woman's BP, urine for presence of protein; look for oedema and record her weight. If there is a rise in BP, monitor the woman's BP weekly.

Table-2: Dangers

Maternal	Fetal/Neonatal
Eclampsia	IUGR
Cerebral oedema, haemorrhage, thrombosis,	Stillbirth
Acute renal failure	Neonatal asphyxia
Aspiration bronchopneumonia, Pulmonary oedema,	
HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count)	
Disseminated intravascular coagulopathy (DIC) leading to hemorrhage	

1.4 Management of mild pre-eclampsia

1.4.1 Gestation more than 37 weeks:

- Admit the woman to a hospital. Check BP. Test for proteinuria. Monitor FHR.
- Assess cervix. Induce labour if cervix is ripe. Do not allow her to cross her EDD.
- In mild cases, when maternal and fetal condition are well, can wait for spontaneous onset of labour until 37-38 weeks.

1.4.2 Gestation less than 37 weeks

Advise her to avoid exertion. Encourage her to rest in left lateral position. Counsel her about diet. She is allowed to take normal salt in food but no extra salt should be added to the food.

- Hospitalization.
- Check BP, urine protein, urine output, weight daily.
- Check for generalized body oedema.
- Exclude symptoms of severe pre-eclampsia
- Monitor foetal growth, ask the woman about foetal movements, check daily fetal movement count (DFMC), FHR.
- Biweekly LFT, KFT, Platelet count, NST
- Antihypertensive drugs are not beneficial in mild hypertension.
- Diuretics are not recommended.
- During observation if BP starts rising, (diastolic BP 100 mm Hg or more) start antihypertensive medicines.
- Tab Alpha Methyl Dopa 250-500 mg 6-8 hourly orally (maximum up to 2 gm in 24 hours) OR

- Tab Nifedipine sustained release preparation 10 mg orally twice a day (maximum up to 80 mg in 24 hrs.) OR
- Tab Labetalol 100 mg orally twice a day
- Terminate pregnancy if there are signs of fetal compromise, BP is persistently rising or if there is worsening proteinuria.
- Mode of delivery: Vaginal or caesarean section depending on gestational age, cervical status, maternal and fetal condition.

Anytime during observation if systolic BP is ≥ 160 mm Hg &/or diastolic BP ≥ 110 mm Hg: Manage as severe preeclampsia. She should be referred to a district hospital/medical college for further care.

1.5. Management of Severe preeclampsia

- Hospitalize patient.
- Control hypertension: Give anti-hypertensive drugs. Maintain diastolic blood pressure between 90-100 mmHg.

Nifedipine 10 mg orally. After 30 minutes, if BP is not brought under control, another 10 mg of the drug can be repeated. Caution needed while using Nifedipine as there may be sudden and massive fall in B P.

Labetalol can be given.

- Monitor vital signs, reflexes & fetal heart rate.
- Auscultate lung bases frequently and look for signs of pulmonary edema. If rales are heard give Inj. Frusemide 40 mg IV.
- Perform bedside clotting test.
- Watch for warning symptoms and signs which may appear before getting fits. Sharp rise of BP, increased proteinuria, exaggerated knee jerk. Severe headache, drowsiness, mental

confusion, visual disturbances (e.g. blurred vision, flashes of light, double vision) epigastric pain, nausea, vomiting, decreased urinary output.

- Prevent fits: Give prophylactic Magnesium Sulphate (full loading & maintenance dose as in Eclampsia)
- Encourage the woman for delivery at the FRU

1.6. Investigations

Urine- Albumin, sugar, culture, 24 hrs. urine protein, Hb%, PCV, Platelets, bleeding time, clotting time, serum fibrinogen, Blood urea, serum creatinine, serum uric acid, serum bilirubin, serum proteins, SGOT, SGPT. Fundoscopy.

1.7. Obstetric management

- Gestation < 24 weeks: Fetal salvage is difficult so proceed with termination of pregnancy.
- If gestation > 24 weeks < 34 weeks: Treatment should be individualized.
- Give Inj. Dexamethasone 6 mg 12 hourly 4 doses.
- If BP controlled keep woman under regular maternal & fetal surveillance.
- Deliver the woman at 37 wks. Induce labour before 37 weeks if BP uncontrolled or worsening of clinical/ biochemical parameters or appearance of signs of fetal compromise.
- Assess cervical status. If cervix is unripe induction by oral or vaginal Misoprostol. 25 mcg tablets. Intra cervical Dinoprostone gel can be used for pre-induction ripening of the cervix.
- If cervix is ripe induction can be done with Oxytocin infusion and Amniotomy.
- LSCS may be done for deteriorating maternal condition, adverse fetal condition, failed induction or other obstetric indications.

2. Eclampsia

Eclampsia is characterized by hypertension and proteinuria. (Preeclampsia) and convulsions/fits followed by coma.

Convulsions may occur in the antepartum, intra partum or the postpartum period.

Status eclampticus: Convulsions continue one after the other.

2.1. Differential diagnosis of convulsions during pregnancy

- Eclampsia: Hypertension, proteinuria.
- Epilepsy: Past H/O fits, normal BP.
- Cerebral malaria: Fever, Anemia, jaundice, Malaria tests positive.
- Meningitis/encephalitis, Tetanus.

2.2. Management of Eclampsia

Principles:

- i. General care
- ii. Control fits
- iii. Control blood pressure
- iv. Expedite delivery
- v. Maintain fluid balance
- vi. Postpartum care

2.2.1 General care

- Keep the patient in a quiet room in a bed with padded rails on sides. Place the woman on her left side.
- Evaluate vital signs.
- Clean the mouth & nostrils by applying gentle suction.
- Give oxygen.
- Prevent Injury to tongue by putting airway or by placing padded tongue blades.
- Start IV line with Ringer lactate/ normal saline: 60 ml/hour.
- Catheterize patient. Monitor hourly urine output.
- Send investigations as for severe pre-eclampsia
- If not breathing check airway and give bag and mask ventilation.

2.2.2 Controlling fits

Magnesium sulfate is the drug of choice.

- Loading Dose: Give Inj. Magnesium sulphate 4 gm (20 ml of 20% solution) slow IV in 5 min. (not to be given as a bolus rapidly). Then administer Inj. Magnesium sulphate 10 gm deep IM, 5 g in each gluteus muscle (10 ml of 50% solution, in each buttock) with 1 ml of 2% Lignocaine in the same syringe.
- If convulsions recur after 15 minutes, give additional 2 g of Magnesium sulphate (10 ml of 20 % solution) IV over 5 minutes.

- Maintenance Dose: Give 5 gm of 50% Magnesium sulphate solution IM with 1 ml of 2 % Lignocaine every 4 hours alternately in each buttock.
- Magnesium sulphate to be continued till 24 hours after delivery or the last convulsion whichever occurs later.
- Before giving the next dose of Magnesium sulphate, ensure:

- The Urine output is at least 100 ml in previous 4 hours.
- o Knee jerk reflexes are present
- The respiratory rate (RR) is at least 16 breaths/min.
- Postpone the next dose if the above criteria are not met.

Table-3: Magnesium sulphate solution for loading dose

Preparation of 20 % Magnesium sulphate solution for loading dose

- ▶ Inj. Magnesium sulphate is supplied as a 50 % solution in 2 ml vial. 1 amp of 2 ml = 1 gm MgSO4
- > 4 amp of 2 ml 50% solution = 4 gm Magnesium sulphate in 8 ml solution
- Add 12 ml distilled water or saline to make 20 ml 20 % Magnesium sulphate solution
- ➢ Give slowly IV in 5 min

For initial intramuscular dose:

▶ 5 amp of 2 ml 50% solution =5 gm MgSO4. Give 10 ml deep IM in each buttock

Table-4: Magnesium sulphate for maintenance dose

Preparation of 50 % Magnesium sulphate for maintenance dose

1 amp 2 ml 50% solution = 1gm. 5 amp = 5 gm Magnesium sulphate

Give 10 ml deep IM in alternate buttock every 4 hourly

Table-5: Precautions

Precautions:

- DO NOT give 50 % Magnesium sulphate solution IV without diluting it to 20 %
- **DO NOT** give rapid IV infusion of MgSO4 as it can cause respiratory failure or death.
- If respiratory depression occurs (RR < 16 breaths/minute) do not give the next dose.
- Give antidote Calcium gluconate 1 g IV (10 ml of 10 %) over a period of 10 minutes.

2.2.3 Controlling blood pressure

Nifedipine the drug of choice for controlling BP. Tab. Nifedipine 10 mg orally, followed by 6 hourly, after taking BP.

Labetalol: Intravenous Labetalol ($\alpha 1\beta$ blocker) is another commonly used drug for severe hypertension. It is given in a dose of 20 mg IV initially. If BP has not decreased to the desirable level in 10 min then 40 mg is given. If the BP has still not decreased in the next 10 minutes, an incremental dose of 80 mg may be given. This may be followed by another 80 mg every 10 min if needed till a maximum of 220 mg has been administered. Cardiac monitoring is required. Avoid in bronchial asthma and in cardiac disease.

2.2.4 Delivering the baby

The mode of delivery should be decided depending on whether or not the woman has gone into labour and the stage and progress of labour. In eclampsia, delivery should occur within 12 hours of the onset of convulsions.

- If woman is in active labour: Monitor the progress of labour and deliver. Watch for signs of fetal distress. Augment labour by Amniotomy and Oxytocin infusion as required.
- Cut short the second stage of labour.
- Do not give Inj. Methyl ergometrine.
- If woman is not in labour: Assess condition of cervix and induce labour with vaginal or oral Misoprostol 25 mcg tablets or ARM & Oxytocin infusion.
- Mode of delivery depending on fetal condition and condition of cervix.
- Perform LSCS if: Cervix unfavorable, Fetal distress, Fits are not controlled, labour is not progressing well despite induction/ augmentation or for any other obstetric indication.

2.2.5 Maintaining the fluid balance

- Monitor urine output. It should be at least 30 ml/hour.
- Record the fluid intake. Give all the necessary fluids slow IV @ 60 ml per hour.
- Maintenance of proper fluid balance is essential to prevent water intoxication, dehydration, hyponatremia, or pulmonary oedema.
- Diuretics should not be used unless there are signs of pulmonary edema.

2.2.6 Post-Partum Care

Fits can also occur for the first time in the immediate postpartum period. Monitor BP and the urine output after delivery. Continue antihypertensive to maintain diastolic BP between 90 - 100 mm Hg. Advise the woman to have her BP checked regularly until BP returns to normal. If BP remains high at 12 weeks diagnose her as chronic hypertension and refer her to physician for evaluation.

3. Vaginal Bleeding During Early Pregnancy

A woman may present with history of a short period of amenorrhea followed by vaginal bleeding. Abortion, vesicular mole and ectopic pregnancy are the common underlying conditions, while an occasional woman may simply have a delayed menstruation.

3.1 Ask patient following

- Period of amenorrhea, LMP, symptoms suggestive of pregnancy. Prior H/O abortion, ectopic pregnancy.
- Amount and duration of bleeding, it may be scanty in threatened and missed abortion and profuse in incomplete / inevitable abortion.
- Nature and severity of pain (severe in ruptured ectopic pregnancy)
- Clinical presentation may be as Threatened abortion, Inevitable abortion, Incomplete abortion, Complete abortion, Missed abortion or Septic abortion.
- Arrange for ultrasonography and pregnancy test.
- Arrive at the diagnosis with the help of following chart. (Table-6)
- Refer to specialist for further management.

3.2 Differential diagnosis

Particulars	Threatened abortion	Incomplete abortion	Missed abortion	Hydatidiform Mole	Ectopic pregnancy
Uterine Size	Equal to POA Internal os closed	Smaller Internal os open	Smaller	Bigger	Smaller
Vaginal bleeding	Slight	Profuse	Absent or brownish discharge	Recurrent small	Slight
Pain	Mild or Absent	Cramping pain significant	Absent	Absent	Severe, continuous
G.C. pallor /tachycardia	Fair	Proportional to blood loss	Fair	Fair	Out of proportion to visible blood loss.
Tenderness Abdominal /vaginal	Absent	Absent (Unless infected)	Absent	Absent	Marked
USG	Intrauterine viable pregnancy	Some products in uterine cavity	Nonviable pregnancy	Snowstorm appearance	Empty uterus pelvic / adnexal mass
Risks	Preterm /IUGR	Hemorrhage Sepsis	Blood coagulation failure	Hemorrhage	Shock
Management	Expectant	Surgical evacuation Antibiotics	Termination of pregnancy	Suction evacuation Blood transfusion if required	Laparoscopy or Laparotomy, Blood transfusion

Table 6: Differentiating Features according to Condition

3.3. Management of Threatened abortion

Avoiding exertion, sexual abstinence and regular follow up to monitor the growth of the fetus are

recommended. The role of progesterone therapy or human chorionic gonadotropin is unproven.

Follow up: Regular ANC check up every 15 days. The pregnancy has higher risk of having recurrent episodes of bleeding, bleeding in late pregnancy due to APH, fetal growth retardation and preterm delivery.

3.4. Management of Missed Abortion

Investigation: Ultrasonography. Bleeding time, clotting time, prothrombin time, platelet count (risk of coagulation failure with prolonged retention of dead fetus).

Treatment: The pregnancy needs to be terminated for maternal anxiety and for risk of blood coagulation failure leading to excessive bleeding.

Uterus < 12 weeks: Manual/electrical vacuum aspiration.

Uterus > 12 weeks: Pregnancy termination by Misoprostol.

3.5. Septic abortion

Causes: Unsafe abortion. Anemia, delay in emptying the uterus in cases of incomplete abortion, failure to follow adequate aseptic precautions.

Symptoms and signs: Fever, tachycardia, lower abdominal pain, offensive vaginal discharge and pelvic tenderness.

Investigations - Bleeding time, clotting time, prothrombin time, platelet count are done as there is risk of coagulation failure.

Treatment: Antibiotics. Inj. Ampicillin 1gm stat IV followed by Inj. ampicillin 500 mg.6 hourly, Inj. Gentamicin 80 mg 12 hourly, Inj. Metronidazole 100 ml IV 8 hourly slowly.

Surgical evacuation of uterus if there are retained products.

Cases having severe sepsis with complications need to be referred to district hospital.

3.6. Ectopic pregnancy

The commonest site of ectopic pregnancy is the fallopian tube.

Clinical presentation: Unruptured tubal pregnancy; tubal abortion or tubal rupture with severe intraperitoneal bleeding leading to shock.

3.6.1 Unruptured ectopic pregnancy:

Diagnosis requires a high index of suspicion. The woman can reveal some of the risk factors for ectopic pregnancy, will present with signs and symptoms of pregnancy, may have a unilateral adnexal mass. UPT positive; ultrasonography reveals an empty uterus.

Refer to specialist for laparoscopic surgery.

Medical management for selected cases: Inj Methotrexate under careful monitoring.

3.6.2 Tubal abortion:

Symptoms:

Significant pain in lower abdomen, vaginal bleeding could be slight or even absent.

Signs: Tenderness in lower abdomen.

P/V: Cervical movements extremely painful and tenderness in fornices.

Pallor and tachycardia depending on the amount of internal bleeding.

Clinical condition usually Stable.

Investigations: Ultrasonography reveals free fluid in abdomen.

Treatment: Referral to gynecologist for surgical management.

3.6.3 Ruptured ectopic pregnancy:

Severe and acute pain in abdomen with severe intraperitoneal bleeding which is continuing.

Signs: The patient presents with tachycardia, severe pallor and hypotension. The abdomen is tender and distended. Signs of free fluid in abdomen (shifting dullness). Vaginal examination is extremely painful and an ill-defined mass may be felt in posterior and lateral fornix.

USG: Free fluid in abdomen and vague ill-defined pelvic mass may be seen.

Culdocentesis: Blood is aspirated which fails to clot on observation.

Treatment: Management of shock. Urgent surgical exploration to stop the bleeding. Blood transfusion as required.

3.7 Hydatidiform Mole

An abnormal pregnancy where there is no fetus, no amniotic sac and the uterus is full of proliferated trophoblastic tissue.

Symptoms: The pregnancy vomiting is often exaggerated and there is recurrent vaginal bleeding rarely with passage of grape like vesicles.

Signs: Uterus is larger than the POA and soft / doughy in consistency. Tachycardia, pallor.

Investigation: Ultrasonography reveals snow storm appearance, absence of fetus, amniotic fluid. The ovaries may be enlarged with multiple cysts.

Pregnancy hormone beta HCG is markedly elevated.

Treatment: The pregnancy is terminated by suction evacuation under general anesthesia. Blood is arranged.

Complications:

- Severe hemorrhage and uterine perforation during evacuation.
- Infection.
- Development of gestational trophoblastic neoplasia.

Follow up: Baseline X ray chest and β HCG levels tested before discharge. Regular β HCG monitoring till it falls to normal level and for 6 months thereafter as there is risk of development of gestational trophoblastic neoplasia.

It is important to avoid pregnancy by using reliable contraception during this follow up period. Oral pills can be given. **Intrauterine contraception is not recommended.**

During follow up: Look for abnormal vaginal bleeding, enlarged soft uterus, evidence of any metastasis (lungs, brain, anterior vaginal wall)

Pregnancy is allowed when β HCG remains normal for 6 months. Referral to tertiary care center is indicated for chemotherapy if β HCG levels show initial decline followed by a rise or it remains static (plateau).

4.Antepartum Haemorrhage (APH)

4.1. Definition:

Bleeding from vagina after 20 weeks of pregnancy and before the birth of child. Two major causes:

- Abruptio placentae: Bleeding from premature separation of normally located placenta in the upper uterine segment.
- Placenta Previa: Bleeding from premature separation of low lying placenta located in lower uterine segment.



Figure 4.1: Placenta praevia

4.2. Dangers:

Maternal mortality is high due to hemorrhagic shock and its complications like acute renal failure (ARF), coagulation failure due to DIC. There is increased risk of atonic PPH after delivery. Lacerations of friable cervix also cause bleeding. Operative delivery rates are high.

Fetal and neonatal mortality is high due to prematurity (Spontaneous & iatrogenic), LBW and asphyxia.

4.3. Case Management of APH

4.3.1 History:

- Ask the period of amenorrhea, fetal movements, time of onset of bleeding, amount, prior H/O warning hemorrhages.
- Abdominal pain present or absent and its severity.
- Passage of urine.
- Prior USG reports if available.
- H/O trauma, internal examination/ coitus aggravating the bleeding.

4.3.2 Examination:

- Note pallor, tachycardia, general condition, record blood pressure.
- In concealed accidental hemorrhage, although the woman is in shock, her BP may be normal as she could be having prior hypertension. Therefore, degree of pallor and tachycardia are more important for assessment of amount of bleeding and general condition.
- P/A:
 - Uterus is relaxed, contracting and relaxing or hard.
 - Uterine tenderness present/absent (localized or generalized)
 - o Fetal presentation, FHS

No vaginal examination to be done until placenta praevia is ruled out

4.3.3 Investigations:

• Urgent USG to locate placenta and assess the fetal condition and its gestational age.

4.4 Placenta Previa

- Blood tests: Hb, PCV, Coagulation profile (Bleeding & clotting time, clot observation test, platelet count, prothrombin time).
- Liver function and renal function tests.

4.4.1 Differentiate between Placenta Previa and Abruptio placentae

Particular	Placenta praevia	Concealed or mixed abruptio placentae
Pain	Painless recurrent vaginal bleeding	Painful bleeding
General condition	Pallor, tachycardia, restlessness proportional to visible amount of blood lost	Pallor, tachycardia disproportionately more than the visible amount of vaginal bleeding
Uterus	Relaxed, non-tender, fetal parts felt well	Tense, tender, woody hard, fetal parts cannot be felt
Presentation	May be abnormal or head may be high floating	Presentation cannot be made out
Fetus	Fetal condition usually well if mother not in shock	Fetus often distressed or dead. FHS may not be heard
Maternal Dangers	Hypovolemic shock	Shock, acute renal failure, DIC
Action	No P/V examination. IV fluids, referral	IV fluids, referral, monitor

Table-7: Differentiation between Placenta Previa and Mixed Abruptio Placentae

4.4.2 Complications:

Maternal: Hypovolemic shock, malpresentation, operative delivery, PPH

Baby: Prematurity, low birth weight, fetal/neonatal asphyxia

4.4.3. Management:

- Even if bleeding is only a very small amount, hospitalization is required.
- Assess the blood loss.
- Full blood count and clotting studies.
- Arrange cross matched blood

Perform gentle palpation of abdomen to assess the gestational age of fetus, presentation and position.

- Assess fetal condition. Arrange urgent ultrasound.
- Rh negative woman: With every episode of bleeding, give prophylactic anti-D immunoglobulin.

- If pregnancy duration is > 36 weeks or if there is life threatening bleeding, then pregnancy is terminated.
- Expectant management until 36 weeks for clinically stable patient with moderate blood loss having fetus alive and preterm
- P/S: Exclude local genital causes (cervical growth, polyps, vascular ectropion)
- Pregnancy is terminated in maternal interest at any time during the observation period if there is furious uncontrolled bleeding or when pregnancy > 36 weeks.

4.4.4 Mode of delivery:

- Caesarean section for major degree placenta praevia and for cases having type II posterior placenta praevia with a salvageable fetus.
- Previous CS & anterior placenta praevia: There is risk of morbidly adherent placenta leading to furious hemorrhage. Refer such case to tertiary care centre. Such case needs to have 4 units of blood to be kept ready and preparations for CS hysterectomy if required.
- Cases having low lying placenta, (type II anterior) can be delivered vaginally by

Amniotomy followed by Oxytocin infusion. Arranging blood and the delivery at wellequipped institution is necessary.

4.5 Abruptio Placentae



Figure 4.2: Abruptio placentae

4.5.1 Diagnosis:

- May present with vaginal bleeding and abdominal pain.
- Ask H/O trauma, prior hypertension.
- Assess blood loss: Look for pallor, tachycardia, general condition, urine output, record BP. Hypertension and proteinuria may be present.
- P/A uterus hard, tender, not relaxing (Hypertonus)
- In cases of severe abruption, fetal parts may not be felt well, fetus may be distressed/dead. When the fetus is dead at least 1,500 ml of blood is lost, hence shock is usual, uterus is firm-to-hard and very tender. Blood pressure may be normal in spite of being in shock as the initial BP may be high. Coagulation failure leading to severe bleeding is common.
- USG shows placenta in upper uterine segment and may show retroplacental hematoma.

Avoid Errors in Diagnosis

- The case could be misdiagnosed as preterm labour.
- A patient of concealed accidental hemorrhage can be misdiagnosed as patient in labour with severe anemia with fetal death.
- In labour, uterine rupture can be misdiagnosed as concealed accidental hemorrhage.

4.5.2 Management:

- If fetus is alive and salvageable an urgent CS is performed, hence referral to nearest facility is indicated.
- If fetus is dead, it indicates massive retroplacental hemorrhage. Hemodynamic stability is assessed (pulse, BP, pallor). Foley's catheter is introduced and hourly urine output is monitored. Coagulation profile is checked.
- Correction of hypovolemia: Ringer lactate infusion and by blood transfusion.
- Refer the woman to tertiary level of care having facility of providing blood and blood products for treatment of DIC.
- Induce labour by ARM and Oxytocin infusion.

4.5.3 During Labour:

- First stage of labour is augmented by ARM and Oxytocin infusion.
- If fetus is alive, watch for signs of fetal distress.
- Second stage of labour is cut short.
- Active management of third stage of labour.
- Placenta is examined and the retro placental clot is weighed.
- Hemoglobin and hematocrit estimation following delivery.

5. Preterm labour

Prematurity and low birth weight is the important causes of neonatal deaths in India.

Preterm labour is onset of labour from 24 weeks to before 37 completed weeks of gestation.

From survival point of view, the gestational age of 34 weeks is important as after this period the fetal lungs are mature enough for independent survival outside the uterus.

A single course of antenatal corticosteroids administered between 24 to 34 weeks of pregnancy has been shown to reduce the neonatal mortality significantly in preterm born babies.

5.1. Risk Factors

• Maternal weight < 40 Kg in first trimester. (BMI < 19.8 kg/m2), Obesity (BMI > 30 kg/m2).

- Teenage pregnant women
- Short inter pregnancy interval.
- Short stature, maternal undernutrition, anemia.
- Inadequate weight gains during pregnancy
- Strenuous working, long hours of work, inability to take adequate rest.
- Tobacco, smoking, heavy alcohol use, illicit drug use.
- Previous H/O preterm birth, mid trimester abortion. Risk increases with each additional preterm birth.
- Incompetent cervix, uterine malformations.
- Vaginal infections: Bacterial vaginosis.
- Over distended uterus (Twins, polyhydramnios), Vaginal bleeding during pregnancy, Chorioamnionitis.
- Diabetes, hypothyroidism, heart disease, chronic hypertension, HIV illness, urinary tract infection(UTI), periodontal infections.
- Fall, psychological stress, abdominal surgery during pregnancy.

5.2. Prevention

- Treatment for UTI, correction of anemia, improving oral hygiene.
- Nutritious diet and consumption of IFA tablets.
- Counseling: Avoid strenuous work, take extra rest, quitting tobacco, alcohol.
- Women with previous history of preterm birth: Refer to specialist.
- Vaginal sonography to see the length of cervix.
- Weekly Injections of 17 hydroxyprogesterone.
- Cervical os tightening in selected cases are some of the preventive interventions.

5.3. Prediction by warning symptoms

- Cramping discomfort/pain in lower abdomen, low backache.
- Increased mucoid vaginal discharge.
- Intermittent uterine contractions felt during discomfort.
- Heaviness in pelvis (something descending down).

5.4. Diagnosis

Assess pregnancy duration accurately

Confirm onset of labour: Intermittent painful uterine contractions (4 in 20 min or 8 in 60 mins) along with progressive change in the cervical dilatation and effacement assessed over 2 hours

5.5. Management principles

5.5.1 If pregnancy is < 34 weeks

- Antenatal Dexamethasone therapy
- Inhibition of preterm labour by administering tocolytic medicine.
- 'In Utero transfer'.
- Safe conduct of preterm labour.
- Special care of preterm neonate.

a) Antenatal corticosteroid therapy

- Injection Dexamethasone administered between 24- 34 weeks of pregnancy in cases of threatened preterm labour is effective in lowering the risk of respiratory distress syndrome (RDS) & neonatal mortality if birth was delayed by at least 24 hours after initiation of therapy. The effect persists for 7 days after completion of course of steroid therapy.
- Injection Dexamethasone 6 mg IM 12 hourly for 48 hours, total 4 doses. Repeated courses should not be given.

Who can receive the therapy?

- All women having spontaneous onset of preterm labour and those having complicated pregnancies (Severe preeclampsia, antepartum hemorrhage etc).
- Preterm rupture of membranes before the onset of labour pains (PROM)

Which women should not receive corticosteroid therapy?

If there is chorioamnionitis, (infection of fetal membranes) delivery is not to be postponed. Early delivery is safer for both mother and her baby. Signs are.

- Fever, lower abdominal pain, Tenderness over the uterus
- Fetal tachycardia (FHR > 160/minute), Foul smelling liquor
- b) Inhibition of uterine contractions by tocolytic drugs:

Tocolytic drugs stop contractions temporarily but rarely prevent preterm birth. However, the delivery is delayed long enough for getting the benefits of administration of Dexamethasone.

Indication for Tocolysis

- If labour starts between 24 weeks to 34 weeks.
- The cervix is < 3 cm dilated.
- There is no foetal distress.
- Tocolysis is not indicated in Amnionitis, preeclampsia, active bleeding & foetal distress.
- Nifedipine 20 mg oral stat followed by 10 mg 4 hourly till contractions stop or maternal pulse exceeds 120 beats/min. Continue drug till steroid cover is complete or for 8 hours after last contraction. Then taper the dose slowly.
- Alternatively, beta adrenergic receptor agonists (Ritodrine, Terbutaline, Isoxsuprine) can be given. However, their value is not proven. Terbutaline is a selective beta agonist having lesser cardiovascular side effects and can be administered subcutaneously.
- Cardiac disease needs to be excluded before giving these drugs. While administering tocolytic drugs careful monitoring is required for side effects. Monitor pulse, BP, signs of respiratory distress, uterine contractions, loss of amniotic fluid or blood, FHR, fluid balance, blood glucose. Watch for maternal acute pulmonary oedema when combination of steroids and tocolysis is used.
- Do NOT give tocolytic drugs for more than 48 hours.

c) 'In Utero transfer': If the period of gestation is < 34 weeks, and labour continues despite tocolysis, or if the woman is already having cervical dilatation >3 cm, refer to district hospital/medical college with NICU/ SNCU facility for delivery. Before referral always give at least the initial shot of steroid mentioning the time on the referral note. Record BP, urine test result on referral note.

d) Safe conduct of preterm labour

- Do not inhibit preterm labour and allow it to progress if
 - The cervix is > 3 cm dilated.
 - There is active bleeding, severe preeclampsia, Amnionitis
 - Fetus is distressed, dead or has an anomaly incompatible with survival.
- If a woman presents with preterm labour which is progressing rapidly, there may not be adequate time available for referral. Still give the first dose of Inj. Dexamethasone as even 1

dose few hours before delivery has some beneficial effects.

- Check fetal presentation as possibility of abnormal presentations is higher in preterm labour needing skilled attendance.
- Monitor the progress of labour.
- Prevent birth trauma and birth asphyxia while conducting breech delivery.
- Avoid delay in second stage by giving episiotomy to prevent trauma to the baby's head. Do not use vacuum extractor as the risk of intracranial bleeding is high.
- If labour continues and the gestation period is < 37 weeks; although evidence is not in favour of prophylactic antibiotics but as woman is coming from hygienically poor area, antibiotics can be given to reduce the chances of infection in the neonate. (Cap. Ampicillin, Tab Metronidazole and Inj. Gentamicin)

e) Special care of preterm neonate

If needed, refer neonate to the nearest NICU/ SNCU after appropriately stabilizing new born

6. Intrapartum & Postpartum Complications

6.1 Abnormal Presentation:

During labour fetal malpresentation is diagnosed by palpating the presenting part on vaginal examination.

6.1.1 Breech Presentation

In complete breech presentation the presenting part is buttocks with feet, in frank breech buttocks only and in footling presentation only feet are felt.

The chance of cord prolapse is highest in footling breech while it is lowest in frank breech. It is necessary to avoid rupturing membranes during vaginal examination.

6.1.2 Transverse lie

Fetal shoulder felt. After rupture of membranes hand prolapse occurs, cord prolapse may occur. These cases need to be referred to FRU urgently for caesarean delivery.

6.1.3 Face presentation:

Chin and face felt. If the chin is anterior (left or right mento-anterior position) vaginal face delivery is possible specially in a multipara having average sized baby. However, in mentoposterior position, caesarean delivery is often required. Refer these women to FRU, however a midwife should accompany with a delivery tray during referral.

6.1.4 Brow presentation:

Baby's forehead and part of the face is felt and chin is not easily felt. A good sized baby's head is unable to get engaged in the pelvis and the labour is likely to get obstructed. Such woman should be referred to FRU for caesarean section.

7. Breech delivery

If the full term breech presentation is diagnosed in early labour the case should be referred to higher centre. Decision to deliver by Caesarean Section or allowing vaginal delivery needs to be taken after careful evaluation by specialist. Maternal age, parity, pelvic capacity, status of membranes, type of breech, estimated weight of baby, other complicating medical /obstetrical conditions, scar on uterus are some of the important considerations for this decision.

However sometimes woman arrives in advanced labour when you are required to conduct her delivery by performing the following steps:

- Check the delivery tray, episiotomy tray, baby tray and newborn resuscitation equipments
- Start IV infusion
- Tell the woman to bear down with contractions only when the cervix is fully dilated and the buttocks are in the vagina
- Wash hands with soap and water and put on sterile gloves.
- Take the woman to the edge of the table
- Clean the vulva with antiseptic solution and drape the mother.
- Catheterize the bladder, if necessary.
- Give pudendal block or local perineal infiltration for episiotomy.
- Give episiotomy when the buttocks distend the perineum.
- Do not pull the baby or interfere in any way
- When the baby is born up to umbilicus, pull a loop of cord and keep it aside. Cover the baby with a clean sheet and gently hold the buttocks of the baby, but do not pull.
- If the legs do not deliver spontaneously, deliver one leg at a time.
- Ask the mother to continue pushing with contractions.

- Check whether arms are on the chest. Allow the arms to get rotated and disengage spontaneously.
- If the arms are stretched above the head or folded around the neck (Winging of the scapulae indicates extension of arms) use Lovset's manoeuvre to deliver arms.
- Deliver the head by Burns Marshal manoeuvre or Mauriceau-Smellie-Veit manoeuvre (jaw flexion and shoulder traction).
- Observe baby's breathing and initiate resuscitation as required (Breech born baby takes longer time to start normal breathing)
- Perform active management of the third stage of labour.
- Check the birth canal for tears and repair episiotomy.

8. Twins delivery

A case of diagnosed twin pregnancy arriving in early labour should be referred immediately as the babies are often LBW and require special care. However, if the mother is admitted in advanced labour the following steps should be taken to deliver her.

- Check presentation of first baby, check FHS
- Start IV Ringer lactate
- If first baby is presenting by vertex allow labour to progress and deliver the first baby. Leave a clamp on the maternal end of the cord of the first twin and do not give Oxytocin.
- Palpate abdomen to determine the lie of second fetus. If it is breech or transverse attempt to perform external cephalic version.
- Check the presentation of second baby by vaginal examination. If vertex, allow labour to progress and deliver the baby.
- If the uterine contractions become weak and delivery of second baby is getting delayed start Oxytocin drip slowly.
- If the presenting part is breech, conduct breech delivery. If delivery is getting delayed or if fetus shows signs of distress, perform breech extraction carefully.
- If second baby is transverse refer to FRU for CS. (If the baby is small and very preterm sometimes it may deliver spontaneously, hence it is necessary to accompany her with preparations to conduct delivery during transfer)
- Give Oxytocin soon after delivery of second baby and conduct active management of third stage of labour.
- Anticipate risk of PPH and check for hardness of uterus and massage the uterus. Start IV Oxytocin infusion (20 IU in 500 ml of Ringer lactate)
- Weigh both babies and take steps for special care needs if the babies are LBW.

9. Cord Prolapse

Cord prolapse is more common if the presentation is breech or transverse. In vertex presentation cord gets severely compressed as head descends and baby gets severely asphyxiated and needs urgent delivery and prompt resuscitation

- Determine the lie and the presenting part
- Note the fetal heart rate
- P/V examination to determine the stage of labour and the presenting part
- Gently feel for the cord pulsations. Push the cord in the vagina
- If first stage; push the head upwards to relieve pressure by presenting part on cord and give head low position to mother. Alternatively, insert a Foley catheter in urinary bladder and rapidly fill the bladder with 300-500 ml Normal Saline to elevate the fetal presenting part. Clamp the catheter. The clamp must be released and the bladder drained before any delivery attempt.
- Refer immediately to FRU for Caesarean Section.
- If in second stage; expedite delivery with episiotomy and vacuum/forceps delivery.
- Perform breech extraction, if the baby is presenting by breech.
- Check for breathing and resuscitate the newborn.
- If the baby is very premature or dead spontaneous delivery can be awaited.

10. Shoulder Dystocia

If the baby is big the chances of shoulders getting stuck after delivery of head are high. Many babies die within few minutes of the head being delivered. It is important, therefore, to manage the problem efficiently and carefully so as to avoid injuries to the baby or mother.

- Call for help
- Discourage maternal bearing down effort

- Apply Mc Roberts maneuver i.e. bring knees as far as possible up to the chest and abduct and rotate legs outwards
- Apply firm suprapubic pressure using the heel of the hands
- Make adequate episiotomy
- Rotate the baby's body so that the posterior shoulder moves anteriorly.
- If these measures fail to deliver shoulders, attempt internal rotation maneuvers to deliver anterior or posterior shoulder. (Application of pressure to the anterior/posterior shoulder in the direction of the baby's chest)
- Check baby's breathing and initiate resuscitation
- Complications: PPH, vaginal/perineal tears and Injuries to baby.

11. Obstructed Labour

11.1 Causes

- Cephalopelvic disproportion due to contracted pelvis or large head.
- Transverse lie, Brow presentation, Mento posterior face presentation, Deep transverse arrest of fetal head.

11.2 Diagnosis

- Non progressive labour, prolonged labour.
- Maternal exhaustion: Tachycardia, restlessness, sweating, mild fever, signs of dehydration.
- Signs of fetal distress or death.

11.3 Signs of obstruction

- Lower uterine segment over stretched
- Bandl's ring seen which is rising progressively towards umbilicus.
- Suprapubic bulge Edematous bladder.
- Presenting part High, abnormal or showing excessive molding.
- On catheterization, scanty high colored/ blood stained urine.
- Early signs include edematous cervical lip, increasing molding of fetal head.

11.4 Action

Urgent referral. IV infusion during referral will help in correcting dehydration. Delay in referral can result in uterine rupture, infection due to prolonged labour and foetal death. Vesicovaginal fistula may develop if presenting part is deep in the pelvis for prolonged period as in deep transverse arrest.

At the institution the patient will be delivered mostly by Caesarean Section.

12. Uterine Rupture

12.1 Causes

- Scarred uterus (previous Caesarean Section, uterine perforation etc.)
- Obstructed labour
- Incorrect use of oxytocic agents like Oxytocin, Prostaglandins
- Internal Podalic version, manual removal of placenta.

12.2 Diagnosis

- Cessation of uterine contractions after prolonged obstructed labour.
- Continuous pain in abdomen.
- Maternal tachycardia, restlessness, increasing pallor, hypotension, fresh vaginal bleeding, haematuria.
- Fetus dead.
- Abdomen distended, tender all over, fetal parts may be felt easily.
- Signs of free fluid in abdomen.
- In labour, presenting part may recede upwards.

12.3 Dangers:

Maternal shock and death due to severe internal hemorrhage.

12.4 Action

- Urgent referral to tertiary care centre where blood transfusion facility is available.
- IV infusion of 1 liter of fluids, start antibiotics, monitor vitals.
- Exploratory laparotomy, blood transfusion.
- At laparotomy: Hysterectomy or repair of tear.
- If only repair of the rent is done, there is risk of uterine rupture in subsequent pregnancy and an elective Caesarean Section is required.

13. Third Stage Complications

13.1 Postpartum Hemorrhage

13.1.1Definition:

Excessive vaginal bleeding > 500 ml after delivery.

Even moderate amount of bleeding could lead to shock in women who are anemic or have preeclampsia.

13.1.2 Types:

Immediate (Primary) PPH: Within first 24 hours after childbirth.

Delayed (Secondary) PPH: After 24 hours up to 42 days after childbirth.

Although PPH is more common in cases of twin pregnancy, APH, over distended uterus, grand multiparas, it is an unpredictable complication and can occur in any low risk woman.

13.1.3 Probable cause of Excessive Bleeding and Shock after childbirth.

Table 8: Causes of Excessive Bleeding and Shock after Childbirth.

Clinical Presentation	Probable diagnosis
Uterus is flabby, not contracted, fundus is rising.	Atonic PPH
Uterus is well contracted. Speculum examination of cervix and vagina reveals cervical/vaginal lacerations/tears.	Traumatic PPH
Placenta not delivered within 30 minutes after delivery.	Retained placenta
Placenta incomplete, torn vessels in the membranes.	Retained lobe of placenta
Bleeding continues even after treatment. Blood does not clot for > 15 minutes.	Coagulation failure
Uterine fundus not felt per abdominally. Inverted uterus seen in vagina or outside vulva.	Acute Uterine Inversion
Delayed PPH. Uterus is larger for the postnatal day, soft, may be tender, bleeding may have foul smell.	Uterine infection, retained lobe/fragments of placenta

13.1.4 Initial Management of Atonic PPH:

The management includes, uterine massage, bimanual compression of uterus, administering uterotonic drugs along with blood volume replacement and evaluation to look for other causes.

- Check for flabby uterus, retained placenta, tears and lacerations in vagina and cervix.
- If the uterus is flabby/soft, give continuous uterine massage and bimanual compression of uterus immediately (Fig 4.3).
- Secure intravenous access and collect blood samples – Two 16 or 18 gauge cannulae are inserted in two veins for correcting hypovolemia by rapid infusion of IV Ringer lactate/Normal Saline.
- Start Oxytocin infusion 20 IU in 500 ml of Ringer lactate@ 40-60 drops/minute. If uterus remains flabby, other oxytocic agents (Inj Methyl ergometrine, tablets Misoprostol, Inj 15 methyl PGF_{2 à},) can be given in dosage given in table 9.
- Send blood sample for cross matching of 3-4 units of blood
- Send blood sample to laboratory for full blood count, coagulation profile, baseline urea and electrolytes testing.
- Inspect placenta if she is recently delivered.
- Auscultate lung bases frequently to rule out pulmonary edema.
- Oxygen is given by face mask: 8 L/min
- Keep the woman warm. Elevate the legs.
- Wash hands and wear surgical gloves.
- Insert Foley catheter and monitor hourly urine output. Urine output less than 20 ml /hour indicates poor perfusion of tissues.

- Monitor woman's GC, pulse, blood pressure, respiratory rate, temperature every 15 minutes. Note pallor. Assess whether the woman has heavy bleeding and she is in shock.
- Uterine tamponade with condom catheter may be tried.
- If bleeding is controlled by drugs, repeat uterine massage every 15 minutes for 2 hours.
- Monitor vitals closely every 10 minutes for 30 minutes, every 15 min for next 30 mins, and every 30 min for next 3-6 hours or until stable. Continue Oxytocin infusion. (max 100 IU in 24 hours)
- If the bleeding does not stop, explore uterine cavity for retained placental bits/lobe.
- If the uterus is hard, explore the lower genital tract in good light to look for genital trauma and repair any tears followed by firm packing of vagina to stop the oozing from lacerations.
- If the blood sample fails to clot, reversal of coagulation defects needs to be achieved by administering blood and blood products. Execute urgent referral to higher facility.
- Arrange urgent referral to FRU, with accompanying HCW and relatives. Continue Oxytocin infusion & oxygen during transit.
- If the woman is in shock manage shock. The first drip should be fast and timed for approximately 1 litre in 20 min. Estimated replacement is usually 3 times the blood loss. Attempt compressing the abdominal aorta by firm pressure with a closed fist just above the umbilicus during transit (Fig 4.4).
- If it is delayed PPH, look for signs of infection and administer the first dose of antibiotic.

	Oxytocin	Methyl Ergometrine	Misoprostol	15 Methyl PGF2alfa
Dose & Route	IV infusion 20 IU in 500 ml of RL/NS @ 40-60 drops/min	0.2 mg IM or IV slowly. Can be repeated after 15 mins, thereafter 4 hourly if required	800 micrograms (4 tablets of 200 mcg each) per rectally	0.25 mg IM Can be repeated every 15 minutes
Maximum dose	100 IU in 24 hours	5 Injections (Total 1.0 mg)	800 micrograms	8 doses (Total 2 mg)
Precautions & contraindications	Do not give as IV bolus Inj	Preeclampsia, hypertension	Safe drug	Rule out Asthma
Side effects	Hypotension if given IV bolus	Vomiting	Nausea, vomiting, chills	Nausea, vomiting, diarrhea

Table-9: Management of Atonic PPH

	Oxytocin	Methyl Ergometrine	Misoprostol	15 Methyl PGF2alfa
Remarks	Safe and effective Refrigeration is required	Causes sustained retraction of uterus Refrigeration is required	Stable at room temperature, cheap	Refrigeration is required

a) Oxytocics for Management of Atonic PPH

First start Oxytocin, if not responded give Methyl Ergometrine, if uterus still flabby Misoprostol, if no response 15 Methyl PGF₂ alfa.

b) Uterine Tamponade with a sterile condom is a simple procedure which can be carried out by any SBA as a life saving measure before referral. After putting the patient in the lithotomy position, under aseptic precautions, a sterile rubber catheter is inserted within the condom and tied near the mouth of the condom by a silk thread and introduced in the uterine cavity. The condom is inflated with 250-500 ml normal saline. The uterine condom kept in position by ribbon gauze pack placed in the vagina. The condom catheter is kept for 24-48 hours, depending upon the initial intensity of blood loss. It is gradually deflated when bleeding is controlled. Uterine contraction is maintained by Oxytocin drip for at least 6 hours after the procedure. Antibiotic cover is given.

For uncontrolled PPH surgical procedures may be required.

13.2 Retained Placenta

13.2.1 Definition:

When the placenta fails to deliver for 30 minutes after the birth of baby in spite of routine measures including administration of Oxytocin Injection and controlled cord traction, it is necessary to perform manual removal of placenta (MRP)

13.2.2 Management (Manual removal of placenta)

- Check woman's pulse, blood pressure, respiration. Assess the amount of blood loss. Check whether she is in shock.
- Wash hands, Wear plastic apron, gown, mask and long surgical gloves.
- Start IV Ringer lactate, start Oxytocin infusion.
- Catheterize the bladder and monitor urine output.
- Give premedication. Clean the perineum and the vagina with antiseptic solution.

- Hold the cord with left hand and introduce the whole right hand in the vagina, pass it through the cervical canal into the uterine cavity. Support the fundus with left hand. Locate the edge of the placenta, separate it completely by slicing movement, remove the separated placenta and explore the uterine cavity to check that it is empty Continue Oxytocin drip, massage the uterus.
- Inspect the birth canal for any injury.
- Give antibiotics. Monitor the vital signs.

If the placenta cannot be removed completely, if the woman is in shock and needs blood transfusion refer her to tertiary care centre immediately.

If placenta cannot be separated suspect morbid adhesion and refer her as morbidly adherent placenta causes furious bleeding requiring urgent lifesaving hysterectomy.

13.3 Acute Inversion of Uterus

13.3.1 Definition:

Uterus turns inside out and may protrude outside vulva.

The woman gets very severe hemorrhage and goes in profound shock.

Usually occurs due to mismanaged third stage, giving traction on cord when placenta is yet not separated and the uterus is relaxed.

13.3.2 Management:

O/E: Pulse feeble and fast. Hypotension.

P/A: Cupping of fundus, fundus may not be felt at all.

Inverted uterine fundus may be seen at vulva or in vagina.

Check whether placenta is separated and delivered or is still attached.

No oxytocics to be given until the fundus is manually reposed.

Start IV infusion preferably in two veins, give Ringer lactate infusion, arrange Blood transfusion and treat Shock.

The freshly inverted uterus can be often replaced in position by pushing up on the fundus with the hand immediately after inversion. In case of delayed diagnosis short uterine relaxant inhalation anesthesia helps successful reposition.

After reposition, inhalation anesthesia is discontinued and Oxytocin 20 IU in 500 ml of Ringer lactate is given along with bimanual compression of the uterus to maintain the uterine retraction and the reposition.

Correct management of third stage of labour is important for preventing this dangerous complication. Do not give any traction on cord until the uterus is firmly contracted and retracted which indicates the onset of placental separation.

14 Puerperal Sepsis

About 8% of maternal deaths are attributed to postabortal or postpartum sepsis. It is a preventable complication. Early detection and prompt treatment can save a lot of morbidity associated with it.

14.1 Causes of Fever in Puerperium:

- Wound infection: Episiotomy, Caesarean Section.
- Puerperal sepsis due to postpartum endomyometritis, pelvic cellulitis, septicemia
- Urinary tract infection
- Mastitis, Breast abscess.
- Thrombophlebitis, deep vein thrombosis
- Malaria, Pneumonia, Typhoid, HIV related infections.

14.2 Symptoms:

- Fever with chills and rigors, Foul smelling lochia.
- Malaise, headache, nausea, anorexia, vomiting.

14.3 Signs:

Depending upon severity of infection

- Tachycardia, fever, hypotension in septic shock, swelling in one leg, calf tenderness. (DVT)
- P/A: Subinvoluted tender uterus, tenderness in iliac fossae, distension, guarding, absent peristalsis in peritonitis.

- Local examination: Episiotomy wound oedematous, pus discharge, foul smelling lochia.
- Vaginal examination: Uterus subinvoluted, tender, fornices tender, Bogginess in posterior fornix if pelvic abscess.

14.4 Risk Factors:

- Prolonged labour, PROM, Unclean delivery.
- Anaemia, Undernutrition, Diabetes Mellitus, HIV infection.

14..5 Investigations:

- Complete blood count with WBC total and differential count.
- Smear for malarial parasite.
- Urine routine, microscopy, culture & sensitivity in suspected cases of UTI.
- USG for retained products, collection in pelvis.
- Chest X-ray.
- High vaginal and cervical swab smear, culture sensitivity.
- Blood culture in seriously ill cases.

14.6. Management:

a) Genital sepsis

On examination patient has pallor, tachycardia, uterus is soft, tender, subinvoluted (i.e. large for the day of puerperium), abdomen soft, non-tender.

Mild sepsis can be treated at PHC level by giving oral antibiotics for 7 days.

Cap Ampicillin/Amoxicillin orally 500 mg 6 hourly for 7 days, Tab Metronidazole 400 mg orally 3 times a day.

Give Haematinics. Review after 48 hours.

If response is seen, continue treatment for 7 days. In case of deterioration, refer the patient. Antibiotics including Inj. Gentamicin 80 mg IM should be initiated by ANM/MO at the first suspicion of sepsis before referral.

b) Pelvic peritonitis

If P/A examination reveals tenderness in lower abdomen (iliac fosse) with or without distention and guarding, suspect pelvic peritonitis and refer to FRU after giving loading dose of antibiotics. Inj. Ampicillin 1 gm IV, Inj. Gentamicin 80 mg, IV Metronidazole 500 mg. This patient needs hospitalization and parenteral antibiotics for 7-10 days. Inj. Ampicillin 1 gm IV followed by 500 mg 6 hourly. Inj. Gentamicin 80 mg twice a day I.M. IV Metronidazole 500 mg 8 hourly. IV fluids are given. After 48 hours if patient improves and resumes oral intake, Cap Ampicillin and Tab Metronidazole can be given orally.

Change in antibiotics as per results of antibiotic sensitivity testing is required if there is no response to treatment within 48 hours.

c) Generalized peritonitis or septicemia:

If a patient of puerperal sepsis is looking very ill, febrile, restless, has marked tachycardia and has distention of abdomen, guarding, tenderness over abdomen, peristalsis is sluggish or absent then such patient is having generalized peritonitis. She may have septicemia. Refer her to tertiary care center, as she requires special care in critical care unit, higher antibiotics and may need surgical interventions. There is risk of death due to Endotoxic shock, acute renal failure, DIC. Any degree of puerperal sepsis when associated with severe anemia, there is risk of rapid deterioration. Hence refer such patient to a specialist at district hospital.

d) Surgical treatment:

- Retained products in uterine cavity: Evacuation of uterine cavity.
- Pelvic abscess is drained by colpotomy.
- General peritonitis may require laparotomy.
- Breast abscess: Incision and drainage.

14.7 Prevention of sepsis:

- During pregnancy: Nutrition, IFA prophylaxis, Anaemia correction, Diabetes control.
- Intranatal and postnatal: Strict aseptic measures, minimum number of vaginal examinations, early delivery in PROM cases & antibiotic prophylaxis.



Fig 4.3: Bimanual Compression of uterus

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Fig 4.4: External Aortic compression

5. MEDICAL DISORDERS COMPLICATING PREGNANCY

1. Anemia in Pregnancy

Anemia during pregnancy is a major contributory cause for maternal deaths. Nutritional deficiency is the commonest cause.

1.1. Definition:

Anemia in pregnant women is Haemoglobin level of less than 11 g/dL.

Table-1: Anemia Classification

Degree	Hb(gm/dl)
No Anemia	11
Mild	9 -10.9
Moderate	7- 8.9
Severe	Less than 7

1.2. Causes of Anemia During Pregnancy

- i. Nutritional (Iron, folic acid, proteins)deficiency anemia commonest cause.
- ii. Worm infestations.
- iii. Twin/ multifetal pregnancy.
- iv. Malaria.
- v. Urinary tract infection, Tuberculosis.
- vi. Blood loss.
- vii. Acquired hemolytic anemia.
- viii. Haemoglobinopathies: Sickle cell disease, Thalassemia.
- ix. Rare causes: Aplastic Anemia, Leukemia.

1.3. Symptoms

Weakness, Lassitude and fatigue, Anorexia and indigestion, palpitation, dyspnoea, giddiness and swelling of legs.

1.4. Signs

• Pallor, glossitis and stomatitis, edema, signs of nutritional deficiencies, Koilonychias, Platonychia.

- Precordial soft systolic murmur.
- Signs of cardiac failure, crepitation at the base of lungs.

1.5. Investigations

- Hb estimation, Haematocrit, Blood counts.
- Peripheral blood smear (PBS) to know whether Iron deficiency or B 12/ Folate deficiency.
- Haematological indices- MCV, MCH, MCHC.

To find out the cause of anemia.

- Examination of stool for worms and occult blood.
- Urine examination routine, microscopic, culture if required.
- PBS (thick and thin smear) for malarial parasite.
- Sickling test: Solubility test.
- Hemoglobin electrophoresis, HPLC in suspected haemoglobinopathy.
- Serum bilirubin: Hemolysis, Megaloblastic anemia.

1.6. Complications

Anemia contributes to 20 % of maternal death

Table 2: Complications

Maternal	Baby
Increased risk of cardiac failure.	Fetal growth restriction. (IUGR)
Inability to withstand third stage hemorrhage, can go in shock with moderate blood loss.	Prematurity
During Puerperium: Risk of puerperal sepsis, Poor establishment of lactation.	Poor stores of iron at birth, risk of anemia during infancy.
Puerperal venous thrombosis, Pulmonary embolism.	

1.7 Treatment

Treatment depends upon:

Severity of anemia, cause of anemia and gestational period at which anemia is detected.

1.7.1 Nutritional deficiency anemia

a) Prophylaxis (Hb =/> 11 G/dl):

Oral IFA tablets (Elemental iron 100mg with Folic acid 0.5mg/day) one tablet daily for 6 months during pregnancy and 6 months during lactation.

b) Mild/Moderate anemia: (Hb< 11 G/dl):

Therapeutic double dose: Oral IFA one tablet twice a day. For better absorption it should be taken before meals. Check response after 4 weeks. The treatment should be continued till the Hb becomes normal, then one tablet daily is to be continued to replenish the iron stores.

Nutritional guidance is given. Protein is supplemented.

Tab Albendazole (400mg) single dose after first trimester for deworming.

c) Response of therapy is evidenced by

- Sense of well-being, increased appetite within 3-4 days.
- After a lapse of 2 weeks, haemoglobin starts rising at the rate of 1gm % per week.
- If no significant improvement is evident clinically and haematologically, after 4 weeks refer the woman to FRU as diagnostic re-evaluation is needed.

d) Parenteral iron therapy is indicated if:

- Intolerance to oral iron.
- Patient not co-operative to take oral iron.
- Poor responders to oral iron.
- Severely anemic mothers.

Iron sucrose is safe. It helps in replenishing iron stores faster than oral iron.

Calculated dose: (2.4 \times W \times D +500mg for storage iron)

Where,

W= weight in kg (Pre-pregnancy),

D = (target Hb in gm% - actual Hb in gm%),

Rounded up to the nearest 100 mg.

Total iron is given in divided doses.

Oral iron is to be stopped before 24 hours.

200 mg iron sucrose in at least 200 ml of normal saline is given over a period of 30 minutes on alternate days. Keeping emergency drugs is a must at every clinic.

e) Severe anemia:

Patients with Hb level <7gm% or moderately anemic patients with associated obstetrical or medical complications should be referred to specialist for evaluation and management. Hospitalization helps in complete evaluation for cause of anemia.

Blood transfusion is indicated if

- Hb< 5 gm/dl any time during pregnancy
- Severe anemia seen after 36wks of pregnancy
- Refractory anemia
- Patients needing operative delivery
- Continued bleeding

Packed red cells are transfused slowly and under supervision, diuretics are given to avoid circulatory overload.

1.7.2 Treatment of other causes for anemia

Should be given as indicated. Antimalarial treatment, appropriate care for bleeding piles etc.

1.7.3 Management during Labour

a) 1st Stage:

- The patient should be in bed, propped up position, Oxygen inhalation.
- Strict asepsis. Monitor vitals, auscultate lung bases.

b) 2nd Stage:

• Avoid exertion and delay: Episiotomy, prophylactic low forceps or vacuum delivery.

c) 3rd Stage:

- Active management of 3rd stage of labour with prophylactic Oxytocin.
- Blood transfusion if required
- Precaution against post-partum overloading of heart.

d) Puerperium

- Prophylactic antibiotics
- Anti anemic therapy continued till 3 months after normalization of Hb.

Baby:

Prone to sepsis in neonatal period.

2.2. Effects of GDM on the Mother & Baby

Table-3: Effects of GDM

Mother	Baby
Pre-eclampsia	Unexplained sudden fetal death during third trimester.
Polyhydramnios	Congenital abnormalities.
Caesarian section more common due to fetal macrosomia and CPD.	Macrosomia (big baby > 4 Kg), Birth trauma. (due to shoulder dystocia)
Shoulder dystocia, maternal Injuries due to macrosomia.	Neonatal hypoglycemia
Chorioamnionitis and postpartum endometritis.	Hyaline membrane disease. (RDS)
Miscarriages in uncontrolled Diabetes.	Hypocalcemia, hypomagnesemia.
Recurrent UTI, Fungal vaginitis, skin infections.	Prematurity, Jaundice, Apnea and bradycardia.
Long term risk of type-2 Diabetes Mellitus.	Polycythemia/Hyperviscosity syndrome.

The risk of fetal anomalies is high in pre-gestational DM. It is not increased in GDM patients. However, the risks of unexplained still births (during the last 4-8 weeks of gestation) is similar to pre-gestational Diabetes.

2.3. Screening and diagnosis of Diabetes in pregnancy

All Indian women should be screened for gestational Diabetes Mellitus as they belong to a high risk

Special care if low birth weight. Iron supplements as stores are poor.

1.7.4 Patient education and counseling

- Proteins, Iron and vitamin rich diet
- Correct cooking habits
- Precaution against malaria
- Spacing between pregnancies

2. Diabetes Mellitus (DM) in Pregnancy

Due to lifestyle factors and dietary habits, incidence of type 2 DM is increasing. Gestational Diabetes Mellitus (GDM) results in higher maternal and perinatal morbidity and requires meticulous control and special care. Of all GDM patients, > 50% develop type 2 DM in later life.

2.1. Two types of cases

- Pre-Gestational DM: DM diagnosed before pregnancy.
- Gestational DM: Glucose intolerance detected for the first time during pregnancy.

ethnicity. Screening should be done at first visit (12-16 weeks) and again between the 24th and 28th weeks of pregnancy. One step screening and diagnostic test is very convenient for pregnant women in Indian scenario. As recommended by GOI guidelines 75 gm Glucose load is given orally to women attending ANC irrespective of meal status and blood sugar is estimated after 2 hours.

A plasma glucose value of 140 mg and above is considered diagnostic of GDM. A value of < 120 mg/dl is normal. Value between 120 - 139 mg/dl is considered as gestational glucose intolerance.

2.4. Management of Diabetes in Pregnancy

The woman should be referred to gynecologist at district hospital. A team of specialists including Diabetologist, Sonologist and neonatologist should manage the case in consultation.

2.4.1 Medical Nutrition Therapy(MNT):

Calories calculation for 24 hours according to weight and occupation.

30-35 kcal/ kg body weight and for obese women 25-30 kcal/kg/day.

Composition of diet– It should include carbohydrates (50-55%), proteins (20-30%) and fats 20-30%) with saturated fat <10%.

Three major meals and 3 snacks are advised.

2.4.2 Exercise:

Walking 3-4 times weekly for 20-30 min/session.

Blood sugar monitoring should be done after 2 weeks of starting diabetic diet.

Recommended glycemic goals: Fasting blood glucose < 95 mg/dl

Two-hour postprandial blood glucose < 120 mg/dl

BSL monitoring twice weekly up to 28 weeks, weekly from 28 weeks onwards. More frequently as required.

2.4.3 Insulin Therapy: If MNT fails to achieve glycemic goals within two weeks of therapy.

Insulin requirements increase as pregnancy advances. Consult a physician or endocrinologist for insulin administration. Oral antidiabetic medicines are not to be given during pregnancy as they are not yet proven to be safe for the baby. Refer Annexure and national guidelines for details of MNT and insulin therapy.

2.4.4 Obstetrical Management:

Antepartum foetal monitoring: Non-Stress test and Amniotic fluid index are performed periodically during third trimester to prevent IUD and plan delivery. Umbilical artery Doppler studies help in timing the delivery. More frequent monitoring is done if there is history of stillbirth, hypertension, and uncontrolled blood sugar. Women with GDM with poor glucose control, those with hypertension, previous still birth & other complications often require to be delivered earlier. The timing of delivery should be discussed with the obstetrician.

For well controlled uncomplicated cases can wait upto 40 weeks. It is safe not to allow to cross EDD in any case.

Caesarean Section is done for large baby and other obstetric indications.

Management during labour:

- Regular glucose monitoring and urine testing for sugar and ketones.
- Regular insulin: Low-dose infusion depending upon blood sugar levels.
- Prophylactic antibiotics.
- Baby: Monitoring for hypoglycemia and for other complications like respiratory distress, and needs special care.

2.4.5 Postpartum management:

After delivery insulin requirement decreases hence dose needs to be adjusted. Fasting and postprandial blood sugar should be checked before discharge.

Patients who are diagnosed to have GDM during pregnancy are subjected to oral glucose tolerance test with 75g glucose at 6 weeks.

2.4.6 Contraception:

Condoms. Intrauterine device safe

2.5. Care of Pre-Gestational Diabetes Mellitus

2.5.1 Before pregnancy:

To reduce the risk of fetal malformations and fetal macrosomia, **c**ounsel for planning pregnancy when glycated hemoglobin levels are normal. Advise pre conceptional Folic acid 5 mg daily 3 months prior to conception. Optimize BMI.

2.5.2 During pregnancy: Special investigations for detecting fetal malformations, as practicable.

First trimester biochemical screening (PAPP – A, hCG), USG for nuchal translucency (11-12 weeks).

Second trimester biochemical screen (16-18 weeks), USG (18-20 weeks)

2.5.3 Third trimester:

Assess fetal growth and liquor volume by USG. NST and umbilical artery Doppler studies to decide time and mode of delivery.

2.5.4 Delivery:

For uncomplicated pregnancy with well controlled Diabetes one can wait until 40 weeks. However, in view of adverse perinatal outcomes associated with poor control, induction of labour at 38 -39 weeks is a safe practice unless indicated earlier. Elective LSCS if foetal weight 4 kg.

3. Pregnancy with Cardiac Disease

Cardiac disease complicates around 1% of pregnancies. The disease worsens during pregnancy, delivery and postpartum period. It is one of the important indirect cause of maternal mortality.

Rheumatic valvular disease (Mitral stenosis, regurgitation) is common form in India. Currently congenital heart disease cases are being diagnosed early and surgically corrected. Such women are also presenting during pregnancy.

3.1. Risks

Maternal	Fetal/Neonatal
Cardiac failure, Acute pulmonary edema	Prematurity
Subacute bacterial endocarditis	IUGR leading to LBW
Thromboembolic complications	
Atrial fibrillation & other arrhythmias	

Table-4: Maternal, Fetal/Neonatal Risks in Cardiac Disease

3.2. Risk of failure

The cardiac patient faces greater risk of cardiac failure at each stage when there is significant rise in cardiac output. (E.g. at around 12-16 weeks, 28-32 weeks, during labour with each successive stage of labour, soon after delivery and first 24 hours after delivery).

The following obstetric and medical complications increase the risk of cardiac failure.

- Infection: Urinary, Respiratory, Dental.
- Anemia, Hypertension, Preeclampsia, Hyperthyroidism.

- Twins, polyhydramnios
- Tachycardia due to any cause: Physical exertion, emotional upset

3.3. Diagnosis

Normal pregnancy is associated with fatigue, dyspnea, decreased exercise capacity, peripheral edema, and jugular venous distention. Most pregnant women have audible physiologic systolic murmurs and a physiologic third heart sound (S3). This makes diagnosis of heart disease difficult during pregnancy.

NYHA Class	Symptoms
Ι	No symptoms on ordinary physical activity such as walking or climbing stairs.
II	Mild symptoms and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity such as walking short distances (20-100 m). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while at rest.

Table-5: New York Heart Association (NYHA) Functional Classification

3.4. Symptoms

- In grade 1 and 2 the woman may not have any symptoms.
- Grade 3: Breathlessness on accustomed exertion, fatigue, syncope, chest pain.
- Grade 4: Breathlessness even at rest, nocturnal cough, hemoptysis.

3.5. Signs

- Cardiac murmurs: Systolic grade 3 or more, any diastolic murmur.
- Abnormal heart sounds, Arrhythmias.
- Signs of cardiac failure: Progressive dyspnoea, Neck veins engorged, Palpable tender liver, HJR positive.
- Pitting oedema over feet.
- Crepitation at the lung bases.

3.6. Investigations

• ECG, 2D Echo Cardiography

3.7. Management:

3.7.1 Before conception

Women with preexisting cardiac lesions should receive preconception counseling regarding maternal and fetal risks during pregnancy and long-term maternal morbidity and mortality. Women with NYHA class III and IV face a high mortality and morbidity. These women should be strongly cautioned against pregnancy.

3.7.2 Care during pregnancy:

• Every pregnant woman should be completely examined between 4-6th month by the medical officer. Chest auscultation is a must during this

examination. Suspected cases must be referred to physician.

- Complete evaluation by physician/ cardiologist. Risk determination, counseling
- Watch for signs of cardiac failure.
- Care of teeth, treatment of urinary tract infection.
- Prevention or early correction of anemia.
- Monitoring blood pressure.
- Early detection and prompt treatment of pregnancy complications.

3.7.3 Hospitalization:

- Grade 3 & 4 dyspnoea kept in hospital throughout pregnancy.
- Women with obstetric or medical complications.
- Around 28-32 weeks depending on social circumstances.

3.7.4 Care during labour:

Await spontaneous labour. Induction only for obstetric indications.

a) First stage:

- Rest in propped up position; Avoid fluid overload.
- Prophylactic antibiotics depending upon risk of complications: Ampicillin, Gentamicin.
- Pain relief.
- Monitor pulse, respiratory rate, BP, temperature, lung bases.
- Monitor oxygen saturation; Oxygen as required.
- Keep cardiac drug tray ready.
- Monitor labour progress, fetal condition.

b) Second stage:

- Do not allow the woman to have strenuous bearing down effort.
- Cut short the 2nd stage by prophylactic vacuum/forceps delivery.

c) Third stage:

- Do not give Inj. Methyl ergometrine. Provide physiologic management.
- Give Inj. Oxytocin IM 10 IU if there is excessive bleeding.

d) During Postpartum period:

- Monitor vitals. Watch for signs of failure.
- Encourage ambulation to prevent thromboembolism.
- Watch for signs of puerperal sepsis and give antibiotics.
- Breast feeding is advisable.

3.7.5 Contraception:

- Combined Oral Contraceptive pills are contraindicated.
- Condom is safe.
- After completing family tubal ligation can be performed at district hospital or in tertiary care centre. Male partner may be encouraged to undergo vasectomy.
- MTP (if requested) can be performed at district hospital after stabilizing cardiac condition.

3.8. Peripartum Cardiomyopathy

This is an idiopathic dilated cardiomyopathy that develops in the last month of pregnancy or within 5 months of delivery and is characterized by left ventricular systolic dysfunction with ejection fraction (LVEF) < 45%.

3.8.1 Risk factors:

Advanced maternal age, Multiparity, multiple gestation, obesity, malnutrition, gestational hypertension, preeclampsia, cesarean section, family history, abuse of tobacco, alcohol, or cocaine.

The mortality rate is 25% to 50%, half of those die within the first month of presentation and the majority die within three months postpartum. Death results from progressive cardiac failure, thromboembolic events, and arrhythmias. Of the patients who survive, approximately 50% recover normal left heart function. Prognosis is related to left ventricular dysfunction at presentation.

3.8.2 Management

Medical management includes fluid and salt restriction, Digoxin, Diuretics, Vasodilators, and Anticoagulants. These women need to be in a tertiary care centre having cardiac ICU facility under care of cardiologist.

3.9 Pregnant Women receiving anticoagulation for surgically corrected heart disease

Women with surgically corrected heart disease with prosthetic valves are receiving Warfarin, a vitamin K antagonist orally for anticoagulation. Warfarin freely crosses the placenta and causes adverse fetal effects. The risk of Warfarin Embryopathy is highest when Warfarin is administered during 6 to 12 weeks of gestation. Heparin does not cross the placenta and is safe for the fetus.

- Anticoagulation: Warfarin must be Heparin discontinued and should be administered during this period. After 12 weeks, Heparin is stopped and Warfarin is restarted and continued until 36 weeks. After 36 weeks again LMWH or unfractionated Heparin is started which is stopped 4-6 hours before delivery and restarted 6 hours after delivery if there are no bleeding complications (24 hours after Caesarean Section).
- There is risk of profuse bleeding hence delivery should always take place in tertiary care institution. Protamine sulfate is antidote.
- These cases need regular laboratory monitoring to adjust the dose of medications. Also they might need fresh frozen plasma for bleeding hence they should be under care of specialists at the tertiary care centre during pregnancy and delivery.
- Breast feeding is allowed.
- Combined Oral Contraceptives are contraindicated.

4. Malaria during Pregnancy

Malaria during pregnancy is more common, more atypical, can be more serious and fatal.

4.1. Effects on Pregnancy Outcome

• The chances of spontaneous abortion, preterm birth, fetal growth retardation, fetal distress and still birth are increased.

• Rarely transplacental spread to the foetus resulting in congenital malaria.

4.2. Complications

Profound Anaemia may develop rapidly as there is a reduction in Haemoglobin. Anaemia can have serious consequences during labour. In the last two decades in India, an increasing proportion of P. falciparum infections is proving to be resistant to Chloroquine.

4.3. Clinical presentation

- Fever, Splenomegaly
- Anemia may be the presenting feature of Malaria; therefore, all cases of anemia should be tested for M.P.

• Fetal growth retardation.

4.4. Diagnosis

- Microscopy of stained thick & thin smear is the gold standard.
- Rapid diagnostic tests. All fevers particularly in Malaria endemic areas should be tested for Malaria.
- In endemic areas the pregnant woman should be routinely tested for Malaria at all antenatal visits by RDT even if she does not manifest any symptoms of Malaria.

Table-6: Treatment of Uncomplicated Malaria during Pregnancy

	Day	1	Day 2	Day3
P. Vivax	CQ 250 mg, (150 mg bas	se) 4 tablets	CQ 250 mg, 4 tablets	CQ 250 mg, 2 tablets
P. Falciparum				
First trimester	Quinine 10mg/kg 3 times daily.		Quinine 10mg/kg 3 times daily.	Quinine 10mg/kg 3 times daily Continue upto 7 days.
2 nd , 3 rd trimester	AS 200 mg 1 tablet SP 2 tablets (750 + 37.5mg each)		AS 200 mg 1 tablet	AS 200 mg 1 tablet
P. Vivax + P. Falciparum (Mixed)	AS 4 tablets of 50 mg 3 SP tablets (500mg + 25 mg each)		AS 4 tablets of 50 mg	AS 4 tablets of 50 mg

CQ: Chloroquine 250 mg tablet (150 mg base); 25 mg/kg body weight divided over three days i.e.10 mg/kg on day 1, 10 mg/kg on day 2 and 5 mg/kg on day 3. *AS:* Artesunate *SP:* Sulfadoxine + Pyrimethamine

4.5. Treatment

4.5.1 Vivax Malaria: Chloroquine for 3 days. Primaquine is contraindicated in pregnant women. To prevent the relapse, suppressive chemoprophylaxis with Chloroquine 300mg base, once a week until delivery.

4.5.2 P. Falciparum Malaria: As pregnant women having falciparum Malaria are more prone to develop complications, they should be referred to nearest FRU immediately.

- Induce hypoglycemia; do not give Quinine on empty stomach and tell the woman to eat regularly, while on Quinine treatment.
- ACT is not to be given in first trimester of pregnancy
- During 2nd & 3rd trimester: ACT+ SP

4.5.3 Mixed infections (P. vivax + P. falciparum) treated as falciparum Malaria

4.6 Prevention:

Use of personal protection measures including insecticide treated bed nets (ITN) / Long lasting insecticidal nets (LLIN) should be encouraged.

5. HIV Infected Pregnant Woman

Every pregnant woman during her first visit should be counseled for voluntary HIV testing at an ICTC to know her HIV status.

5.1. Care of HIV positive pregnant woman during pregnancy

5.1.1 Points to be noted in history:

- High risk behavior of herself and her spouse
- Pregnancy duration, number of previous pregnancies, live children, receipt of any antiretroviral medicines earlier.
- If pregnancy is unwanted and is < 20 weeks refer her to MTP Centre.
- If she wishes to continue current pregnancy, refer her to PPTCT Centre soon after confirmation of pregnancy to start the antiretroviral medicines for reducing the risk of vertical transmission. ART Centre will be initiating anti-retroviral medicines from 14 weeks and will be doing CD4 testing and other evaluations.
- Screen for TB and STI at every visit.

5.1.2 Specific education & counseling regarding:

- Nutritious diet, consumption of micronutrient supplements.
- Safe sex practices for preventing new STIs and other strains of HIV which could harm the baby.
- Adherence to HIV medicines.
- Follow up at ART & PPTCT centers as instructed.
- Institutional delivery.
- Safe infant feeding practices, infant care.

5.2 Care during delivery

Insist on institutional delivery at a PPTCT Centre as she requires ARV medicines during labour which need to be continued postpartum. C. section is performed only for obstetric indications. If she is in advanced labour, conduct delivery by following principles:

- Follow universal safety precautions
- Minimum number of vaginal examinations
- Do not rupture membranes unless indicated.
- Avoid: Routine Episiotomy, perineal injuries, vacuum delivery.
- Avoid suctioning of baby unless there is meconium staining
- Give ARV medicines as per protocol.
- Clean the baby of the maternal blood and body fluids before handing over to the relatives.

5.3 Care of mother after delivery

- Refer to PPTCT centre for care of mother and baby.
- Watch for signs of sepsis.
- Nutritious diet, nutrient supplements.
- Encourage to use reliable contraception; safe sex practices.

5.4 Treatment at PPTCT/ART Centre

- The new protocol consists of giving triple ART to pregnant woman and extended Nevirapine therapy for the exposed infant.
- Triple antiretroviral therapy (ART) initiated from 14 weeks irrespective of CD4 count and continued lifelong. Refer table below.
- If there is no prior exposure to EFV/NVP, triple ARV regime consisting of Tenofovir, Lamivudine and Efavirenz is started from 14 weeks onwards to be continued throughout pregnancy, labour, postpartum period and thereafter.
- If there is H/O prior exposure to NVP/EFV then EFV is not given and a protease inhibitor is given (Lopinavir/Ritonavir).
- Baseline investigations: Hb, urine, VDRL, screening for hepatitis B and C, ALT, CD4, urea/creatinine, blood sugar, lipid profile.
- If CD4 is < 250 Cotrimoxazole (CPT) 1 ds tab daily.

Laboratory tests:

- After 2 weeks of initiating ARV \rightarrow Hb, ALT.
- At 4,8,12 weeks \rightarrow Hb testing.
- Every 6 months → BUN, Sr. Creatinine, ALT, CD4, Urine analysis (imp in TDF based regimes).
- If on PI based treatment: BSL & Lipid profile every 6 months.

Name of ARV	Dose	Major Side Effects
Tenofovir (TDF)	300 mg Once a day	Nephrotoxicity, hypophosphatemia
Lamivudine (3 TC)	300mg Once a day	Very few. Hypersensitivity, rarely pancreatitis
Efavirenz (EFV)	600 mg Once a day	Neuropsychiatric symptoms (hallucinations, suicidal ideation, nightmare)
*Lopinavir/Ritonavir (LPV/r)	400/100 mg BD	GI disturbance, glucose intolerance, lipo-dystrophy & hyperlipidemia

 Table-7: Dosage Schedule and Associated Side Effects of ARV Medicines

* For prior exposure to EFV or NVP; LPV/r FDC tablet of LPV 200 mg/r 50 mg: 2 tablets BD

5.5 Care of HIV Exposed Infant:

- 5.5.1 Infant is given Nevirapine (NVP) syrup for 6 or 12 weeks as advised by PPTCT Centre depending on duration of ART received by mother.
- 5.5.2 Appropriate infant feeding is initiated as per informed choice following AFASS counseling: Upto 6 months: Exclusive breast feeding (preferred option) or exclusive replacement feeding. No mixed feeding.

5.5.3 At 6 weeks:

- Start Cotrimoxazole (CPT) prophylaxis,
- **Early infant diagnosis** (EID) by dry blood spot test (DBS); if DBS +ve Whole blood spot (WBS) confirmatory testing.
- EID +ve babies are started on ART at pediatric ART Centre irrespective of CD4

- 5.5.4 After 6 months, if breast feeding option has been chosen, breast feeding is continued as per EID status:
 - EID –ve babies: Continue BF upto 1 year + Complementary feeding; No abrupt stopping of breast feeding
 - EID +ve baby on ART: Breast feeding continued upto 2 years
- 5.5.5 Growth & nutrition monitoring, immunization as per schedule.
- 5.5.6 Confirm HIV status:

Repeat HIV testing at 6, 12 months and 6 weeks after stoppage of breast feeding; (If rapid test + ve DBS followed by WBS).

At 18 months: Confirmation of HIV status by 3 rapid antibody tests.

5.5.7 Counseling: Counsel for adherence to ARV and AKT if co-infected with tuberculosis. Encourage safe infant feeding practices.

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6. COMPREHENSIVE ABORTION CARE: MEDICAL TERMINATION OF PREGNANCY

Unsafe abortion still accounts for nearly 9 % maternal deaths. Provision of easily accessible, free safe abortion service can reduce the burden of maternal morbidity and mortality.

1. Legal limit

Upto 20 weeks of pregnancy. A trained medical officer at Primary Health Center is authorized to perform MTPs up to 12 weeks.

2. Indications

- Pregnancy likely to endanger woman's life or likely to cause grave Injury to her physical or mental health.
- Child is likely to suffer from serious physical or mental disability.
- Pregnancy caused by rape.
- Pregnancy following contraceptive failure.

3. Consent for MTP

An adult woman who is not mentally ill can undergo MTP with only her own consent. Consent of legal guardian is required for minors and mentally ill women.

4. Record and register

- Consent in form C of the MTP Act.
- Certify MTP within 3 hours on form I specifying the indication.
- Admission register (Form III). This register is a secret document.

5. Clinical evaluation

Medical history, date of LMP, complete physical examination. Determine size of uterus on vaginal examination.

6. Investigations

- Hemoglobin, blood group, Rh typing, urine testing for sugar and protein.
- Ultrasonography is not mandatory. It may be helpful for knowing the pregnancy duration in women having irregular cycles, Lactational amenorrhea, when on examination the uterine size is uncertain or discrepant with the period of amenorrhea and to exclude an ectopic gestation.

7. Methods for first trimester MTP

7.1 Surgical methods

- Manual vacuum aspiration (MVA) is the safe recommended technique for pregnancy up to 12 weeks.
- Electrical vacuum aspiration is also safe and effective.
- D&C should not be used as a method for MTP.
- Give antibiotics prophylaxis: Cap. Doxycycline 100 mg orally twice a day for seven days may be given.
- Follow adequate infection control practices.

a) Before MVA:

- Check the eligibility and consent
- Give pain relief
- Start prophylactic antibiotics
- If uterus is > 9 weeks of size, cervical softening can be achieved by administering 400 mcg (2 tablets) of Misoprostol orally/vaginally 3-4 hours before MVA.
- Keep the charged syringe ready, perform the MVA procedure.

b) Complications:

- Incomplete abortion, hemorrhage, infection,
- Uterine perforation
- Anesthesia related complications.
- Failed termination with continuation of pregnancy. Woman should be counseled to report if she continues to be amenorrhoeic after MTP. Repeat termination of pregnancy can be undertaken.

7.2. Mifepristone-Misoprostol abortion (MMA)

MMA is permitted up to 49 days by MTP Act.

a) Information to Women:

MMA is safe and effective. Success rate 95-98 %

- Three visits are necessary, day 1 Mifepristone, day 3 Misoprostol tablets and day 14 for post treatment evaluation
- If treatment fails, surgical termination is necessary; pregnancy cannot be continued, as there is risk of fetal malformation.
- Vaginal bleeding and uterine cramping may occur for 7-14 days.

b) Protocol

Table-1: Protocols for Mifepristone-Misoprostol Abortion

	Mifepristone		Misoprostol		
	Day 1		Day 3		
Gest Age	Dose	Route	Dose	Route	
Upto 49 days	200 mg (1 tab)	oral	400 mcg (200 mcg, 2 tablets)	Oral/vaginal	

The clinic where medical abortion drugs are prescribed by an RMP as under the MTP Act, does not need site approval as long as it has referral linkage to an MTP approved site. The clinic should display a certificate to this effect from the owner of the approved place.

The Central Drugs Standard Control Organization, Director general of health services, Govt. of India has approved a Combi pack of one 200 mg tablet of mifepristone and four 200 mcg tablets of Misoprostol for MTP for gestation between 49-63 days. However, MTP Act allows MMA up to 7 weeks

In the event that vomiting ensues within 30 minutes of oral Misoprostol the medication should be repeated. There is no need to keep a woman in the facility till she aborts.

Misoprostol given by vaginal route has lesser side effects, is absorbed slowly and is effective for a longer time whereas by oral route it is absorbed quickly, is effective for a shorter time and leads to more gastrointestinal side effects.

c) Contraindications:

- Ectopic pregnancy, either confirmed or suspected, or undiagnosed adnexal mass
- Hemorrhagic disorder.
- Allergy to mifepristone, Misoprostol or another prostaglandin.
- Current anticoagulant therapy, Current use of long-term systemic corticosteroids.
- Intrauterine device in place (remove before giving Mifepristone).
- Chronic adrenal failure. Inherited porphyria.
- Caution: Haemoglobin<8 gm %.
- Uncontrolled hypertension, BP > 160/100
- Uncontrolled seizure disorder.

d) Contraception after MMA:

- OC pills or Inj. DMPA can be started on day 3 or day 15 when abortion process appears to be complete.
- IUCD insertion after one normal menstrual period.
- Tubal ligation after next cycle.

e) Counseling:

Early abortion is safer. MTPs should not be performed repeatedly. Using contraception is important. Concomitant female sterilization or IUCD insertion at the time of surgical abortion is safe. Inform the woman to report early for symptoms suggestive of complications.

8. Methods for Second Trimester MTPs

a) Mifepristone – Misoprostol:

These drugs are recommended by WHO for second trimester MTPs. Combination improves the efficacy.

The suggested protocol is 200 mg mifepristone followed after 36 - 48 hours by 400 micrograms of vaginal, sublingual or oral, Misoprostol every 3 - 6

hours up to 5 doses. Before 18 weeks 3-4 hourly interval is preferred. At or after 18 weeks consider 6 hourly dosing interval.

Vaginal Misoprostol is more effective than oral

Cautious use in cases having scarred uterus. Check and record vital signs 4 hourly until onset of strong contractions, thereafter 2 hourly.

b) Use of Misoprostol alone

Is less effective as compared to combination

400 mcg 3-6 hourly upto 5 doses.

If fetal expulsion does not occur after 24 hours from initial dose perform abdominal examination and USG to rule out uterine rupture.

c) Extra amniotic Instillation of Ethacridine Lactate, 0.1 %, 150 ml

Safe method, however the drug is not currently available.

d) Abdominal Hysterotomy:

Major surgery, done only in selected cases. Eg. Cancer cervix.

Second trimesters MTP procedures require hospitalization for few days. They are likely to be associated with more bleeding, more chances of incomplete abortion and other complications. Provider needs to make sure that the woman is not seeking a sex selective abortion.

Additional legal requirements: MTP Centre approved for 2nd trimester MTP, experienced RMP as stated in MTP Act, and the requirement of opinions of two registered medical practitioners are necessary.

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7. COMMON GYNAECOLOGICAL PROBLEMS

1. Dysmenorrhoea 1.1 Definition:

Dysmenorrhoea is painful menstruation.

1.2 Types:

Spasmodic (Primary) and Congestive (Secondary).

1.2.1 Spasmodic Dysmenorrhoea

a) Symptoms: Colicky pain in lower abdomen starts on first day of menstruation and usually lasts for a day. Nausea, vomiting may occur.

b) Treatment:

- Usually reassurance is adequate.
- Majority get relief with antispasmodics (Dicyclomine) or antiprostaglandins like Mefenamic acid 500 mg t.d.s as needed.
- If no response: Combined oral contraceptive pills for 3 cycles.
- Rarely pain may be very severe not responding to medicines and may need further evaluation and management by specialist.

c) Investigations: If pain is persistent, USG to rule out Endometriosis, Uterine anomalies, or Submucous myoma.

1.2.2 Congestive Dysmenorrhoea:

a) Common causes: Chronic Pelvic inflammatory disease (PID), endometriosis, Genital Tuberculosis, Pelvic adhesions.

b) Symptoms: Chronic pelvic pain. Pain is experienced few days before period and is usually relieved with the onset of menstrual bleeding. It is continuous dull aching pain in lower abdomen and low backache. Often associated with dyspareunia, vaginal discharge, menorrhagia and infertility.

c) Signs of chronic PID:

- P/A: Tenderness in lower abdomen
- P/S: Discharge from cervical canal in STIs
- P/V: Retroverted fixed uterus, cervical movements painful. Tenderness in fornices and tender adnexal mass. Nodularity in posterior fornix in endometriosis.

d) Investigations:

- Ultrasonography to detect pelvic pathology (adnexal mass, ovarian cyst).
- Diagnostic laparoscopy helps in diagnosis of genital TB, endometriosis, adhesions.

e) Treatment:

- Assurance and treatment of cause.
- NSAIDs for pain relief: Mefenamic acid, Ibuprofen.
- Antibiotics for treatment of PID.
- Progestins for Endometriosis.
- Specialist referral for treatment of Endometriosis, genital TB.
- Laparoscopy for diagnosis of cause for chronic pelvic pain and treatment of adhesions, endometriosis.

2. Abnormal Uterine Bleeding

Abnormal uterine bleeding (AUB) accounts for 20% of all gynecologic visits.

2.1. Definitions

- **Menorrhagia**: Excessive uterine bleeding occurring at regular intervals or prolonged uterine bleeding lasting > 7 days.
- **Dysfunctional uterine bleeding (DUB):** Abnormal uterine bleeding occurring in the absence of identifiable pathology. It can be ovulatory or anovulatory DUB.
- **Metrorrhagia:** Acyclical intermenstrual bleeding occurring at irregular intervals.
- **Breakthrough bleeding:** Unpredictable bleeding that occurs while on exogenous hormones (eg hormonal contraception).
- **Oligomenorrhea:** A menstrual cycle interval of more than 35 days.
- **Polymenorrhea:** A menstrual cycle interval of less than 21 days.
- **Postmenopausal bleeding**: Vaginal bleeding occurring more than 12 months after the last menstrual period.
- Amenorrhea: Absence of menstrual bleeding.

2.2. Differential diagnosis of AUB

Abnormal uterine bleeding can occur in all age groups. The diagnostic testing and treatment approach should take into consideration the age group and fertility choices of the woman.

- Adolescent girls: Anovular DUB is common. Pregnancy related conditions should be kept in mind. Bleeding disorders can be present in some cases.
- Women between 20-40 years: Pregnancy related conditions, ovular DUB, pelvic infection and benign neoplasia like fibroids are common.
- Perimenopausal AUB is commonly due to anovulation and fibroids. However, it is necessary to exclude polyps, endometrial hyperplasia and malignancies.
- Postmenopausal bleeding: Malignancy needs to be excluded.

2.2.1. History

- Detailed menstrual history, sexual history, past medical history, gynecologic and obstetric history. Note any change in the patient's weight, and exercise pattern.
- History of bleeding disorders, possible pregnancy.
- H/O Drugs, IUCD.
- Antipsychotic medications (Dopamine antagonists), Phenothiazine and antidepressants can cause hyperprolactinemia and AUB.
- Combination OC pills can cause breakthrough bleeding.
- Prolonged use of high doses of progestational agents.
- Anticoagulant medications.
- Digitalis, Phenytoin, and Corticosteroids.
- Copper- intrauterine devices.

2.2.2. Physical Exam

- Note pallor, thyroid examination.
- Breast examination: Galactorrhea.
- P/A examination: Pelvic mass (fibroids), tenderness in lower abdomen. (PID)
- Speculum examination: Cervical pathology. (growth, ulcer, polyp)
- P/V: Cervical motion tenderness, size and contour of uterus, and presence of any palpable adnexal masses or tenderness.

2.2.3. Investigations

- Complete blood count
- Ultrasonography abdomen and pelvis and transvaginal ultrasonography (TVUS) for diagnosing fibroids, polyps, adnexal mass. Thickness of endometrium can be measured. Intrauterine pregnancy and ectopic pregnancy can be excluded.
- Endometrial biopsy in women over age 35 and women having risk factors for endometrial cancer. It helps in diagnosing ovular or anovular bleeding, endometrial hyperplasia, endometrial precancerous lesions and endometrial carcinoma.
- Endometrial sampling is a rapid, safe, and costeffective procedure that can be performed in the office to evaluate AUB. A potential drawback is that the biopsy does not sample the entire endometrium and a localized lesion may be missed.
- Dilation and Curettage (D&C) can be both diagnostic and therapeutic.
- Hysteroscopy is the gold standard for evaluating the endometrial cavity. It provides direct visualization of the endometrial cavity and can be performed in the office setting or operating room. It can be both diagnostic and therapeutic, allowing for directed biopsies and excision of polyps and small sub mucus myomas.
- Saline infusion Sonohysterography for detecting endometrial polyps or uterine leiomyoma.
- Thyroid stimulating hormone (TSH) and Prolactin estimation: Hypothyroidism and hyperprolactinemia can lead to anovulation and DUB.
- Bleeding disorders should be evaluated if historical or clinical features suggest specific conditions.

2.3. Dysfunctional Uterine Bleeding

- Dysfunctional uterine bleeding (DUB) is a diagnosis by exclusion of other causes of AUB without a demonstrable pathologic cause. The predominant causes of DUB are anovulation or oligo ovulation.
- Long-term anovulation, polycystic ovary syndrome: Resulting unopposed estrogen state increases the risk of endometrial hyperplasia.

Morbid obesity with associated elevated estrogen levels can also cause DUB.

• In some cases, DUB may be associated with ovulatory cycles.

2.3.1 Medical therapy:

Treatment of Anemia should be initiated. Women who are severely anemic or not responding to medical treatment should be referred to specialist.

a) Nonsteroidal anti-inflammatory drugs (NSAIDs) may reduce menstrual blood loss by about 30% in women with menorrhagia and can be given as the first line therapy. They need to be taken during menses only. The options are:

- Mefenamic acid 500 mg three times a day for 5 days during menstruation.
- Ibuprofen 600-1200 mg /day.
- Naproxen sodium 500 mg × 1, then 250 mg every 6-8 hourly.

b) Hormones:

• Oral Progestins: Useful in anovulatory bleeding.

For acute heavy bleeding: Medroxyprogesterone acetate 10 mg tablet 2-3 times a day till bleeding stops. It is tapered and continued 1 tab daily for a total duration of 21 days. Withdrawal bleeding starts in few days. Further cyclical therapy (10 mg daily) is given from day 5 to 25th day for 2 cycles. Response more is awaited. Norethisterone tablet 5 mg can be used similarly for a period of 21 days, initially 1 tab 3 times a day until bleeding stops, then 1 tab twice a day for few days and then tapered to 1 tab daily thereafter. Side effects of Progestins include breast tenderness, weight gain, and headaches.

• Combined oral contraceptive pills can be given from day 5 to 25 for 3-6 cycles in women between 20-40 years of age not having contraindications for estrogens and who also wish to have contraception.

c) Antifibrinolytic medications (e.g. Tranexamic acid) decrease menstrual blood flow by 50%, and need to be taken during menses only. However, there are concerns about their pro-thrombotic potential.

2.3.2 Surgical Treatment for DUB:

• Diagnostic D&C may have some therapeutic effect. However, it does not have a sustained therapeutic effect.

- Endometrial ablation: Endometrial ablation is not recommended in women who desire future fertility.
- Hysterectomy provides definitive treatment for menorrhagia and may be a reasonable option in women with severe menorrhagia, who have completed their childbearing and are refractory to medical and less invasive surgical therapy.

The women not responding to medical line of treatment should be referred to specialist at FRU/DH for surgical treatment.

2.4 AUB: Other Conditions

- **i.** General medical causes associated with menorrhagia: Hypertension, cardiac failure, hepatic dysfunction can be associated with AUB.
- **ii. Genital Infection:** Chronic PID and genital tuberculosis.

iii. Endometrial Pathology & Genital Neoplasia:

a. Endometrial Polyps can present as Menorrhagia or Metrorrhagia. Diagnosis is by saline infusion sonogram and hysteroscopy. Endometrial polyps can be removed by operative hysteroscopy.

Cervical polyps can be removed by grasping with forceps, twisting them off, and cauterizing the base.

b. Endometrial Hyperplasia: A precursor to endometrial carcinoma. It is classified into simple or complex, based on architectural features, and typical or atypical, based on cytological features. Endometrial hyperplasia tends to occur during periods of long-term unopposed estrogen exposure, either secondary to anovulatory cycles or exogenous use. AUB is the most common presenting symptom.

- Diagnosis by endometrial biopsy
- Treatment depends upon age, desire for future fertility and presence of atypia in the pathology specimen.
- Simple hyperplasia can be treated with progestogens.
- Cyclic Medroxyprogesterone Acetate (MPA 10 mg/day for 12 to 14 days/cycle for 3 to 6 months) in young anovulatory women to induce monthly withdrawal bleeding.
- Local Progesterone administration: Levonorgestrel-releasing intrauterine system. (LNG IUS)
- Atypical endometrial hyperplasia is more likely to progress to carcinoma or may coexist with

endometrial carcinoma. Hence hysterectomy is advised.

- c) Uterine Fibroids: Menorrhagia is the most common presenting symptom. Diagnosis is by USG. Symptomatic fibroids need surgical treatment by a specialist.
- d) Endometrial Cancer: It is mostly seen in postmenopausal women and is rare in patients younger than 40. Postmenopausal bleeding is the presenting symptom which should be assumed to represent endometrial cancer until proven otherwise. The women should be referred to specialist for further evaluation and treatment.
- e) Cervical Cancer: Bleeding patterns associated with cervical carcinoma are intermenstrual and post-coital bleeding. Cervical biopsy is diagnostic. The woman should be referred early to district hospital or a cancer treatment centre.

f) Ovarian Cancer: Estrogen-producing ovarian tumors, can produce endometrial hyperplasia and AUB. USG will reveal the tumor. The woman should be referred to district hospital or a cancer treatment centre for surgical treatment.

3. Postmenopausal Bleeding

3.1 Definition:

Vaginal bleeding occurring one year after cessation of menses.

Uterine bleeding is often due to endometrial cancer, polyps or endometrial hyperplasia. Genital tract malignancy and precancerous conditions need to be excluded in these cases. Hence immediate referral to gynecologist is necessary for further evaluation and treatment.

3.2 Differential diagnosis:

Table-1:	Differential	Diagnosis	of Postmeno	nausal Vagina	l Bleeding.
I able I.	Differential	Diagnosis	of i obtilition	pausai ragina	Diccums

 Endometrial causes Endometrial Hyperplasia Endometrial carcinoma DUB Endometrial polyp Senile endometritis TB endometritis 	 Ovarian causes Benign tumors of ovary Malignant tumors of ovary Hormone secreting tumors of Ovary (Granulosa- theca cell tumor) 		
 Cervical causes: Cancer cervix, Polyp, Decubitus ulcer on prolapse Cervicitis 	 Exogenous Estrogens – HRT for menopausal symptoms can cause irregular bleeding 		
 Vulvovaginal causes: Senile atrophic vaginitis Foreign body in vagina like pessary Malignant lesions on vulva 	 Non genital causes: Hypertension Bleeding diathesis Urethral and rectal bleeding can sometimes be misreported as vaginal bleeding 		

3.2.1 Endometrial Cancer

a) Risk Factors for Endometrial Cancer:

• Conditions associated with excessive estrogens unopposed by cyclical Progesterone are commonly responsible for postmenopausal bleeding. Estrogens could have been produced in the body (endogenous) or given in medicinal form (exogenous)

- Age at menopause > 52 years
- Nulliparity, infertility.
- Obesity, Hypertension, Diabetes Mellitus
- Family history of uterine, breast, colorectal cancer
- Drugs: Estrogen therapy, Tamoxifen

b) Management:

At PHC:

History:

- Ask symptoms: Irregular bleeding, spotting, brown discharge, post-coital bleeding, number of bleeding episodes
- Age of woman, late menopause (> 52 years), Nulliparity,
- H/O receiving hormones (estrogen /progestogen), drugs (tamoxifen)
- Family history of endometrial cancer
- Diabetes Mellitus, Hypertension

Examination:

- Look for Obesity, Hypertension
- Examination of vulva for any lesion, ulcers, growth, atrophy, polyp.
- P/S examination of vagina and cervix to look for any growth, polyp, ulcer, estrogenization, infection. If cervical pathology is seen refer to gynecologist for cervical biopsy, polypectomy as necessary
- Vaginal examination: Look for uterine enlargement, Suspect Pvometra if soft uniformly enlarged tender uterus with intermittent offensive vaginal discharge. Suspect uterine myoma or malignancy if firm irregularly enlarged uterus
- Refer the woman to the gynecologist for further evaluation.
- Refer for USG including TVS for assessment of endometrial thickness and other uterine and ovarian pathology.

c. Management

At RH/DH:

- If gross cervical pathology seen cervical biopsy, Polypectomy
- Pap smear if no gross lesion seen
- Colposcopy for unhealthy cervix, abnormal Pap smear, vulval lesions
- Ultrasonography: TVS or Saline infusion sonography
- Hysteroscopy and directed biopsy is the gold standard for evaluation: It permits direct visual inspection of uterine cavity with greater accuracy of diagnosis as focal lesions are not missed. It is superior to blind dilatation and curettage in which neoplasia may be missed.

- Fractional curettage under GA if hysteroscope unavailable
- Treatment is planned according to pathology found.
 - Endometrial hyperplasia: Abdominal total hysterectomy with bilateral salpingo oophorectomy
 - Cervical / Endometrial malignancy: Treatment as per stage
 - Ovarian tumors: Staging laparotomy (Total hysterectomy + bilateral salpingooophorectomy for benign tumors. Treatment as per stage for malignant tumors)
 - Senile vaginitis: Vaginal estrogen therapy

d. Screening for endometrial cancer:

TVS for endometrial thickness is a simple, noninvasive, reliable screening technique for the diagnosis of Ca Endometrium. All postmenopausal women should have annual trans vaginal sonographic assessment for endometrial thickness. Women having endometrial thickness > 4 mm need further evaluation to rule out endometrial cancer.

3.2.2 Cervical Cancer

Cervical cancer is the second most common cancer among women in India, the first being breast cancer. In 2012, about 1,23,000 new cases of cancer cervix were detected in India and 67,000 women died due to cervical cancer, but **this cancer can be prevented**. Human papillomavirus (HPV) is the causal agent for cervical cancer and about 15 high-risk types of HPV are known. HPV infection is a very common infection. Those women who cannot clear this infection by their natural immunity are at risk of cervical cancer.

a. Screening for Cervical Cancer:

Cervical cancer is preceded by a long phase of premalignant lesion called as Cervical Intraepithelial Neoplasia (CIN). This stage may last for about 10-20 years before developing into a frank malignancy. When the abnormal cells are limited to the lower third of the epithelium it is labeled as CIN 1, when lower two thirds of the epithelium are involved, it is termed as CIN II and when the entire thickness of the epithelium is involved, it is termed as CIN III. CIN can be diagnosed by a pathologist on histopathological examination of cervical biopsy specimen. CIN lesions can be treated by simple treatment modalities and cervical cancer can be prevented.

The World Health Organization (WHO) has recommended that at a minimum, as a priority, screening is recommended for every woman 30–49 years of age at least once in a life time. Screening should be initiated at 30 years of age. For HIV-infected women screening should be initiated as soon as the infection is diagnosed.

Cervical cancer screening can be done by following different tests:

- Pap smear (cytology screening)
- Visual inspection of cervix with acetic acid (VIA)
- HPV testing for high risk types (it is expensive and available only in some centers. Useful in increasing the screening interval in women above 30 years of age)

i) Pap smear (cytology screening):

Pap smear requires laboratory infrastructure, quality control, a system for informing the results to the women and further referral for colposcopy.

Material: Cusco's bivalve speculum, Ayre's spatula, endo-brush, fixative spray or jar containing fixative



Figure 7.1: Scraping from Ectocervix

ii) Visual inspection of cervix with acetic acid:

VIA involves application of 5% acetic acid on the cervix for one minute and visual inspection of colour changes in the cervix. VIA is simple, cheap, it can be performed by trained health care workers and provides an opportunity to treat screen positive women during the same sitting thus reducing the challenges of lost to follow up.

Materials and equipment needed for screening:

- Sterile gloves and Cusco's speculum
- Autoclaved cotton balls and sponge holding forceps/ autoclaved cotton-tipped swabs
- Freshly prepared acetic acid solution (5%)

solution, 95% ethanol or ether-alcohol mixture, glass slides.

Procedure:

- Schedule the procedure following abstinence for 24 hours and when there is no vaginal bleeding. Confirm that no vaginal examination has been done in previous 24 hours. No vaginal antiseptics should be used
- Use speculum to inspect the cervix
- Do not take smear if there is vaginal bleeding or active infection
- Place the Ayre's spatula against the cervix and rotate it firmly against the cervix through 360 degrees to scrape the transformation zone. Spread the sample on the spatula evenly on a glass slide. Endocervical cells are obtained by endo-brush or cotton tipped applicator rolled in cervical canal. Place the sample on another glass slide (Fig 1 & 2)
- Fix the smears by putting in fixative jar or by a fixative spray. Send the smears to an expert cytopathologist
- Currently Bethesda system (2001) is used for reporting of smears.



Figure 7.2: Endocervical sampling

Procedure:

- Insert a bivalve speculum, view the cervix
- Remove any discharge, blood or mucus from the cervix by a saline moistened cotton swab
- Identify the squamocolumnar junction (SCJ) and the area around it
- Apply acetic acid to the cervix for one minute and simultaneously observe if any color change develops
- Observe any changes in the color/ appearance of the cervix after one minute. Give special attention to abnormalities close to the SCJ
- Look for any raised and thickened white plaques or acetowhite epithelium.

• Use a fresh swab to remove any remaining acetic acid solution from the cervix and vagina. Gently remove the speculum.

The test results are reported as follows:

- VIA positive (Fig 3) when there is a dense white, opaque lesion touching the squamocolumnar junction (SCJ)
- VIA negative when such lesion is not seen
- VIA positive-invasive cancer when there is a clinically visible growth which turns dense acetowhite and bleeds on touch.



Figure 7.3: VIA positive cervix

After screening

- If the test is negative, repeat test in 3-5 years.
- For HIV infected woman repeat testing within 3 years.

b) Treatment strategy for screen positive cases:

GOI-WHO (2006): Screening women between age 30 -59, by VIA by trained HCW at PHC. Referral of VIA positive to district hospital (DH).

At DH repeat VIA followed by pap smear, colposcopy, directed biopsy from abnormal areas followed by cryotherapy /LEEP as required at the same sitting.

WHO guidelines (2013):

"Screen and Treat" approach, using a screening test giving immediate results (like VIA) followed by "on the spot" treatment (cryotherapy) of detected lesions to eligible women, without any further tests, when there is no suspicion of cancer. This has been shown to be an effective strategy. This is a single visit approach which is convenient for rural women. **Primary prevention of cervical cancer** is now possible by HPV vaccination of girls between 9 to 13 years of age before initiation of sexual activity. But HPV vaccines are currently expensive and not yet available in the government programs.

A comprehensive approach to cervical cancer prevention and control involves health education and screening of all women and treatment before progression to invasive disease. Awareness among women and doctors that cervical cancer can be prevented by early detection and treatment of CIN is critical for the success of cervical screening.

4. Infertility

4.1 Definition:

Infertility is defined as failure to conceive after one year of regular, unprotected sexual intercourse. It can be primary or secondary:

Primary: The woman has never conceived in past.

Secondary: The woman has previously conceived but is subsequently unable to conceive for 12 months.

Infertility affects 10 - 15% of the general population. It is the problem of the couple. Male factor is responsible in 30%, female factor in 50%, and the combined factors are responsible in 10%. Unexplained infertility is observed in 10%.

4.1.1 Infertility – Male Factor

a) History in Male Partner: Age, occupation, previous marriage if any, substance abuse, coital history (Frequency of intercourse / Knowledge of fertile period / Coital difficulties related to erection / penetration / ejaculation).

Past history:

Mumps/Genital trauma/STIs/Surgery/Tuberculosis, Current history: Any illness, drug treatment.

b) Physical Examination - Rule out Hernia, Hydrocele, Undescended testis, Absence of Testis. Note the size of testis. Look for Varicocele in upright position.

c) Investigations:

Semen Analysis: Sample collected after 2-3 days of abstinence and examined within 2 hours of collection. Male partner should be evaluated first by performing semen analysis. In humans it takes about 75 days for spermatogonia to develop into mature spermatozoa.

Blood and urine routine testing

Table 2: Minimum normal criteria for semenanalysis: WHO revised criteria.4

Parameter	Lower Ref Limit	
Volume	1.5 ml	
Sperm concentration	15 million/ml	
Progressive motility	32 %	
Normal forms	4 %	

If semen report is abnormal, the test should be repeated twice at least at 2-4 weeks of interval before considering it as abnormal. Ideally, a period of 3 months should be considered for evaluation of any improvement as a result of therapy.

Persistently abnormal semen parameters require further evaluation at specialty infertility clinic

d) Counselling for male:

Regular intercourse 2-3 times/week, stop smoking, alcohol & any addictive drugs, wear loose fitting underwear and trousers, and avoid occupational or social situations that might cause testicular heating. Treat any psycho-sexual problem if present.

e) Male Partner- Treatment

- i. **Medical therapy:** Antioxidants, clomiphene citrate, gonadotropins.
- ii. **Intra-uterine Insemination (IUI):** Mild male factor infertility.
- iii. **Donor insemination**: For azoospermia (Primary testicular failure)
- iv. Adoption: For cases with recurrent unexplained failed IVF cycles.
- v. **Surgical restoration of duct patency**: For obstructive azoospermia (previous vasectomy).
- vi. Intra-cytoplasmic Sperm Injection (ICSI): For severe male factor or for recurrent unexplained failed IVF cycles.

4.1.2 Infertility – Female Factors

a) Causes

Anovulatory infertility-

- i. Polycystic ovarian disease.
- ii. Thyroid disorders, Hyperprolactinemia
- iii. Decreased ovarian reserve, Premature ovarian failure.
- iv. Anorexia nervosa / Stress related / Exercise induced.

Tubal infertility:

Genital Tuberculosis, PID, Tubectomy

Uterine factors:

Submucous fibroids, Tubercular endometritis, Endometrial polyps, Intrauterine adhesions.

Peritoneal factors: Endometriosis, Chronic PID

Cervical factors:

Immunologic, infections, surgical trauma (amputation, conization, deep cauterization of cervix).

b) History in female Partner:

Age, years since marriage, occupation, substance abuse. History suggestive of reproductive tract (Vaginal discharge/Pruritus infections pelvic vulvae/Chronic pain), Tuberculosis, Endocrinopathy (Weight & hair changes/discharge from nipples/cold intolerance/dryness of skin), Premature ovarian failure (H/O Mumps, hot flushes/Oligomenorrhea/amenorrhea), Abdominal Surgery. coital frequency & knowledge of fertile period, dyspareunia

H/O previous pregnancies, post-partum infection.

Family history: e.g. Tuberculosis

Menstrual history: Menarche, regularity of cycles, Dysmenorrhoea, flow, LMP: Regular cycles, Dysmenorrhoea, Mittelschmerz (mid cycle pain), premenstrual breast heaviness and mastalgia are **suggestive of ovulatory cycles.**

c) Examination of female partner:

Look for obesity or gross underweight (BMI), Lymphadenopathy, Thyroid enlargement, Hirsutism, Galactorrhea, Acanthosis Nigricans.

Systemic exam

Speculum & per vaginal exam: Look for anatomic abnormalities.

d) Investigation in Female Partner

- Pelvic USG: Diagnosis of PCOS, uterine fibroids, adnexal mass, uterine malformations, endometrioma.
- Serial trans-vaginal sonography for confirmation of ovulation and timing of ovulation.
- Hormonal assay: Mid luteal serum progesterone.

- TSH, Prolactin, FSH & LH in selected patients with anovulation
- •
- Endometrial sample collected by premenstrual curettage is sent for histopathological and microbiological evaluation to exclude endometrial tuberculosis in suspected cases. Laparoscopy helps in diagnosis.
- **Hysterosalpingography** (**HSG**)- Tubal patency test. It also helps in detecting intra uterine pathology. Performed between Day 6 Day 11 of the menstrual cycle, at the Radio- diagnosis department.

Contraindications: Current PID, suspected Genital TB, cervicitis.

• Laparoscopy with chromotubation -

This is an invasive surgical procedure indicated when HSG is abnormal, there is failure to conceive within 6 months in spite of a normal HSG, and for suspicion of endometriosis. Usually done in the pre ovulatory period. Diagnostic laparoscopy helps in diagnosing tubal, uterine pathology and also helps in detecting Endometriosis, Pelvic adhesions, Pelvic Tuberculosis. Surgical treatment for adhesions, Endometriosis can be done laparoscopically.

• Hysteroscopy:

Endoscopic direct visualization of the uterine cavity. Submucous fibroids, Endometrial polyps, adhesions, Malformations and Foreign bodies can be visualized.

Hysteroscopic resection of uterine septum, submucous myoma and adhesiolysis for intrauterine adhesions can be offered. Laparoscopy can be combined with hysteroscopy.

e) Counselling for Female Partner:

Regular intercourse 2-3 times/week. stop smoking, alcohol and addictive drugs, and follow a supervised weight loss programme if obese. Treat psycho-sexual problem if present.

f) Treatment of Female Partner:

Induction of Ovulation:

For women with ovulatory dysfunction.

- Clomiphene citrate is the first line drug for ovulation induction.
 50 mg orally daily for 5 days from day 2 to day 6 of menstrual cycle. Ovulation is monitored by USG follicle monitoring. If ovulation does not occur, the dose can be increased by 50 mg daily in successive cycles up to 150 mg daily. Risk of multiple pregnancies should be explained. Failure of ovulation or conception after treatment for 3 cycles requires careful review and evaluation.
- Women with polycystic ovary syndrome who have not responded to clomiphene citrate should be offered laparoscopic ovarian drilling
- Gonadotropins for ovulation induction requires special expertise and careful monitoring. There is risk of multiple pregnancy
- Hyperprolactinemia: Treatment with bromocriptine.
- Hypothyroidism: Thyroxine therapy restores ovulatory cycles

Intra-uterine Insemination (IUI): Unexplained infertility and cases with minimal endometriosis.

Surgical Treatment

- i. Laparoscopic adhesiolysis
- ii. Hysteroscopic tubal cannulation, resection of intrauterine adhesions, polyp or submucous myoma.
- iii. Laparotomy for tubal reconstructive surgery.

5. Pelvic Organ Prolapse

Pelvic organ prolapse (POP) is a common gynaecological problem in women above the age of 40 years.

POP includes anterior vaginal prolapse (cystocele), uterine prolapse, and posterior vaginal prolapse (rectocele, enterocele).

5.1 Symptoms

Lower urinary tract symptoms	Bowel symptoms	Sexual symptoms	Other symptoms
Incomplete emptying, recurrent UTI, frequency, urgency, nocturia	Constipation	Interference with sexual activity	Pelvic pressure, heaviness, pain
Stress urinary incontinence	Straining	Dyspareunia	Presence of vaginal bulge/mass
Voiding difficulty: may require to reduce prolapse before passing urine	Incomplete evacuation	Decreased sexual desire	Low back pain

Table-3: Symptoms

5.2. Signs

- P/A: Look for any lump, ascites, obesity
- Local exam: Inspection of the vulva with coughing and straining demonstrate severe prolapse and may demonstrate stress incontinence (provided the bladder is full)
- Examination using Sims speculum: Note type of prolapse, degree of uterine descent, anterior and posterior vaginal wall prolapse. Look for decubitus ulcer on cervix, elongation of cervix
- Vaginal examination: Note uterine size, position, adnexal mass
- Rectal examination, to differentiate rectocele from enterocele
- Assess anal sphincter tone, assess levator tone, perineal body

5.3. Predisposing factors

- Injuries to supports of pelvic organs during childbirth
- Obesity, chronic cough, chronic constipation,
- Atrophy and loss of tone due to aging, estrogen deficiency
- Congenital weakness of tissues can lead to nulliparous prolapse

5.4. Management

5.4.1 At PHC:

Preoperative care given at PHC can help in shortening the hospital stay and reduce the number of visits to the district hospital.

- Treat urinary tract infection. Correct Anemia, control Diabetes
- Treat the decubitus ulcer before surgery: Reposition of prolapse and maintaining it in reposed position helps in healing of ulcer. Glycerine-acriflavine vaginal packing daily helps in healing. In older women having severe vaginal atrophy, vaginal application of estrogen cream helps in rapid healing. The ulcer will usually heal within 7 days.

5.4.2 At FRU/DH:

The women should be evaluated completely for planning the treatment modality. Most women will be candidates for surgical treatment. In a young woman following childbirth conservative measures should be advised for 3 to 4 months.

a) The nonsurgical treatment is useful in following women

- Women having mild degree of prolapse
- Pregnancy and Postpartum
- Unfit or unwilling to undergo surgery.

Kegel exercises to strengthen the pelvic supports.

Ring pessary: Can provide temporary relief for woman unfit for surgery,

b) Surgical treatment:

Surgery can be of two types.

- Vaginal hysterectomy with vaginal wall prolapse repair and
- Uterus conserving surgical repair. Choice of surgical procedure depends on age, parity and wish for further pregnancy and medical fitness.

In younger women desirous of retaining fertility, conservative surgical repair operations are indicated, whereas in the peri-menopausal and menopausal

women, vaginal hysterectomy with repair of the pelvic floor is performed.

5.5. Prevention

- Antenatal physiotherapy, relaxation exercises and due attention to weight gain and Anemia correction are important.
- Proper management of second stage of labour. Avoiding undue prolongation of second stage by timely assisted delivery.
- Immediate and accurate suturing of perineal tear after delivery.
- Postnatal exercises and physiotherapy are beneficial.
- Early postnatal ambulation.
- Provision of adequate rest for the first 6 months after delivery
- Contraception: Too many births at too short intervals are avoided.

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Simplified Partograph

Annexure 2



Annexure 3






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4. Dermatology

1.INFECTIONS OF SKIN

1. Bacterial Infections of Skin

It is infection of skin caused by bacteria. Persons with poor personal hygiene, old age, Diabetes and Immunocompromised patients are prone to bacterial infection of skin.

1.1. Pyoderma

1.1.1 Types

a. Impetigo

- Starts as an erythematous macule
- Develops into a vesicle with erythematous halo
- Vesicle breaks down with oozing
- Dries to form golden yellow crust, spreads by auto inoculation

b. Ecthyma

- Deeper infection
- An ulcer covered with adherent crust
- Heals with scarring
- Rule out Diabetes Mellitus in case of recurrent furunculosis and carbuncle.

c. Folliculitis

• Follicular oriented pustules without perifollicular oedema.

d. Furuncle

- Erythematous, painful, tender
- Forms discrete follicular nodules with perifollicular edema.





e. Carbuncle

- Infection of multiple adjoining hair follicles
- Presents with painful, tender plaque with ulceration.

Rule out Diabetes Mellitus in case of recurrent furunculosis and carbuncle



Figure 1.2 Carbuncle

1.1.2 Investigations: - CBC, BSL (R) **1.1.3 Treatment guidelines:**

- Good personal hygiene
- Wash with soap and water
- Topical antibacterial cream Neomycin 0.5% / Framycetin 1% / Mupirocin / Fusidic acid local application two times in a day for 7 days
- Systemic antibiotics Erythromycin 250 or 500 mg QID / Cefadroxil 250 or 500mg BD for 7 days
- Azithromycin 250 mg BD for 7 days

For recurrent pyoderma:

• Topical application of Mupirocin 2% to carrier sites especially anterior nares and natal clefts for 2 weeks

1.1.4 Erysipelas

- Erysipelas is a superficial form of bacterial cellulitis
- Usually seen over the face
- H/o minor injury may be present
- Present as sharply defined erythematous tender swelling



Figure 1.3 Erysipelas

• With partial alopecia showing broken off hair.

Treatment:

- Systemic antibiotics- Azithromycin 250 BD for 5-7 days / Cloxacillin / Amoxicillin-Clavulanic Acid - 625 mg BD for 7 days.
- IV antibiotics for severe cases -Inj. Cefotaxime 1 gm IV BD for 7 days.
- Non-steroidal anti-inflammatory drugs Tab. Ibuprofen for pain & fever

1.1.5 Cellulitis

- Cellulitis is a deep subcutaneous infection with lymphangitis and adenopathy
- Borders of the lesion are poorly demarcated
- Vesicles and bullae may be seen over the surface in both conditions.



Figure 1.4 Cellulitis

Treatment:

- Non-steroidal anti-inflammatory drugs Tab. Ibuprofen 1 BD for 7 days.
- Amoxycillin-Clavulinic Acid 625mg BD / Cloxacillin for 7-10 days
- IV antibiotics for severe cases Inj. Cefotaxime 1 gm IV BD for 7 days.

2. Fungal infections of skin

2.1 Dermatophyte infections

Superficial fungal infections of keratinized tissue caused by dermatophyte fungi. Persons with Diabetes, Obesity and Immunocompromised patients are prone to infections.

Dermatophytosis is classified according to the body part involved.

2.1.1 Tinea Capitis

Presents with different clinical types and is more common in children.

a. Gray patch

• Well defined scaly circular patches

b. Black dot

• Broken off hair near the surface give

appearance of black dot.

• Diffuse and poorly circumscribed lesions with low grade folliculitis.

c. Kerion

• Boggy purulent inflammatory nodules and plaques with sinus formation and pus discharge which leads to thick crusting and matting of adjacent hair.

d. Favus

- Perifollicular erythema and matting of hair with fetid odour
- Heals with scarring alopecia.

e. Investigation: - CBC, BSL (R)



Figure 1.5 Tinea Capitis

f. Treatment

Topical anti-fungals (lotions / creams)

- Clotrimazole 1% local application BD for 15 days 1 month.
- Miconazole 2% local application BD for 15 days 1 month.

Systemic anti-fungals

• Tab. Griseofulvin 10mg/kg/day for 4 to 6 weeks



Figure 1.6 Kerion

Systemic antibiotics in case of secondary infection - Azithromycin 250 BD for 5-7 days / Cloxacillin / Amoxicillin - Clavulanic Acid - 625 mg BD for 7 days.

• Tab. Fluconazole 150 mg / week for 4 to 6 weeks.

2.1.2 Tinea Corporis

Dermatophytosis of the skin with the exclusion of palms, soles and groin is called Tinea Corporis

- Well defined scaly annular patches
- With or without vesicles, pustules at margin
- Single or multiple scattered lesions
- Itching present



Figure 1.7 Tinea Corporis

2.1.3 Tinea Cruris

Infection of groin area and includes infection of genitalia, pubic area, perianal and perineal skin.



Figure 1.8 Tinea Cruris

Topical

Treatment:

- Clotrimazole 1% local application BD for 4-6 weeks.
- Miconazole 1% local application BD for 4-6 weeks.

Systemic

- Tab. Griseofulvin 250 to 500 mg BD for 3 weeks
- Tab. Fluconazole 150mg one per week for 4 -6 weeks

2.1.4 Tinea Pedis

Dermatophyte infection of the feet.



Figure 1.9 Tinea Capitis

2.1.5 Tinea Manuum

Dermatophyte infection of palms and inter-digital areas of hand.

Treatment:

Topical

- Clotrimazole- 1% local application BD for 4-6 weeks.
- Miconazole 2% local application BD for 4-6 weeks.

Systemic

- Tab. Griseofulvin 250 to 500 mg BD for 3-6 weeks
- Tab. Fluconazole 150mg one per week for 4-6 weeks

2.1.6 Tinea Unguium

Dermatophyte infection of nail plate causing whitish or brownish yellow discoloration of nail, separation of nail bed and nail plate

• Nails becomes friable and discolored

Treatment:

- Griseofulvin 1 gm per day for 4-6 months for finger nails and 12-18 months for toe nails
- Tab. Fluconazole 150mg one per week for 6 months for finger nails & 12 months for toe nails.

2.1.7 Candidiasis:

Caused by yeast like fungi Candida albicans

a. Chronic Paronychia

- Infection of nail fold
- Erythema and swelling of finger tips

b. Intertrigo

- Seen over intertriginous areas, groin, in between toes, below the neck in children
- Sharply demarcated polycyclic erythematous eroded patches with satellite pustules
- Rule out underlying Diabetes Mellitus

c. Diagnosis

• **KOH mount** Direct examination of scrapings under microscope from lesions with KOH mount will reveal budding yeasts with hyphae or pseudo-hyphae.

d. Treatment

- Keep intertriginous areas dry
- Clotrimazole– 1% local application BD for 4-6 weeks.
- Miconazole– 2% local application BD for 4-6 weeks.
- Tab. Fluconazole 150 mg per week for 4 weeks.
- Treat underlying Diabetes mellitus

2.1.8 Tinea Versicolor

Chronic superficial fungal infection caused by *Malassezia furfur*. Lesion vary color from white, pink to brown usually consisting of coalescing macules, patches with fine scales on trunk and neck.



Figure 1.10 Tinea Versicolor a. Treatment

• Oral Fluconazole given as single dose of 400 mg

b. Topical

- Ketoconazole 2% shampoo Local application half an hour before bath for 4 weeks.
- Selenium sulphide suspension Local application half an hour before bath for 4 weeks.
- Clotrimazole 2% solution local application for 4 to 6 weeks

3. Viral infections

3.1 Herpes Zoster



Figure 1.11 Herpes Zoster 3.1.1 Prodromal Symptoms

- Pain at the site
- Closely grouped reddish papules, vesicles and pustules in continuous or interrupted band in one or adjacent dermatomes
- Usually unilateral and does not cross the midline
- Thoracic segments most commonly involved.

3.1.2 Treatment

• Tab. Acyclovir 800mg 1-1-1-2 for 5-7 days.

3.1.3 Herpes Eye: In Herpes Zoster Ophthalmicus, refer to ophthalmologist for further management.

Disseminated herpes zoster occurs in immunocompromised individuals

3.2 Herpes simplex

3.2.1 Infection occurs in two types

- a. HSV1 usually responsible for cutaneous, oropharyngeal and ocular infections.
- b. HSV2 involved in genital infections and infections of new born.
 - Oral Infections shows vesicles, erosions on buccal mucosa or peri-oral skin with severe pain and high fever.
 - Vulvo-vaginitis in female and painful penile lesions in men with appearance of vesicles and erosions seen.



Figure 1.12 Herpes Zoster 3.2.2 Treatment:

Tab. Acyclovir 200mg 1-1-1-2 for 5-7 days

3.3 Molluscum Contagiosum

3.3.1 Clinical manifestations:

Molluscum Contagiosum – A viral infection of the skin caused by a poxvirus. It is a contagious disease particularly common in young children. After approximately two to three months of the virus incubating, small, round growths begin to emerge. These are light pink or tan coloured and can look similar to warts. They can sometimes become red and irritated or have a tiny white depressed spot in the centre.

3.3.2 Treatment:

- Needling
- Curette most common, cryotherapy with liquid Nitrogen
- Electrocautery
- Topical 50% Trichloroacetic Acid Twice a week till resolution

3.4 Warts

- Filliform or digitate warts
- Plantar warts

a. Common warts:

• Firm keratotic papules with rough horny surface



Figure 1.13 Common warts

b. Plane warts

• Multiple smooth, flat or slightly elevated round or polygonal papules (1 to 5 mm)



Figure 1.14 Filiform or Digitate c. Filiform or Digitate

• Composed of one or more finger like projections 2 to 10 mm in length.

d. Planter Warts

- Sharply defined rough keratotic lesion.
- Surrounded by a smooth collar of thickened horn.



Figure 1.15 Planter Warts

- e. Treatment:
 - Spontaneous resolution possible
 - Topical keratolytics
 - 16.7% Salicylic acid and 16.7% Lactic acid in collodion base local application daily till resolution

OR

50% Trichloroacetic acid applied weekly till resolution

OR

- Topical 5% Fluorouracil ointment local application daily till resolution.
- Electrocautery or liquid Nitrogen therapy
- Podophyllin 25% for genital wart twice weekly till resolution
- Cryotherapy with liquid nitrogen for genital wart

4. Cutaneous Tuberculosis

It is infection of skin caused by mycobacterium tuberculosis. Persons with malnutrition and immuno-compromised patients are prone to tuberculosis.

4.1 Clinical types

- Lupus vulgaris
- Tuberculosis verrucosa cutis
- Scrofuloderma

4.1.1 Lupus vulgaris:

Reddish brown plaque which spreads with foci of scarring

• It has an active advancing edge and areas of scarring.



Figure 1.16 Lupus Vulgaris

4.1.2 Tuberculosis Verrucosa Cutis:

• Indurated warty papule or nodule or a plaque, with surrounding erythema.

- There may be fissuring and discharge from the surface.
- Irregular extension leads to serpiginous margin.

4.1.3 Scrofuloderma:

- It occurs over the underlying focus of tuberculosis like caseating lymph gland, bone, joint etc.
- Present as painless bluish red swelling which breaks down to from sinus or undermined ulcer.

4.2 Investigations

- TT (Tuberculin Test)
- X-ray chest PA view Screen for Pulmonary

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Tuberculosis.

- Skin biopsy to confirm diagnosis.
- Treatment after confirmation.

4.3 Treatment

Anti-tubercular treatment - 6 months

- 4 Drugs 2 months
- 2 Drugs 4 months
- Refer RNTCP guidelines.

2. NON INFECTIOUS SKIN CONDITIONS

1. Urticaria

- Elevated erythematous itchy swelling
- Transient in nature
- Worsened by scratching
- Acute < 6 weeks
- Chronic >6 weeks
- Angioedema involves the deeper structures

1.1. Provoking factors

- i. Drugs
- ii. Food
- iii. Infection
- iv. Stress
- v. Systemic Diseases



Figure 2.1: Urticaria

1.2. Treatment

- Eradication of the etiological factor
- In Acute Urticaria with laryngopharyngeal edema 0.5 to 1 ml of subcutaneous epinephrine.

Antihistamines

• Tab. Cetirizine 10 mg /day for 2 to 4 weeks

OR

• Tab. Levocetirizine 5 mg per day for 2 to 4 weeks

OR

• Tab. Loratadine 10 mg /day for 2 to 4 weeks

Severe extensive cases or unresponsive cases

- Tab. Prednisolone 0.5 to 1 mg /kg per day and gradually tapered
- Tab. Albendazole 400 mg single dose

2. Psoriasis

2.1 Clinical features

- i. Erythematous well defined scaly plaques
- ii. Predominantly extensor aspect of the body
- iii. Scales are silvery and loosely attached to the lesion
- iv. Koebner phenomenon (it is isomorphic reaction to trauma or injury) in which positive new lesion occurs at the site of trauma



Figure 2.2: Psoriasis

Scalp:

• Scaling that extends beyond the hair line

Nails:

Collection of keratotic material under the nail lifting the distal part of the nail from the nail bed and Nail pitting.

Joints may be involved.

2.2 Treatment

- i. Potent topical steroids for localized lesion– Clobetasol Propionate 0.05% local application twice daily for 2 weeks.
- ii. Topical keratolytics– Salicylic acid 3% or 6% twice daily for 2 weeks.
- iii. Liquid paraffin for external use or use of any other emollients local application twice daily for 2 weeks.
- iv. Tab. Cetirizine 10 mg per day if itching persists
- v. 5% Coal tar ointment local application twice daily for 2 weeks.
- vi. Calcipotriol cream local application twice daily for 2 weeks.

Patients with extensive involvement, arthropathy, pustular psoriasis, erythrodermic psoriasis refer to higher centre.

Refer to higher centre if no response

3. Lichen Planus

3.1 Clinical features

- Well defined, violaceous, polygonal flat topped papules
- Itching present
- Common Sites: leg's and forearm
- Koebner Phenomenon +ve
- Involvement of hair leads to alopecia
- Oral mucosa: lacy network of whitish patches



Figure 2.3 Lichen Planus

3.2 Treatment

- Topical steroids Clobetasol Propionate 0.05% twice daily for 2 weeks.
- Tab. Cetrizine 10 mg daily for 2 weeks.
- Generalized forms Systemic steroids like Tab. Prednisolone 0.5 mg/kg per day. Gradually tapered over the next 4-6 weeks
- Refer to higher centre for extensive lesions.

4. Pityriasis Rosea

4.1 Clinical features

- Initial lesion 2 to 6 cm circular asymptomatic erythematous scaly plaque called herald patch
- Within days, crops of papules and oval patches with wrinkling surface and a border of fine scales
- Trunk and proximal extremities are commonly involved (covered part)
- Lesion heal with hyper or hypo pigmentation

4.2 Differential diagnosis:

- Psoriasis
- Secondary syphilis
- Tinea Corporis Note - Systemic Steroids should be avoided

4.3 Treatment

- Mostly do not require treatment
- Symptomatic treatment like topical moisturizers local application BD for 7 days.
- Tab. Cetirizine 10 mg per day if itching is present Daily sun exposure of covered part if possible.

Sequelae: Hyper pigmentation and scarring.



Figure 2.4 Pityriasis Rosea

5. Acne

5.1 Clinical features

Common in adolescents

Present as comedones, papules, pustules, nodules, abscess, cysts, scars Sites –Face, chest, back, shoulder

5.2 Treatment

5.2.1 Grade I

Comedones / Occasional papules.

- Washing of face with soap and water when it becomes oily.
- Removal of comedones by comedone extractor.
- Topical Retinoic acid (0.025%) local application HS for half hour for 2 to 4 weeks.
- Benzoyl Peroxide (2.5%) local application HS for half hour for 2 to 4 weeks

5.2.2 Grade II

Papules, comedones, pustules:

- Washing of face with soap and water when it becomes oily.
- Topical antibiotics Clindamycin local application BD for 2 weeks.
- Erythromycin 2 to 4% local application BD for 2 weeks.
- Benzoyl peroxide local application HS for half hour for 2 to 4 weeks.
- Retinoic acid local application HS for half hour for 2 to 4 weeks.

5.2.3 Grade III

Predominant, pustules, nodules, abscesses:

Washing of face with soap and water when it becomes oily.

- Topical benzoyl peroxide local application HS for half hour for 2 to 4 weeks.
- Retinoic acid local application HS for half hour for 2 to 4 weeks.
- Azithromycin 500 mg OD 3 days per week, may be repeated; maximum duration 3 months, or
- Doxycycline 100 mg OD for 2 to 6 weeks, or
- Isotretinoin 0.5mg/kg/day for 16 to 24 weeks (only after expert opinion)

5.2.4 Grade IV

Mainly cysts, abscess, widespread scarring

- Washing of Face with soap and water when it becomes oily.
- Systemic antibiotics and Isotretinoin as in grade III
- Aspiration of cysts
- Intralesional steroids for cysts Inj. Triamcinolone Acetonide 10 mg / ml weekly.

6. Pityriasis Alba

6.1 Clinical features

- Asymptomatic round or oval hypo-pigmented scaly patches usually on the face.
- Single or multiple
- Spontaneous resolution may recur.
- Age group 3 years to puberty

6.2 Differential diagnosis

- Early vitiligo
- Pityriasis versicolor
- Leprosy
- Post inflammatory hypo pigmentation

6.3 Treatment

- Reassurance
- Bland emollients for external application like liquid paraffin local application BD.
- Multivitamins like Calcium carbonate+ Vitamin D3 Syrup 1 tsp HS for 1 month.
- Treatment of worm infestation if any– Tab. Albendazole 400 mg HS single dose.
- Cap. Vitamin A 50,000 IU OD for 4-6 wks.

7. Miliaria

7.1 Clinical features

- Small discrete itchy non follicular erythematous confluent macules or papules forming sheets
- Pricking sensation
- Occurs more in summer and heat condition.

7.2 Treatment

- Avoidance of excessive exposure to heat
- Calamine lotion local application BD for 5 days.
- Antihistamines Tab. Cetirizine 10 mg HS for 5 days.
- Tab. Vitamin C 500 mg OD for 5 days.
- Loose cotton dresses
- Plenty of oral fluids etc.

8. Eczema

8.1 Stages

- These are synonymous terms signifying inflammatory response of skin to different factors
 Caused by exogenous or endogenous factors
- Caused by exogenous or endogenous factors
- Generally, 3 stages acute, subacute and chronic



Figure 2.5 Eczema / Dermatitis

- **8.1.1** Acute Stage- Characterised by erythema, oedema, vesicles and oozing
- **8.1.2** Subacute stage- Erythema, oedema, vesicles decrease and are replaced by moderate oozing, crusting & Scaling
- **8.1.3 Chronic Stage-** Mainly consists of hyper pigmentation and lichenification. Highly pruritic in all stages.

8.2 Treatment

• Treatment is according to stage of dermatitis.

8.2.1 Acute Eczema

- Wet soaks / compresses of Potassium permanganate (4-5crystals in 500ml water) local application 3 times in a day.
- Antihistaminic Tab. Cetirizine 10 mg BD for 5 days.
- Topical corticosteroids- Clobetasone 0.05% local application 3 times in a day.
- Systemic Antibiotics if secondary infections.

8.2.2 Subacute Eczema

- Wet compresses for crust removal like acute Eczema local application 3 times in a day.
- Betamethasone or Mometasone topical application in cream form local application 3 times in a day.

• Systemic Antibiotics if secondary infections.

8.2.3 Chronic eczema

- Corticosteroid ointments Clobetasone 0.05% local application 3 times in a day.
- Tab. Cetirizine 10mg BD for 5 to 10 days.

8.2.4 Acute infected Eczema

- i. Wet dressing with light weak pink potassium permanganate soaks for 5 days where indicated
- Whenever infection is present, it has to be treated with appropriate antibiotics like Azithromycin 250 BD for 5-7 days / Cloxacillin 500mg three times a day 5 to 7 days / Amoxicillin-Clavulanic acid - 625 mg BD for 7 days.
- iii. Emollients like liquid paraffin applied daily.
- iv. Mild cases- Hydrocortisone 1% ointment daily
- v. Moderate cases- Betamethasone 0.1% cream or ointment twice daily

8.2.5 Symptomatic relief by Antihistamines

Tab. Chlorpheniramine (4 mg) TID as needed or Tab. Promethazine (10-25 mg) 6-8 hourly as needed in severe cases.

Refer to higher centre to confirm diagnosis and for management of complicated cases.

8.2.6 Management as per severity

Mild or Moderate Infected Eczema

• Soaks or compresses of plain tepid water or normal saline

Severe Eczema

- Tab. Prednisolone (5 or 10 mg) 0.5 to 1mg/kg with gradual tapering,
- If infected, Tab. Erythromycin (250 to 500 mg) QID for 5 to 10 days
- Tab. Chlorpheniramine Maleate (4 mg) TDS for 5 to 10 days.

9. Dermatitis

9.1 Atopic Dermatitis Diagnostic Feature

• Chronic pruritic dermatitis over face, neck and flexures. Seen mainly in infants and children

9.2Treatment Guidelines

- Tab. Chlorpheniramine (4 mg) TDS for 3 Days
- Topical steroid (Clobetasone-Butyrate cream (0.05%)) BD for 3 weeks.
- Tacrolimus 0.03% BD for 3 months topically



Figure 2.6 Atopic Dermatitis

9.3 General Guidelines

- Use soft non irritating cotton clothing
- Use emollients such as oils applied over wet skin. Avoid foods which tend to aggravate lesions e.g. nuts, eggs, wheat, cow's milk, fish.
- Accentuation under Wood's lamp
- Skin as well as mucous membranes can get involved.

9.4 Treatment

- Moderately potent corticosteroids Fluticasone / Mometasone cream topically for 6 weeks
- Tacrolimus 0.03% for face; 0.1% for trunk and extremities for 3 months
- Extensive / resistant cases refer to higher centre

10. Alopecia Areata

10.1 Diagnostic features

Loss of hair in patches

- Patches of non-scarring alopecia especially over the scalp and the beard area. Usually round or oval in shape with a normal appearing superficial skin. Asymptomatic, Exclamation mark appearance of the hair
- Avoid irritating strong soap chemicals
- Exclusive breast feeding in infants
- Review patient after 2 weeks.

10.2 Treatment

Moderately potent corticosteroids (Mometasone Furoate, Fluocinolone Acetonide, Fluticasone) for 6 to 12 weeks

11. Vitiligo

11.1 Diagnostic feature

• Depigmented macules and patches which can occur anywhere on the body (Fig. 7)

12. Androgenic Alopecia

Underlying susceptibility of hair follicles to androgenic miniaturization.

Affects up to 70% of men and 40% of women

Treatment:

Topical Minoxidil solution (2 to 5%) BD application for 4 to 6 months.



Figure 2.7: Vitiligo

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3. SEXUALLY TRANSMITTED INFECTION (STI)

1. Syphilis 1.1 Early Syphilis

1.1.1 Primary Syphilis

- Causative agent- Treponema pallidum
- Incubation period -9 to 90 days
- Genital ulcer (primary chancre)- classical lesion
- Regional lymphadenopathy in 50% of cases
- Primary chancre resolves in 3 to 6 weeks
- After 3 weeks of appearance of chancre, VDRL becomes reactive
- *Treponema pallidum* can be demonstrated from the chancre

In the case of untreated primary syphilis, patient may develop secondary syphilitic lesions in 3-6 months interval.

1.1.2 Secondary Syphilis

Skin Rash

Maculopapular, Psoriasiform or Papulosquamous Annular

Pustular Follicular

- Rash is generalized, bilaterally symmetrical non itchy
- Mucosal lesion
- Snail track ulcers
- Condylomata lata
- Painless discrete, rubbery lymph nodes, systemic manifestations
- Highly reactive VDRL

Treponema pallidum can be demonstrated from the skin lesions, Cutaneous gumma

1.1.3 Early latent Syphilis

- No Signs and symptoms
- 2years period is demarcation between early and late latent syphilis

1.1.4 Treatment:

For all the above 3 conditions

a. Penicillin treatments

Inj. Benzathine penicillin 2.4 mega units deep IM after test dose

b. In Penicillin allergic patients:

• Cap. Doxycycline 100mg orally BD for 2 weeks

OR

• Cap. Tetracycline 500 mg orally QID for 2 weeks

OR

• Tab. Erythromycin 500 mg orally QID for 2 weeks

c. Follow Up:

After treatment the patient should be examined clinically and serologically once a month for first 3 months and quarterly till the end of second year

1.2 Late Syphilis

In untreated early syphilitic patients will have the late syphilitic manifestations after 5-15 years from the onset of infection

1.2.1 Late latent syphilis

After 2 years from the onset of infection No signs and symptoms Non-infectious

1.2.2 Late benign syphilis (Gumma)

After 5-7 years from the onset of infection, visceral gumma

1.2.3 Cardiovascular syphilis

After 10-15 years from the onset of infection; Uncomplicated aortitis, aortic regurgitation, coronary ostial stenosis, aortic aneurysm

1.2.4 Neuro-syphilis

After 10-15 years from the onset of infection; meningitis, meningo-vascular, General Paresis of Insane (GPI), Tabes dorsalis, CNS gumma

1.2.5 Treatment:

a. Late Latent Syphilis and Late Benign Syphilis (Gumma)

- Inj. Benzathine Penicillin 2.4 mega units IM weekly for 4 consecutive weeks OR
- Inj. Procaine Penicillin 1.2 mega units IM daily for 21 days

b. Cardiovascular syphilis and Neuro-syphilis

• Inj. Procaine Penicillin 1.2 mega units IM /day for 21 days under the cover of steroids.

c. Inj. Penicilin allergic patients

- Cap. Doxycycline 100 mg orally BD for 4 weeks
- Cap. Tetracycline 500 mg orally QID for 4 weeks

OR

• Tab. Erythromycin 500 mg orally QID for 4 Weeks

d. Follow-up

The patient should be monitored at 6 months interval, clinically and serologically along with repeat Cerebrospinal examination.

1.3 Congenital syphilis

Untreated early syphilitic mother may deliver a congenital syphilitic child.

1.3.1 Early:

- Skin lesions, rash, bullae, Condylomata lata
- Lymphadenitis, hepatosplenomegaly, osteochondritis
- CNS, kidneys, lungs, testes involvement.

1.3.2 Late:

- Interstitial keratitis
- Neurosyphilis
- Bone and joint involvement
- Sensorineural deafness
- Gummatous lesions
- Cardiovascular syphilis involvement.

1.3.3 Stigmata:

- Bulldog facies
- Hutchinson's teeth
- Rhagades
- Salt and pepper fundus
- Gummatous scars

1.3.4 Treatment:

a. Early: Inj. Benzathine Penicillin 5000 units/kg/day divided in 6 hourly doses for 7 days.

b. Patient Education:

- To seek early and appropriate treatment
- To avoid sexual contact until the treatment is completed
- Insisting epidemiological treatment of sex partners with regular follow-up

c. Prevention:

- Sexual abstinence
- Avoid high risk behavior
- Consistent and correct usage of condoms
- Safer sex practices

2. Chancroid

2.1 Clinical features

- Causative agent Haemophilus ducreyi
- Incubation period Usually 3 to 5 days but it can extend up to 14 days.
- Multiple, painful, non-indurated ulcers with ragged and undermined edges. Floor is covered by a yellowish grey necrotic exudates overlying granulation tissue that bleeds on manipulation.
- In 50% of the patients, a tender inguinal adenitis (usually unilateral) occurs (Inflammatory Bubo)
 - "School of fish appearance" in smears



Figure 3.1 Chancroid

2.2 Treatment

- Cap. Doxycycline 100mg oral BD for 14 days OR
- Tab. Erythromycin 500mg orally QID for 14 days

OR

- Tab. Azithromycin 1 gm oral stat OR
- Tab. Ciprofloxacin 500 mg oral BD for 3 days

3. Lymphogranuloma Venereum (LGV)

- **3.1 Clinical features**
- Caused by *Chlamydia trachomatis* serovars L1, L2, L3
- Incubation period 3 to 12 days
- Primary Transient genital ulcer Lymph nodes (bubo)



Figure 3.2 Lymphogranuloma Venereum

Tertiary complications include:

Elephantiasis of genitalia Fistulae Strictures

3.2 Treatment

- Cap. Doxycycline 100mg oral BD for 3 weeks OR
- Tab. Erythromycin 500mg oral QID for 3 weeks

4. Venereal Granuloma (Donovanosis)

4.1 Clinical features

- Causative agent- Klebsiella granulomatis (Calymmatobacterium granulomatis)
- Incubation period 1 to 3 months
- Granulomatous ulcer
- Calymmatobacterium granulomatis (Donovan bodies) seen inside large mononuclear cells in tissue smears.

4.2Treatment

- Cap. Doxycycline (100mg) BD
- Tab. Cotrimoxazole (800mg + 160 mg) BD OR
- Tab. Erythromycin (500mg) 4 times a day in pregnant women for14 Days

OR

• Inj. Ceftriaxone (1gm) daily IM injection for 7 days

5. Gonorrhoea

5.1 Clinical features

- Causative agent Neisseria gonorrhoeae
- Incubation period 1 to 14 days
- Presents as urethritis in male and cervicitis in female



Figure 3.3 Gonorrhoea 5.2 Complications

5.2.1 Male:

- Posterior urethritis
- Infection of Cowper's and Tyson's gland
- Epididymitis
- Prostatitis
- Seminal Vesiculitis
- Peri-urethral abscesses

5.2.2 Female:

- Salpingitis,
- Peritonitis,
- PID
- Bartholinitis,
- Proctitis
- Chronic urethritis

5.2.3 Other forms:

- Disseminated gonococcal infection
- Gonococcal arthritis
- Meningitis
- Anorectal
- Pharyngeal gonorrhea

5.3 Treatment

- Tab. Azithromycin 2 g oral stat (or)
- Inj. Ceftriaxone 250 mg IM stat

6. Non-gonococcal urethritis

6.1 Clinical manifestation

- Causative agent: Chlamydia trachomatis Ureaplasma urealyticum, Mycoplasma genitalium
- Urethritis
- Increased Polymorphonuclear leucocytes in urethral smear or sediment of first voided urine.

7. Chlamydial infections

7.1 Clinical features

- Agent- Chlamydia trachomatis
- Incubation period -1 to 3 weeks

7.2 Complications

7.2.1 Male:

Urethritis, Littritis, Epididymitis, Prostatitis, Proctitis, Reiter's syndrome

7.2.2 Female:

Cervicitis, Urethritis, Bartholinitis, Endometritis, Salpingitis

Fitz Hugh Curtis Syndrome (It is fever with lower abdominal pain after cervical movement. There are adhesions in pelvic cavity, may lead to infertility.)

7.3 Treatment

- Tab. Azithromycin 1 gm oral stat OR
- Cap. Doxycycline 100 mg oral BD for 14 days OR
- Tab. Erythromycin 500 mg QID for 14 days

8. Bacterial Vaginosis

8.1 Clinical aspects

- Polymicrobial Syndrome
- Gardnerella Vaginalis
- *Mycoplasma hominis*, bacteroides
- Excessive, homogenous, uniformly adherent vaginal discharge, elevated vaginal pH > 4.5
- Positive Amine test (Whiff test)
- Presence of 'Clue-Cells' (Epithelial cells

studded with organisms)

8.2 Treatment

- Tab. Metronidazole 400 mg BD for 14 days OR
- Tab. Secnidazole 2g stat dose

9. Trichomoniasis

9.1 Epidemiology

- Caused by Trichomonas vaginalis
- Incubation period 4 to 28 days

9.2 Clinical features

- Copious, homogenous, malodorous, yellowish green vaginal discharge
- Punctuate hemorrhages over cervix Strawberry cervix
- Demonstration of motile trichomonads in wet film smears under direct microscopy
- Culture gold standard for diagnosis

9.3 Treatment

- Tab. Metronidazole 400 mg BD for 14 days OR
- Tab. Secnidazole 2g stat dose

10. Candidiasis

10.1 Clinical features

- Predominantly caused by Candida albicans
- Predisposed by pregnancy, diabetes, HIV and immunosuppression
- Causes Balanoposthitis in males
- Clinically erythema and swelling with a macular /papular rash over glans penis.
- A white sub-preputial discharge, increased skin marking, fissuring of the glans and foreskin occasionally regional lymphadenopathy may be present
- Causes Vulvo-vaginal candidiasis in females, oedema, fissures, erosions, curdy white discharge.
- Demonstration of candidiasis by 10% KOH yeast cells, mycelia seen.



Figure 3.4 Warts (Condylomata Acuminata)

10.2 Treatment

- Commonly used antifungals include oral Fluconazole and topical Clotrimazole.
- Vaginal candidiasis a single dose of oral Fluconazole (150mg)
- Clotrimazole pessary for 7 days Daily
- Clotrimazole cream (1%) or Miconazole cream (2%) daily BD
- Application for 7 to 14 days

11. Warts (Condylomata Acuminata)

11.1 Clinical features

Caused by Human Papilloma Virus

- Incubation period 1 to 8 month
- Skin coloured multiple verrucous lesions

11.2 Treatment

- Topical Podophyllin 25% local application twice weekly.
- Cryotherapy
- Cautery
- Surgery

11.3 Treatment

11.3.1 For Primary

• Tab. Acyclovir 200 mg 5 times a day for 7 days

11.3.2 For Recurrence

• Tab. Acyclovir 400 mg 3 times a day for 5 days

12. Herpes Genitalis

12.1 Clinical features

- Predominantly by HSV -2 & also by HSV -1
- Incubation period 5 to 14 days
- Ballooning degeneration and giant cells seen in HPE
- Primary Severe associated with systemic symptoms starts as grouped vesicles – ulcerate
 polycyclic margin
- Recurrence less severe

12.2 Complications

- CNS involvement dissemination Secondary infection
- Recurrence
- Transmissible during pregnancy and delivery

Figure 3.5: Herpes Genitalis



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4. SYNDROMIC MANAGEMENT OF STD

1. Syndromic Approach

- Provision of STI/RTI care services is a very important strategy to prevent HIV transmission and promote sexual and reproductive health under the National AIDS Control Programme (NACP III) and Reproductive and Child Health (RCH II)
- Syndromic case management (SCM) with appropriate laboratory tests is the cornerstone of STI/RTI management under NACP III.
- SCM is a comprehensive approach for STI/RTI control endorsed by the WHO.
- Diagnosis is based on the identification of syndromes, which are combinations of the symptoms the client reports and the signs the health care provider observes.
- The provision of the most effective therapy at patient's first contact with a health or medical facility.
- The recommended treatment is effective for all the diseases that could cause the identified syndrome.
- Provides single does treatment as far as possible.
- Comprehensive to include patient education on risk reduction, counseling, condom promotion and provision, partner notification, follow up.

2. Why Syndromic Management

- STI signs and symptoms are rarely specific to a particular causative agent.
- Laboratory services may not be available.
- Dual infections are quite common and both clinician and laboratory may miss one of them.
- Waiting time for lab. Results may discourage some patients.
- Failure of cure at first contact.

3. The common STD Syndromes

- Genital ulcer diseases
- Urethral discharge
- Vaginal discharge
- Bubo (inguinal swelling)
- Lower abdominal pain in female
- Scrotal swelling
- Ophthalmia neonatorum

Syndromic approach for STI s is useful and practical strategy for offering, high quality, effective and acceptable care for prevention and treatment of sexually transmitted infections (Guidelines from National Aids Control Organization (NACO) has been used)

Kit	Color	Composition of kit	Syndrome / disease
1	Grey	Tab. Azithromycin 1 g stat + Tab. Cefixime 400 mg.	Urethral discharge (UD), cervical discharge (CD), anorectal discharge (ARD), Presumptive treatment (PT), Painful scrotal swelling.
2	Green	Tab. Secnidazole 2 g stat + cap. Fluconazole 150 stat	VD (Vaginal discharge)
3	White	Inj. Benzathine Penicillin 2.4 MU IM stat + Tab. Azithromycin 1 g stat	Genital ulcerative disease (GUD); non herpetic
4	Blue	Cap. Doxycycline 100 mg BD for15 days + Tab. Azithromycin 1 g stat	(GUD; non herpetic)
5	Red	Tab. Acyclovir 400 mg TDS for 7 days	(GUD; herpetic)
6	Yellow	Tab. Cefixime 400 mg stat + Tab. Metronidazole 400 mg BD for 14 days + Cap. Doxycycline 100 mg BD for 14 days	Lower abdominal pain (LAP)
7	Black	Cap. Doxycycline 100 mg BD for 21 days + Tab. Azithromycin 1 g stat	IB (Inguinal Bubo)

Table 1: NACO Guidelines

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5. HIV – AIDS

1. HIV

Invades the helper T-cells (CD4 cells) in the body of the host (defense mechanism of a person)

'Human Immunodeficiency Virus' a unique type of virus (a retrovirus)

Preventable, manageable but not curable.

2. AIDS

'Acquired Immunodeficiency Syndrome' HIV is the virus that causes AIDS

Disease limits the body's ability to fight infection due to markedly reduced helper T-cells.

Patients predisposed to multiple opportunistic infections leading to death.



Figure 5.1 The Viral Genome

3. Modes of HIV/AIDS Transmission

- Through Bodily Fluids
- Blood products semen and vaginal fluids
- Sharing needles without sterilization Increases the chances of contracting HIV
- Unsterilized blades
- Through unprotected intercourse oral, sexual, anal
- Mother-to-Baby: before birth, during birth, after birth

4. Stages

4.1 Stage 1 – Primary

• Short, flu-like illness - occurs one to six weeks after infection

- Mild symptoms
- Infected person can infect other people

4.2 Stage 2 – Asymptomatic

- Lasts for an average of ten years
- This stage is free from symptoms
- There may be swollen glands
- The level of HIV in the blood drops to low levels
- HIV antibodies are detectable in the blood

4.3 Stage 3 – Symptomatic

- The immune system deteriorates
- Opportunistic infections and cancers start to appear.

4.4 Stage 4 - HIV AIDS

• The immune system weakens too much as CD4 cells decrease in number.

5. Opportunistic Infections associated with AIDS

CD4 < 200

Bacterial infections, Tuberculosis (TB), Herpes Simplex, Herpes Zoster, Vaginal candidiasis, Hairy leukoplakia, Kaposi's sarcoma

5.1 TB & HIV Co-Infection

- TB is the most common opportunistic infection in HIV and the first cause of mortality in HIV infected patients (10-30%)
- 10 million patients co-infected in the world.
- Immunosuppression induced by HIV modifies the clinical presentation of TB: Subnormal clinical and roentgen presentation
- High rate of MDR / XDR
- High rate of treatment failure and relapse (5% Vs. <1% in HIV)

6. Testing Options for HIV

6.1Anonymous Testing

- No name is used
- Unique identifying number
- Results issued only to test recipient

6.2 Blood Test

HIV enzyme-linked immune-sorbent assay (ELISA)	Screening test for HIV; Sensitivity > 99.9%	
Western Blot	Confirmatory test	
	Specificity > 99.9% (when combined with ELISA)	
HIV rapid antibody test	Screening test for HIV	
	Simple to perform	
Absolute CD4 lymphocyte count	Predictor of HIV progression	
	Risk of opportunistic infections and AIDS when <200	
HIV viral load tests	Best test for diagnosis of acute HIV	
	infection correlates with disease	
	progression and response to HAART	

Blood Detection Tests: Table No. 1

7. Treatment Options

7.1 Treatment of Opportunistic Infections (OIs)

Table 2: Managing OIs before starting ART			
Drug reaction	Do not start ART during an acute reaction		
Acute diarrhea which may reduce absorption of ART	Diagnose and treat first, start ART when diarrhea is stabilized or controlled		
Non-severe anemia (Hb <8g/ liters)	Start ART if no other causes for anemia are found (HIV is often the case of anemia), avoid AZT		
Skin conditions such as PPE and seborrheic dermatitis, psoriasis, HIV-related exfoliative dermatitis.	Start ART (ART may resolve these problems)		
Suspected MAC, Cryptosporidiosis and Microsporidiosis	Start ART (ART may resolve these problems)		
Cytomegalovirus infection	Treat if drugs available; if not, start ART		
Toxoplasmosis	Treat; start ART after 6 weeks of treatment and when patient is stabilized.		



Figure 5.2 HAART (Highly active anti-retroviral treatment)

7.2 Antiretroviral Therapy Regimens

Currently, the national programme provides the following drugs / combinations for first-line regimen.

Fixed- dose combinations (FDCs) are preferred because they are easy to use, have distribution

advantages (procurement and stock management), improve adherence to treatment and thus reduce the chances of development of drug resistance. The current national experience shows that BD regimens of FDCs are well tolerated and complied with.

Table 3: Revised NACO ART Regimen 2012			
Regimen I	Zidovudine + Lamivudine + Nevirapine	First line Regimen for patients with $Hb \ge 9$ gm/dl and not on concomitant ATT	
Regimen I (a)	Tenofovir + Lamivudine + Nevirapine	First line Regimen for patients with Hb<9 gm/dl and not on concomitant ATT	
Regimen II	Zidovudine + Lamivudine + Efavirenz	First line Regimen for patients with $Hb \ge 9$ gm/dl and not on concomitant ATT	
Regimen II (a)	Tenofovir + Lamivudine + Efavirenz	First line Regimen for patients with Hb< 9 gm/dl and not on concomitant ATT. First line for all patients with Hepatitis B and / or Hepatitis C co-infection First line Regimen for pregnant women, with no exposure to sd-NVP in the past.	
Regimen III	Zidovudine + Lamivudine + + Atazanavir/ Ritonavir	Regimen for patients on AZT containing first line regimen, who develop toxicity to both NVP and EFV Also second line regimen for those who are on TDF containing first line regimen if $Hb \ge 9$ gm/dl	
Regimen III (a)	Tenofovir + Lamivudine + + Lopinavir / Ritonavir	For patients of regimen III who develop severe Atazanavir toxicity First line regimen for patients with HIV-2 infection with Hb \ge 9 gm/dl	
Regimen IV	Tenofovir + Lamivudine + Atazanavir / Ritonavir	Second line regimen for those who are on AZT / d4T containing regimen in the first line. Also for patients on TDF containing first line regimen who develop toxicity to both NVP and EFV.	
Regimen IV (a)	Tenofovir + Lamivudine + Lopinavir / Ritonavir	For patients on regimen IV who develop severe Atazanavir toxicity First-line Regimen for patient with HIV 2 infection with Hb \leq 9 gm/dl First-line Regimen for all women exposed to sd- NVP in the past.	
Regimen V	Stavudine + Lamivudine + Atazanavir / Ritonavir	Second line for those who are on TDF containing regmen in the first line if Hb \leq 9 gm/dl	
Regimen V (a)	Stavudine + Lamivudine + Lopinavir / Ritonavir	For patients on Regimen V who develop severe Atazanavir toxicity.	

Antiretroviral Drugs – Highly active antiretroviral treatments

- Nucleoside Reverse Transcriptase inhibitors (NRTI)
 - AZT (Zidovudine), Lamivudine, Ditanosine, Abacavir, Empricitadine, Tenofovir
- Non-Nucleoside Transcriptase inhibitors (NNRTI)
 - Nevirapine (Viramune), Efavirenz
- **Protease inhibitors (PI)** • Ritonavir, Lopinavir

8. Primary Prevention

Five ways to protect yourself:

- Abstinence
- Monogamous Relationship
- Protected Sex
- Sterile needles
- New shaving / cutting blades

9. Post Exposure Prophylaxis (PEP)

Steps to be taken on exposure to HIV infected blood, body fluids and contaminated sharps etc.

9.1. Actions to be taken

Immediately following on exposure:

- i. Needle pricks and cuts should be washed with soap and water
- ii. Splashes to the nose, mouth or skin should be flushed with water
- iii. Eyes should be irrigated with clear water, saline or sterile irrigant
- iv. Do not panic
- v. Pricked finger should not be put in to mouth by reflex
- vi. Do not squeeze the pricked finger to expel the contaminated blood
- vii. PEP should be started within 72 hours of exposure, within 2 hours is ideal.

9.2. Post Exposure Prophylaxis (PEP)

The decision to start PEP is made on the basis of degree of exposure to HIV and the HIV status of the source from where the exposure infection has occurred.

9.2.1 Exposure Code Determination





Figure. 5.4 Exposure Source

9.2.3. PEP recommendation based on exposure code & source code

EC	HIVSC	PEP Recommendation
1	1	PEP may not be warranted
1	2	Considered basic regimen
2	1	Recommend basic regime
		(most exposures are in this category)
2	2	Recommend expanded regimen
3	1 or 2	Recommend expanded regimen
2/3	Unknown	If the source (in the case of an unknown source) the settings were the exposure
		occurred suggested a possible risk for HIV
		exposure and EC 2 or 3 consider PEP basic regimen

Figure 5.5. PEP

9.2.4. PEP regimen

In case of intolerance to Efavirenz, regimen containing Tenofovir + Lamivudine + Pl

- a. (ATV/r or LPV/r) can be used after expert consultation by an experienced physician.
- b. Wherever PEP is indicated and source is ART naïve or unknown: recommended regimen is Tenofovir 300 mg + Lamivudine 300 mg + Efavirenz 600 mg once daily for 28 days. Wherever available, single pill containing these

formulations should be used. Dual drug regimen should not be used any longer in any situation for PEP. The first dose of PEP regular should be administered as soon as possible, preferably within 2 hours of exposure and subsequent dose should be given at bed time with clear instruction to take it 2-3 hours after dinner & to avoid fatty food in dinner.

9.2.5 In case of Sexual Assault:

PEP should be provided to exposed person in case of sexual assault as a part of overall package of post sexual assault care.

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- Change



5. Psychiatry

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1. MENTAL ILLNESS

1. Introduction

- 15 to 20% of general population suffers from major or minor type of psychiatric illness causing significant degree of morbidity and mortality in society.
- It is therefore necessary to know about commonly occurring mental illnesses and their primary management.

2. Classification of Psychiatric Disorders

- 2.1 Psychiatric illnesses can be broadly divided into two categories:
- 2.1.1 Major psychiatric illnesses (Abnormality

in thinking, motions and behavior leading to severe impairment in Social and Occupational functioning)

- Schizophrenia
- Bipolar Mood Disorder
- Major Depressive Disorder

2.1.2 Other disorders

- a) Anxiety disorders
- General Anxiety disorders
- Phobias
- Obsessive and Compulsive Disorders
- Somatoform Disorder and Conversion Disorder

b) Special Group Disorders

- Dementia / Alzheimer's Disorder
- Substance use disorder
- Children and Adolescent Disorder
- Intelligence Disability Disorder
- Psychiatric Emergencies ex. suicide, violence

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2. EVALUATION: HISTORY AND MENTAL STATUS EXAMINATION (MSE)

- History: Data from patient (subjective) / from reliable informant (objective)
- Demographic details:
 - o Name
 - o Sex/Age
 - Address
- Presenting complaints
- Onset / duration / progress
- Past h/o of psychiatric illness / treatment
- Past h/o of medical illnesses (Hypertension, Diabetes, TB, Enteric Fever etc) / surgical

procedures, head injury, Epilepsy and Medico-Legal issues

- Family h/o of psychiatric illness, addictions, suicide
- Developmental history
- Level of education
- Occupational history
- Marital history
- Personal history any addiction
- Premorbid personality

1.	Appearance	□Tidy	□Untidy	□Appropriately dressed	□Over dressed
2.	Attitude	□Cooperative	□Uncooperative		
3.	Behavior (psychomotor activity)	□Dull	□Normal	□Increased	
4.	Eye to eye contact	□Present	□Absent		
5.	Rapport	□Established	□Difficult to	□Not Established	
			Establish		
6.	Speech	□Normal	□Mute	□Speaking very few words	□Over talkative
		□Coherent	□Incoherent	iew words	
		□Relevant			
		\Box Flight of ideas	Thought block		
7	Mood				
· ·	11000		□Sad		
8.	Affect				
		□Tearful		\square Blunt	
9.	Hallucinations	□Auditory			
	(Abnormal perception		□Gustatory		
	without external stimulus)		•		
10.	Delusions	□Persecution	□Grandiosity	□Reference	□Infidelity
	(Firm and false belief)				
11.	Obsessive thoughts /	□Cleaning	□Washing	□Arranging	
	compulsive behaviors	□Rituals	□Verbal		
12.	Suicidal / Homicidal	□Present	□Absent		
	ideation				
13.	Level of consciousness				
14.	Orientation			□Person	
15.	Attention and	□Sustained	\Box Not sustained		
1.6	concentration				
16.	Memory				
17.	Intelligence		□Below average		
18.	Judgment				
19.	Insight	□Present	∐Absent		

Table No. 1: Mental Status Examination Format

**Delusions and Hallucinations are the cardinal features of Schizophrenia / Psychosis

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3. SCHIZOPHRENIA

1. Introduction

Schizophrenia is a mental disorder of chronic nature in which there is primary impairment of thoughts, emotions and behavior which leads to severe sociooccupational impairment and personality deterioration.

2. Symptoms

- Suspiciousness
- Fearfulness
- Social withdrawal
- Muttering / smiling to self
- Poor self-care
- Disturbed sleep
- Work impairment

3. Signs

- Unkempt appearance
- Increased or decreased psychomotor activity

- Anxious and fearful affect
- Poverty of speech
- Delusion of Persecution and/or Reference
- Auditory Hallucinations
- Lack of Insight
- Impaired Judgment

4. Investigations

- No diagnostic investigations available.
- Complete Haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, Chest X-Ray (If necessary)

5. Complications

- Severe agitative behavior
- Homicidal tendencies
- Self-harm / Suicidal behavior
- Catatonic Stupor / Excitement

6. Treatment and Management

6.1 Antipsychotics: Primary care physician can start single drug and titrate it up to optimum level

Table No. 1 Antipsychotics

Name	Strength	Recommended doses**	Duration/ Remark
Trifluoperazine	5 mg	10-40 mg/day	Drugs to be started in low
Trihexyphenidyl	2 mg	2-6 mg/day	doses and gradually
Haloperidol	5 mg	10-30 mg/day	increase till 80 %
Risperidone	2 mg	4-8 mg/day	improvement.
Olanzapine	5 mg	5-20 mg/day	
Quetiapine	50/100 mg	50-300 mg/day	Maintenance doses to be
Clozapine	50 mg	50-200 mg/day	continued long term
Chlorpromazine	50 mg	50-200 mg/day	

**To be given in divided doses: It is to be monitored by psychiatrist.

6.2 Sedatives:

Table No.2 Sedatives

Name	Strength	Recommended doses	Duration/ Remark
Lorazepam	2 mg	1-4 mg HS	Drugs to be started in low
Diazepam	5 mg	5-10 mg HS	doses preferably at night
Clonazepam	0.5 mg	0.5-1 mg HS	and to be tapered off
Nitrazepam	5 mg	5-10 mg HS	within 2 weeks

6.3 Modified Electro-Convulsive Therapy:

Minimum of 8-15 sessions of modified ECT are required.

6.4 Treatment and Management of Complications:

- Inj. Haloperidol, 10mg, BD/ SOS for 2/3 days
- Inj. Promethazine*, 50 mg, BD / SOS for 2/3 days

(*Not to exceed 100mg/day)

- Electro-convulsive Therapy
- Inj. Diazepam, 10mg or Lorazepam, 4mg if required

6.5 Maintenance Therapy:

All patients with Schizophrenia have to be maintained on long term antipsychotics oral or injectable treatment.

Depot Injections:

Inj. Haloperidol Decanoate, 50mg, once a month, deep IM

OR

Inj. Fluphenazine 25mg, once a month, deep IM

OR

Inj. Flupenthixol, 40mg, once a month, deep IM

6.6 Side effects and management:

Table No. 3 Common Side Effects and Management

Side Effect	Management	Duration/ Remark
Tremors at extremities / Salivation	Trihexyphenidyl	2-6mg along with antipsychotic drugs.
Dystonic Reaction / Rigidity	Inj. Promethazine 50mg Stat / SOS	SOS
Dry Mouth / Constipation	Reduce dose of Trihexyphenidyl	1 week
Neuroleptic Malignant Syndrome (Rigidity, Fever, Disorientation, Reduced urine output)	Stop Antipsychotics Inj. Lorazepam, Tab. Bromocriptine, Supportive Management	Till patient recovers and CPK level comes to normal.

6.7 Counseling and Psychotherapy

Refer to higher center if needed.

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4. BIPOLAR MOOD DISORDER

1. Introduction

- Distinctive episodes of abnormally and persistently elevated, expansive, irritable or depressive mood
- Patient may be in Mania or Depression or present with mixed features

2. Symptoms of Mania

- Over talkative
- Hyperactive
- Over familiar / over religious / over spending / over grooming
- Big talks
- Decreased need for sleep
- Work impairment

3. Signs of Mania

- Increased psychomotor activity
- Pressured speech / flight of ideas
- Grandiosity
- Easily distractible / irritable
- Hypersexual behavior
- Auditory hallucinations of God / actors talking to him

4. Symptoms of Depression

- Persistent and pervasive low / sad mood
- Lack of energy / interest / initiative
- Hopelessness / worthlessness / helplessness
- Feeling of guilt, agitation
- Suicidal ideas / attempts
- Decreased sleep, appetite and libido
- Sometimes hearing of voices
- Work impairment

5. Signs of Depression

- Dull, withdrawn
- Poor eye contact
- Decreased productivity of speech
- Crying spells
- Feeling of guilt
- Suicidal thoughts, ideas and plans
- Decreased appetite
- Sleep disturbances

6. Investigations

- No diagnostic investigations available.
- Complete haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, chest X-Ray (If necessary).

7. Complications

7.1 Mania:

- Severe agitative behavior
- Homicidal tendencies

7.2 Depression:

• Self-harm / Suicidal behavior

8. Treatment and Management of Mania

8.1 Antipsychotics:

Primary care physician can start single drug and titrate it up to optimum level.

Name	Strength	Recommended doses*	Duration/ Remark
Trifluoperazine	5mg	10-40mg/day	Drugs to be started in low
Trihexyphenidyl	2mg	2-6 mg/day	doses and gradually
Risperidone	2mg	4-8mg/day	increased till 80 %
Olanzapine	5mg	5-20mg/day	improvement.
Quetiapine	50/100mg	50-300mg/day	
Chlorpromazine	50mg	50-200mg/day	Maintenance doses to be continued long term

Table No. 1 Antipsychotics

*To be given in divided doses

8.2 Sedatives:

Table No. 2 Sedatives

Name	Strength	Recommended doses**	Duration/ Remark
Lorazepam	2mg	1-4mg HS	Drugs to be started in low
Diazepam	5mg	5-10mg HS	doses preferably at night
Clonazepam	0.5mg	0.5-1mg HS	and to be tapered off
Nitrazepam	5mg	5-10mg HS	within 2 weeks.

**To be given in divided doses

8.3 Mood Stabilizers

Table No. 3 Mood Stabilizers

Name	Strength	Recommended	Duration/ Remark
		doses***	
Carbamazepine	100/200mg	100-600mg/ day	Drugs to be started in low
Lithium	300mg	300-900mg/day	doses and gradually
Divalproex	250/500mg	500-1500mg/day	increase till 80 %
			improvement.
			Maintenance doses to be
			continued long term

***To be given in divided doses

8.3.1 Side effects of mood stabilizer:

- **Carbamazepine-** Giddiness, blurred vision, rash, hypotension
- Lithium- Tremors, muscle twitching, disorientation
- **Divalproex Sodium-** Gastric irritation, weight gain, lethargy, confusion, tremors

8.4 Modified Electro-Convulsive Therapy:

Minimum of 8-15 sessions of modified ECT are required.

9. Treatment and Management of Depression

9.1 Antidepressants:

Primary care physician can start single drug and titrate it up to optimum level.

Name	Strength	Recommended doses**	Duration/ Remark
Imipramine	25mg	50 to 150mg/day	To be started in low doses
Amitriptyline	25mg	50 to 150mg/day	and gradually increase till
Sertraline	25/50mg	25 to 100mg/day	80% improvement. To
Escitalopram	5/10mg	5 to 20mg/day	continue at least 6 months
Mirtazapine	7.5/15mg	15 to 30mg/day	and more if necessary
Desvenlafaxine	50mg	50 to 100mg/day	

Table No. 4 Treatment and Management of Depression

**To be given in divided doses

9.2 Sedatives

Table No. 5 Sedatives

Name	Strength	Recommended doses**	Duration/ Remark
Lorazepam	2mg	1-4mg HS	Drugs to be started in low
Diazepam	5mg	5-10mg HS	doses preferably at night
Clonazepam	0.5mg	0.5-1mg HS	and to be tapered off
Nitrazepam	5mg	5-10mg HS	within 2 weeks

9.3 Modified Electro-Convulsive

Therapy: 8-15 sessions if needed.

9.4 Treatment and Management of Complications during Mania:

- Inj. Haloperidol 10mg BD/ SOS for 2/3 days.
- Inj. Promethazine* 50mg BD/ SOS for 2/3 days (*Not to exceed 100mg/day)
- Electro-convulsive Therapy.
- Inj. Diazepam(5mg) or Lorazepam(2mg) stat if needed.
- In case of Depression, Electro-convulsive Therapy 8-12 sessions if patient shows suicidal tendencies.

9.5 Precautions for Suicidal patients

- 24 hrs. continuous supervision.
- Do not isolate the patient.
- Do not keep any sharp objects, ropes, blades near patient.
- Do not allow patient to lock room / bathroom from inside.

Note - Reduce doses of mood stabilizers if above side effects are seen.

9.6 Counseling and Psychotherapy

Refer to higher centre if needed.

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5. MAJOR DEPRESSIVE DISORDER (MDD)

1. Introduction

- Depression is major public health problem
- By 2020, it will be the major cause of worldwide morbidity and mortality
- MDD is a disabling condition that severely affects a person's family work, sleeping, eating habits and general health
- It is the leading cause of suicide
- **2. Types:** Mild, Moderate and Severe

3. Signs and Symptoms of MDD

- Pervasive and persistent low mood
- Low self esteem
- Loss of interest or pleasure in normally enjoyed activities
- Lack of energy and enthusiasm, easy fatigue
- Loss of appetite, weight loss
- Disturbed sleep

- Sometimes psychotic symptoms like hallucination and reference ideas
- Hopelessness, helplessness, worthlessness
- Suicidal thoughts, ideas or attempts
- Atypical symptoms
- Overeating
- Oversleeping
- Agitation
- Decreased libido

4. Investigations

- No diagnostic investigations available.
- Complete Haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, Chest X-Ray (If necessary)

5. Complications

Self-harm / Suicidal behavior

6. Treatment and Management of Depression

6.1 Antidepressants:

Table No. 1 Treatment and Management of Depression

Name	Strength	Recommended doses	Duration/ Remark
Imipramine	25mg	50 to 150mg/day	To be started in low doses and
Amitriptyline	25mg	50 to 150mg/day	improvement. To continue at least
Sertraline	25/50mg	25 to 100mg/day	6 months and more if necessary.
Escitalopram	5/10mg	5 to 20mg/day	
Mirtazapine	7.5/15mg	15 to 30mg/day	
Desvenlafaxine	50mg	50 to 100mg/day	

6.2 Sedatives:

Table No. 2 Sedatives

Name	Strength	Recommended doses**	Duration/ Remark
Lorazepam	2mg	1-4mg HS	Drugs to be started in low doses
Diazepam	5mg	5-10mg HS	tapered off within 2 weeks
Clonazepam	0.5mg	0.5-1mg HS	
Nitrazepam	5mg	5-10mg HS	

**To be given in divided doses; if psychotic features are present, add low dose antipsychotics.

6.3 Modified Electro-Convulsive

Therapy: 8-15 sessions are required.

6.4 Treatment and Management of Complications

Electro-Convulsive Therapy Daily ECTs (if needed), minimum for three days.

Precautions for Suicidal patients

- 24 hrs continuous supervision.
- Do not isolate the patient.
- Do not allow patient to lock room/ bathroom from inside.

Counseling and Psychotherapy play a major role. Refer to higher centre if needed.

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6.ANXIETY DISORDER

1. Introduction

- Anxiety is a normal emotion experienced by everyone at times of stress
- Symptoms of anxiety are psychological, physical or mixture of both
- Anxiety becomes a disorder when symptoms become severe, disabling and reduce quality of life
- Anxiety spectrum disorders tend to be chronic

2. Presentation

2.1 Generalized Anxiety Disorder (GAD)

This involves excessive, unrealistic worry and tension without any definite reason.

Signs and Symptoms

Palpitations, dry mouth, tremors, sweating, difficulty in breathing, frequency of urination, abnormal feeling in the gut, muscle tension, dizziness.

2.2 Panic Attacks

- Can occur in patients with GAD
- Signs and symptoms include all of the above with severe choking sensation and shortness of breath with impending feeling of death or going crazy. The attack lasts for few minutes and subsides on its own
- If panic attacks occur frequently, then it is called as **Panic Disorder**

2.3 Phobia

Intense and irrational fear of a specific object or situation leading to phobic avoidance

E.g. Fear of public places, closed spaces, heights, animals, water etc.

2.4 Post-Traumatic Stress Disorder (PTSD)

Disabling fear, intrusive flashbacks or vivid dreams and emotional disturbances after traumatic life event causing avoidance of similar situations

2.5 Obsessive Compulsive Disorder

Frequent intrusive thoughts producing apprehension and fear or worry and leading to compulsive, repetitive behavior aimed to reduce the anxiety

E.g. Frequent thoughts of contamination leading to compulsive washing; frequent checking of locks, gas knobs, counting money etc.

3. Investigations

- No diagnostic investigations available
- Complete Haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, Chest X-Ray (If necessary).

4. Complications

Any of the anxiety spectrum disorder can lead to 'Major Depressive Disorder', making person vulnerable to suicide.

5. Treatment and Management

Includes Drug and Non-drug treatment

5.1 Drug Treatment

Table No. 1 Drug Treatment

Drug Treatment	Duration/ Remarks
SSRIs (Any One)	As anxiety disorders tend to be chronic, treatments
1. Escitalopram 10-20mg/day	should be given for long duration.
2. Paroxetine 12.5-25mg/day	
3. Mirtazapine 7.5-15mg/day	
4. Fluvoxamine 50-150mg/day (Preferred in OCD)	
5. Sertraline 25-50mg/day	
ТСА	
Clomipramine 25-75mg/day	
Anxiolytic Drugs (Any One)	
1. Clonazepam 0.5-1mg/day	
2. Lorazepam 1-2mg/day	
3. Diazepam 5-10mg/day	
β-Blocker	
Propranolol 10-40mg only in panic disorder	

5.2 Non Drug Treatments

Table No. 2 Non Drug Treatment

- Reassurance
- Supportive Therapy
- Relaxation Therapy
- Exposure and Response Prevention Therapy
- Cognitive Behavior Therapy
- Yoga and Exercises
- Self-help techniques

Comprehensive treatment including drugs and psychotherapies are most effective.

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7. SOMATIZATION DISORDER

1. Introduction

- Excessive thoughts, feelings or behavior related to the somatic symptoms or associated health concern
- Persistently high level of anxiety about health symptoms
- Excessive time and energy spent over these symptoms and their investigations and treatments

2. Signs and Symptoms

- Pain in different areas- Head / Back / Stomach / Joint etc
- GI symptoms like nausea, vomiting
- One pseudo-neurological condition (Paresis, loss of voice, loss of vision)
- Symptoms cannot be explained by a medical condition

3. Conversion Reaction

- Altered voluntary motor or sensory function with incompatibility between the symptom and recognized medical/neurological condition
- Causes significant distress in social, occupational and other important areas of functioning
- Possession Attack is an example of Conversion

Reaction.

4. Investigations

- Psychiatric assessment
- Laboratory Investigation
 - No diagnostic investigations available.
 - Complete Haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, Chest X-Ray (If Necessary)

5. Management

5.1 Antidepressants (Any One)

- a) Tab. Sertraline 25-200mg/day in divided doses
- b) Tab. Amitriptyline 25-100mg/day in divided doses
- c) Tab. Escitalopram 5-20mg/day in divided doses
- **5.2 Anxiolytics (Any One)**
- a) Tab. Lorazepam 2mg HS/SOS
- b) Tab. Clonazepam 0.5-1mg HS/SOS
- c) Tab. Diazepam 5-10mg HS/SOS
- **5.3 Counseling and Psychotherapy**-Supportive, Cognitive Behavioral, REBT
- 5.4 Stress Management and Life style changes.

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8. ORGANIC BRAIN SYNDROME

1. Introduction

• Primary biochemical or structural disturbances (damage) to the brain resulting in impairment of mental functions affecting memory, orientation and cognition

2. Types of organic brain syndrome

- Acute organic brain syndrome- Delirium
- Chronic organic brain syndrome- Dementia
- Other / Sub-acute organic brain syndrome-Amnestic syndrome.

2.1 Delirium

2.1.1. Definition

- It is a clinical syndrome of fluctuating global cognitive impairment of consciousness, attention and orientation
- Additional disturbances in memory, language and perception may occur
- Commonly occurs in hospitalized patient with history of major preexisting medical, neurological or surgical illnesses

2.1.2. Causative Factors

- Substance intoxication (alcohol, cannabis, opioid etc)
- Substance withdrawal
- Medicine induced
- Delirium due to preexisting medical conditions e.g. Hepatic Encephalopathy

2.1.3. Risk factors

Old age, preexisting dementia, very young, postoperative, burns victim, alcoholic and Benzodiazepine dependence

2.1.4. Signs and Symptoms

- Confusion
- Clouding of consciousness
- Visual illusion and hallucination
- Labile affect
- Disorientation to time, place, person
- Hyperactivity or hypoactivity

2.1.5. Investigations

Complete Haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, Chest X-Ray (If necessary).

2.1.6. Management

- Identify and treat the precipitating cause
- Provide environmental and supportive care
- Avoid sedation, but for severely agitated patients it may be necessary to induce sedation in order to facilitate investigations and treatment
- Regular clinical review, close vigilance

2.1.7. Medication

Important:

Watch for **level of consciousness** before giving injectable

Use single medication - Start low, go slow

- Haloperidol 1-4mg /day
- Lorazepam 0.5-1mg daily
- **Risperidone** 1mg daily
- Quetiapine 25mg 100mg daily (To be given in divided doses)

All anti-psychotics and sedatives to be tapered gradually, once symptoms are relieved

2.1.8. Refer to higher center

For counseling and psychotherapy

2.2. Dementia 2.2.1. Definition

It is a syndrome characterized by progressive, usually irreversible, global cognitive deficits primarily characterized by **memory impairment** and dysfunction in Activity of Daily Living (ADL)

2.2.2. Common Causes of Dementia

- Vascular dementia (20%)
- Substance Use Disorders (10%)
- Subdural hematoma
- Vitamin B12 deficiency
- Metabolic causes, Hypothyroidism

All Anti psychotics and Sedatives to be tapered gradually once symptoms are relieved

Refer to higher centre for Counseling and Psychotherapy

2.2.3. Signs and Symptoms:

- Memory impairment.
- **Dysphasia**–Deficiency of words, name generation
- Agnosia –Inability to recognize parts of body

Initially for 3 days, Review and titrate the dose

- Apraxia Inability to perform purposeful action and draw objects
- **Impaired executive function** Inability to plan and take action accordingly
- **Personality disintegration** Resulting in maladaptive behavior

2.2.4. Investigations:

- Complete Haemogram, Blood Sugar, Renal Function Test, Liver Function Test, Serum Electrolytes, Urine Routine, Chest X-Ray, Thyroid Function Test, HIV, Vit. B12, Folate levels, ECG, CT brain, MRI
- EEG abnormal depending upon the cause

2.2.5 Treatment and Management

a. Cognitive Enhancers -

(ACE inhibiter)

Oral Donepezil 2.5-10mg / day lifetime

b. NMDA antagonist-

Oral Memantine 5mg Associated Psychosis/agitation– Tab. Quetiapine 25mg, ½ - ½ - 1

OR

Tab. Risperidone 1mg/day, titrate the dose, regular follow up and then stabilize the dose for two years. Taper the dose after two years.

c. Associated Depression / Insomnia

- Tab. Sertraline 25mg/day, for 2 months and then titrate the dose
- Tab. Escitalopram 10mg/day, Lorazepam 0.5 mg/day

Anti-psychotics and Anti-depressants to be discontinued once symptoms relieved.

2.2.6. Treatment of co-morbid medical illnesses:

a. Supportive:

High doses of Multivitamins

b. Assistance:

For routine activities like bath, food etc.

Keep surroundings of patient safe and calm and well lit

c. Psychological support:

To both patient and caregiver

d. Functional management:

Maximize mobility and independence, assist communication

e. Social management:

Shelter homes, recreational activities, assistance in financial and legal matters

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9. DRUG ABUSE AND SUBSTANCE USE DISORDER

1. Introduction

- When substances like alcohol, caffeine, cannabis, inhalants, opioids, sedatives and hypnotics etc. are taken in excess quantities, they produce intense activation of the brain and disinhibition.
- Addiction- Repeated, compulsive use of a substance that continues in spite of negative

consequences in physical, social, legal aspects of life.

• Patients of addictions, especially of alcohol, are the most commonly treated cases in rural set up.

2. Common Substance Disorder

Condition	Sign and Symptoms	Management	Duration
Alcohol intoxication Caused by excessive consumption of Alcohol	Aggressive behavior, mood lability, slurred speech, incoordination, unsteady gait, nystagmus, impaired judgment.	No active treatment needs to be given. Manage symptomatically. If excited- Inj. Haloperidol 5mg + Inj. Phenergan 50mg IM. IV fluids SOS.	Till symptoms recover
Alcohol withdrawal Caused by Cessation or reduction of alcohol use that	Autonomic hyperactivity, tremulousness, insomnia, psychomotor agitation, nausea / vomiting transient	Inj. Thiamine 100mg and Multivitamin infusion.	5-7 doses on alternate days
has been heavy and prolonged earlier.	visual, tactile, auditory illusion or hallucinations.	Inj. Lorazepam 1 ampule BD/ HS. Shift to Tab. Lorazepam 1mg TDS /SOS.	2-3 days4-5 days and taper gradually
		IV Fluids	As necessary
Delirium Tremens It is a severe form of Withdrawal	Tremors, disorientation, confusion, hyperactivity, insomnia, illusions,	1. IV Inj. Thiamine 100mg /day in 100 ml NS.	10 days
Close monitoring of vita functions like pulse, Bl temperature, input and output is very essential.	hallucinations	 IV 5% Dextrose and DNS. Inj. Diazepam 5mg IM TDS or Inj. Lorazepam 2 mg IM/IV TDS. 	5 days
		4. Tab. Diazepam 5mg TDS or Tab. Lorazepam 6-12 mg/day in divided doses.	As needed

Table No. 1 Common Substance Disorders

Condition	Sign and Symptoms	Management	Duration
		 5. Inj. MVI (Multi Vitamin Injection) + 5% Dextrose drip. 6. If severe agitation or perceptual abnormality present Tab. Olanzapine 2.5-10mg or Tab. Risperidone 2-4mg per day in divided doses. 	6 days Till psychotic symptoms recover
Cannabis intoxication	Dryness of mouth, tachycardia, reduced motor skills, euphoria, sleepiness, apathy, laughter, paranoia, hallucinations.	 Supportive treatment and IV Fluids. Benzodiazepines eg. Clonazepam 0.5mg / Lorazepam 1mg TDS. Tab. Olanzapine 5mg HS 	As needed 5 days
Cannabis withdrawal	Anxiety, irritability, depressed mood, restlessness, insomnia, loose motions,	 Supportive treatment and IV Fluids Benzodiazepines eg. Clonazepam 0.5mg / Lorazepam 1mg TDS. 	3-5 days
Opioid withdrawal Heroin, Gard, Afeem, MD, Brown Sugar, Inj. Fortwin	Dysphoric mood, insomnia, muscle aches, nausea or vomiting, diarrhea, lacrimation or rhinorrhea yawning	1. Lorazepam 1-2 mg oral/I.M./I.V. for anxiety and decreased sleep.	2-3 times a day for 1st week
		2. Tab. Ibuprofen 400mg or Tab. Naproxen 250 – 500mg or Tab. Paracetamol TDS.	As needed
		 Tab. Clonidine 1mg can be given 2-3 times a day during detoxification. 	As needed
		4. IV fluids and Supportive Care.	As needed

Remark:

All patients with substance addiction should be motivated to enroll in de-addiction centres and to attend support groups like Alcohol Anonymous (AA) and Narcotic Anonymous (NA).

All other Substance Use Disorder patients should be treated symptomatically and referred to higher centre.

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10. INTELLECTUAL SUBNORMALITY

A state of mind with intellectual and functional deficits is commonly known as Mental Retardation.

1. Incidence

3 to 4 cases per 1000 population.

2. Etiology:

- i. Prenatal (1 in 1000 live birth) due to genetic, intrauterine viral infections, injuries, maternal metabolic disorders
- ii. Perinatal All causes leading to hypoxia during birth.

iii. Postnatal – CNS infections, trauma, uncontrolled epilepsy.

3. Types of Mental Retardation

- 1. Borderline
- 2. Mild
- 3. Moderate
- 4. Severe
- 5. Profound

Refer to the table for details and management.

Types	Bord	erline		Mild		Moderate		Severe		Profound
IQ range Salient Clinical features	Dull Normal 80 to 90 - Schola backw some but compl - 10th with e - And coach	Slow Learner 70 to 80 astic /ardness in subjects may ete Standard xtra years additional ing	-	50 to 70 Can tell right left laterality Can recognize coins Can use money Can do simple calculation Can tell relations Can pass 10th std. in selected subjects	-	35 to 50 May not be able to tell laterality May not recognize all coins May not tell all colors May not count beyond 10 May not be able to recognize relations like cousins, uncles	-	35 to 15 May not be able to name organs May not be able to recognize oColors oCoins oCount May not recognize own family members but recognize familiar faces	-	up to 15 Cannot feed self Cannot protect self
ADL (Activities of Daily Living) e.g. bath, food, grooming etc.	- Indepo - Can own	endent travel on	-	Independent Can use telephone Good social skills	-	May need help for grooming, bath May be able to move in village but not far away.	-	Needs assistance for bath, cleanliness but Can feed self	-	Needs to be fed, bathed, protected

Table No. 1 Mental Retardation Classifications

Types	Borderline	Mild	Moderate	Severe	Profound
Trainability	- Trainable - Can earn	- Trainable - Can earn	 Trainable for repetitive work under supervision May be able to earn as shepherd, helper 	 Non trainable So remains dependent for life long 	 Non trainable So remains dependent for life long
% Disability	<50 but eligible for subject selection.	> 40 %	> 50 %	> 75 %	100%

4. Management

- Anti-Psychotics in low doses for behavioral disturbances.
- Anti-Epileptics if the patient has co-morbid Epilepsy.
- ADL training.
- Vocational training.
- Caregiver education.

5. Special Guidance

Get IQ assessment certificate and Disability certificate from concerned Civil Hospital.

Enrollment in government employment facility for gainful employment

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11. CHILD AND ALDOLESCENT PSYCHIATRY

1. Introduction

- Child and adolescent form a major percentage of population
- Many psychiatric problems are present in this age group.
- Early detection and treatment of these conditions will prevent complications and significantly reduce the morbidity.
- Commonly seen disorders are behavior disorders, anxiety disorders and depressive disorders.

2. Presentation

The child usually presents as poor scholastic performance, disobedience, somatic pain.

2.1 Assessment:

- History to be taken from parents, school teacher and care taker as well as other colleague children.
- Child to be taken into confidence by talking to him alone.

A large number of these age group patients belong to the intellectual sub normality disorder.

childhood

2.2 Important disorders:

2.2.1 Psycho-developmental disorders

A. Learning Disorders or Dyslexia-

IQ of the child is normal but still he lags in scholastic performance due to specific learning disability i. e.

- Writing dyslexia wrong spellings and grammatical mistakes.
- Reading dyslexia unable to read sentences and words with proper pronunciations.
- Arithmetical dyslexia confusion in mathematical symbols e. g. +, -, x, 3, 6, i.e. geometrically reverse symbols, resulting into poor performance in solving and understanding mathematics.
- Global dyslexia all of above.

Detection of dyslexia is usually done by the school teacher and confirmation has to be done by government approved agency such as special units in teaching hospitals / medical colleges.

Certified patients can avail the provision of a writer and extended time to write a paper in 10th std. and 12th std. board examinations. Remedial teaching by specially trained teachers either one to one or in groups of a few children is very effective.

B. Pervasive Developmental Disorder of Childhood or Autism –

Such children do not mix or communicate verbally or emotionally even with their parents. They are selfengrossed in non-goal directed activities. Usually non-complaining and self-sufficient, odd motor movements, peculiar speech pattern, attracted by music. Those with normal IQ may excel in some arts like music, drawing.

Some such patients may need small dose antipsychotic if self-destructive.

Tab. Haloperidol 1.5 mg half tablet HS.

They need special training in one to one setup.

2.2.2 Psycho emotional disorders

A. Attention Deficit Hyper Activity Disorder

ADHD/ADD is not uncommon

Clinical symptoms -

- Inattention i.e. not able to mentally focus at a given task adequate enough to understand it or perform it. Easily distractible and so, forgetful
- Hyperactivity Always driven by some movements; cannot sit still; runs around; climbs windows; cupboards
- Impulsivity Acts before thinking; shifts from one activity to another; disruptive

Plenty of complaints from the school, neighbors. Most of the children show good response to psycho stimulant medicine.

- Tab. Methylphenidate 5 to 10mg once a day OR
- Cap. Atomoxetine 10 to 25mg once a day.
- **B.** Childhood Depression common.
- C. Nocturnal Enuresis due to environmental stress.
- Common
- Shows good response to Tab. Imipramine 25mg H.S.

2.2.3 Performance Anxiety, Phobia, Childhood Depression, Tension headache, Somatization disorder are also commonly seen among children facing stress at home or at Aashram-shala or in the school. They need to be managed with medication (discussed already) and along with counseling.

Clinical symptoms:

- Headache, pain in abdomen, migraine, loose motions, vomiting, exacerbations of asthma, fever, skin rash
- Irrational fear and phobia of animals, ghosts, darkness, school which ultimately resulting into avoidance to school
- Sleep disturbances, nightmares, enuresis
- Child remains withdrawn and introvert, not playing, crying spells, excessively shy, Temper Tantrums
- Small doses of antidepressant e.g. Tab. Imipramine 25 mg HS or Tab. Sertraline 25 mg ¹/₂ - 0 - 0 to 1-0-0 for a short period. Benzodiazepines should be avoided

2.2.4 Conduct disorder

In adolescents – Antisocial behaviors like violating rules, showing aggression, running away from home, lying, stealing, lack of guilt feeling.

Oppositional – disobedient, negativistic, temper tantrums, argumentative, stubborn, not yielding to rules.

Management:

- Small doses of antidepressant e.g. Tab. Imipramine 25mg, 0-0-1 or Tab. Sertraline 25 mg ¹/₂ - 0 - 0 to 1-0-0 for a short period
- Benzodiazepines should be avoided.
- Behavioral modification using positive / negative reinforcers
- Childhood psychoemotional disorders are usually secondary to environmental stress. So, modification of the stressful environment and improving child's coping capacity must be taken care to relieve symptoms. Medication is as mentioned, to be given only till symptoms active, and in dosages appropriate to weight.
- **Note:** All suspected cases need exhaustive evaluation by psychiatrist, so must be referred if not responding to above medication.

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12. PSYCHIATRIC EMERGENCIES

1. Suicidal Attempt

1.1 Causes:

- Severe depression associated with financial burden, crop failure, love affair failure
- Schizophrenia
- Drug induced psychoses like alcoholic hallucinations
- Impulsive act

1.2 Management:

- Immediate physical resuscitation
- Treatment for psychiatric illness ECT and medicines
- 24 hrs continuous supervision
- Do not isolate the patient
- Do not keep any sharp objects, ropes, blades near patient
- Do not allow patient to lock room/ bathroom from inside
- Counseling and psychotherapy

2. Psychotic excitement

2.1 Causes:

- Schizophrenia
- Mania
- Psychoactive drug abuse like charas, ganja, alcohol

2.2 Management:

- Inj. Haloperidol 10mg IM twice a day
- Inj. Promethazine 50mgIM twice a day till excitement controlled
- Restrain the patient to avoid injuries to self or others or destruction of property
- I.V. fluids if patient not eating / R. T. feeding to maintain hydration and nutrition
- Oral antipsychotics
- ECTs

3. Delirium

Management is given under topic of same name.

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4. Catatonic Stupor

Patient develops complete mental and physical inactivity, becomes statue like and non-communicative. Does not eat for many days and develops dehydration.

4.1 Causes:

- Depression
- Psychotic illness
- Organic brain disorder

4.2 Management:

- I.V. fluids
- Inj.Lorazepam1mg slowly BD till symptoms reduce
- Ryle's tube feeding
- ECTs
- Low doses of antipsychotic

5. Dystonic Reaction

Sustained muscles contractions causing twisting and repetitive movements or abnormal postures e.g. neck dystonia, tongue protrusion

5.1 Causes: -Use of antipsychotics like Haloperidol, Trifluoperazine, Risperidone, Olanzapine

5.2 Management:

- Taper off the anti-psychotic.
- Inj. Phenergan 50mg IM twice a day to be tapered off over next 4-5 days depending upon symptom relief.
- Tab. Benzhexol 2mg 1-1-1 till symptoms subside.
- I.V. fluids till patient able to eat orally.
- Change of antipsychotic by Psychiatrist.

Note: all patients of psychiatric emergencies need to be referred to psychiatrist as early as possible, once they are fit to travel. 4. Diagnostic and Statistical Manual of Mental disorders. 5th Edition. USA: American Psychiatrist's Association. 2013.

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13. PROCEDURE FOR ADMISSION TO A MENTAL HOSPITAL

1. Introduction:

All admissions to any government or private mental hospital are governed by the Mental Health Act-1987.

- The act provides guidance for admission and discharge procedures of civil and criminal patients.
- Management of property of the patients.
- Safeguarding human rights of the patients.

2. Admissions

- **2.1 Voluntary Boarder-** Patient himself signs the application requesting the superintendent of a mental hospital to allow him to stay in the hospital for treatment of his mental illness. Maximum stay 90 days.
- **2.2 U/s. 19 (Mental health Act)**-When patient not capable of signing above application due to his mental illness then, he is examined by 2 Psychiatrist independently and if found to need admission, can be admitted for maximum 90 days.
- **2.3 Detention Order-** A suspected mentally ill person can be admitted to a mental hospital for 10 days of observation period by the Detention Order of the Hon. District Civil Judge or the CP of the area.

This section usually used for admission of wandering mentally ill people suffering of schizophrenia, mental sub normality, delirium, dementia etc. also for criminal people behaving abnormally.

On certification after observation period, the stay in the hospital for treatment can be extended by the 'Reception Order' from the concerned (i.e. the district of the patient's residence) court.

2.4 Reception Order-

- By the concerned District Civil Court is issued only on confirmation of the mental illness and then, the patient can stay beyond 90 days till recovery.
- Such a patient even on discharge can extend his order with the 'leave of absence facility' for 60 days forever (i.e. he can be readmitted with the original R.O. any time in future without further legal procedures).

3. Documents required

Documents required for above legal procedures:

- 1. Address proof of the patient or his caretaker.
- 2. Photographs of the patient and his caretaker.
- 3. Undertaking to pay hospital charges by the caretaker unless exempted by court order or by provisions under the GR (1)
- 4. Medical Certificates by 2 Psychiatrist for U/s. 19 and for obtaining R. O. if patient admitted as D.O.
- 5. Medical Certificates in prescribed format by one Medical Officer in government service and by any Registered Medical Practitioner for application for R.O.

Note:

- i. All court procedures to be done during court timing i.e. between 10 am to 5:45 pm.
- ii. Patient and all documents to be presented to the court.
- iii. No legal procedures needed for admission at a Civil Hospital or a General Hospital, as the patient is not isolated from society. Hence, such admissions to be encouraged.

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6. ENT

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1. ACUTE OTITIS MEDIA (AOM)

1. Acute Otitis Media

1.1 Definition - An acute inflammatory condition of the middle ear space. Common in children – usually associated with fever.



Acute Otitis Media Fig 1.1 Congestion of eardrum



Fig 1.2 Diagramatic representation of middle ear with otitis media

1.2 Symptoms & Signs -

- i. Severe earache Fever, vomiting, may have convulsions
- ii. Mastoid tenderness & tinnitus,
- iii. Congestion of the eardrum (Tympanic membrane), Bulging Tympanic Membrane & loss of normal light reflex
- iv. Hearing Impairment (conductive deafness)
- v. Usually associated with chronic rhinitis & sinusitis, Nasal allergy, Cleft palate, tonsillitis, adenoids, measles
- vi. Young children may cry in agony, pull the affected ear or bang their heads

1.3 Treatment –

Level 1 (PHC) & Level 2 (Rural Hospital): Diagnosis & above-mentioned treatment with regular checkup. Non-improvement or deterioration, then refer to higher center.

- i. *Counselling-* Bed rest, advice mother about correct feeding techniques and plenty of fluids. No ear drops, ear mopping to be done only with good illumination facility, not to blow nose
- ii. *Antibiotics* (Ampicillin, Amoxicillin, 3rd generation Cephalosoprins like Cefixime, Cefpodoxime, Cotrimoxazole (Septran)
- iii. Analgesic (For adults: Tab. Diclofenac -50 mg TID x 5 days)

For children: Ibuprofen 200mg TID, Antipyretic (Paracetamol-325 mg TID x5days) infant &child over 3 months 5 to 10 mg/kg 3-4 times/day max. Daily dose 40 mg/kg/day)

- iv. Decongestant nasal drops (1% for adults and 0.5% for children), like Oxymetazoline (Nasivion) or Xylometazoline (Otrivin). This will reduce congestion
- v. Symptomatic Application of dry local heat to relieve pain

At Level 3-Distrtict Hospitals, Referral Hospital

- 1. Diagnosis, Investigations and Treatment of the case.
- 2. Referred case from Level 1 & Level 2, Reassessment & further treatment.

Treatment:

- If the tympanic membrane is bulging or if complication is suspected myringotomy should be done by ENT specialist under microscope with a coverage of antibiotics (Ampicillin/Amoxycillin (25mg/kg/day for pediatric patients for 5 days) / Adult dose 500mg TID/QID for 5 days/Amoxycillin + Clavulanic Acid 625 mg BD for 5 days.
- Decongestants Oxymetazoline nasal drops, 0.1% Nasal drops, 2-3 in each Nostril thrice daily/ Xylometazoline Nasal Drop 0.05% in children.

- Mastoid exploration may be required in cases of coalescent mastoiditis or other complication.
- Neurological / Pediatric Physician, Ophthalmologist Consultation may be required in unresolved cases & management in accordance with them.

2. Acute Suppurative Otitis Media (ASOM)

2.1. Definition -

An Acute inflammatory condition of middle ear cleft which includes Eustachian tubes, middle ear, Antrum (opening of mastoid air cells in middle ear) with pus discharge from external auditory canal due to bacterial infection or following exanthematous fevers. It is also called safe ear if infection limited to lower part of Eardrum.



Fig 1.3 Perforated ear drum with pus discharge.

2.2. Symptoms-

- i. Severe Pain, Fever
- ii. General malaise
- iii. Vomiting
- iv. Irritability
- v. Discharge from ears:
- vi. Mucopurulent discharge after dry mopping or suction. Tympanic membrane may show congestion, perforation and pulsatile discharge
- vii. Signs of upper respiratory tract infection like sinus, nasal & throat involvement.

2.3. Treatment-

2.3.1 Level 1 (at PHC level) & Level 2 (Rural Hospital)

- 1. Dry mopping, suction, clearance of ear discharge. A wick moistened with antibiotics may be inserted.
- 2. Treatment of other septic focus Eg. Acute Adenotonsillitis, Rhinosinusitis with antibiotics, anti inflammatory drugs and decongestant

- a. Systematic Antibiotics Ampicillin /Amoxycillin (25mg/kg/day for pediatric patients for 5 days / Adult dose 500mg QID for 5 days).
- b. Anti inflammatory /Anti pyretic Ibuprofen (200mg) with Paracetamol (325 mg) in divided doses for 5 days.
- c. Decongestant Oxymetazoline nasal drops, 0.1% nasal drops, 2-3 in each nostril thrice daily/ Xylometazoline Nasal Drop 0.05% in children.

2.3.2 Level 3 (Referral Hospital / Dist. Hospital)

- 1. Dry mopping/ Suction Clearance
- 2. Antibiotic (Ampicillin/Amoxycillin 500 mg thrice daily or, Amoxycillin + Clavulanic Acid 625 mg BD x 5 days). If needed parenteral antibiotics IV Cefotaxime 1 gm 12 hourly in adult for 5 days.
- 3. Anti inflammatory drugs (Tab Ibuprofen 200 mg + Paracetamol 325 mg BD)
- 4. Decongestant Oxymetazoline nasal drops, 0.1% nasal drops, 2-3 in each nostril thrice daily/ Xylometazoline Nasal Drop 0.05% in children.

2.3.3 Stages

a) Inactive stage -

Antibiotic not required and ear toileting may be done regularly Antibiotic ear drops (Ciprofloxacin ear drop 2 drops BID) may be needed.

b) Quiescent stage may not require any active treatment, as the ear is dry

c) Active stage:

In active case of the disease

Symptoms and Signs:

- 1. Ear Discharge Non-foul, smelling / intermittent / profuse and associated with upper respiratory tract infection.
- 2. Hearing Loss conductive in nature and gradually progressive
- 3. May be associated with tinnitus, Autophony
- 4. Otoscopic examination shows central perforation in the tympanic membrane with pulsatile discharge coming out to external canal.

Diagnosis is generally clinical. History with otological findings of central perforation is characteristic. Patient is advised to avoid risk factors in the form of avoiding excessive cold. Treatment is as mentioned above. Patient advised to keep ear dry and avoid water going into the ear.

Treatment:

- 1. Antibiotic treatment as per the culture and sensitivity reports.
- Regular ear cleaning and antibiotics ear drops in addition to oral/parental drugs. (Ampicillin /Amoxicillin 500mg TDS x 5 days or Amoxicillin + Clavulanic Acid 625 mg BD x 5 days. Parental drugs. Inj. Cefotaxime 1gm IV 12 hourly x 5days.

- 3. Treatment of the focus of infection Chronic adenoiditis Tonsillitis, Deviated nasal septum and any sinus pathology.
- 4. The corrective surgery: The myringoplasty / Tympanoplasty can be performed once the ear is dry with perforation with no disease activity.

Indications of Tympanoplasty - There should be

- (i) Well Sensorineural hearing preserved
- (ii) Eustachian tube should be patent
- (iii) Stapes mobile
- iv) Repeated attacks with no healing of disease.

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2. CHRONIC SUPPURATIVE OTITIS MEDIA (CSOM)

1. Definition:

It is a chronic inflammatory disease characterized by foul smelling ear discharge with cholesteatoma formation in atticoantral region. If attic involved, it is called as unsafe ear (infection may get routed to nearby structures leading to complications. The cholesteatoma is aggressive in nature and has got tendency of bone erosion leading to intracranial and intratemporal complications. It is also called as Atticoantral disease or unsafe type of CSOM.

2. Symptoms and signs:

- i. Usually scanty, intermittent, odourless mucoid or mucopurulent discharge. If associated infections foul smelling & continuous ear discharge, occasionally blood stained.
- ii. Gradually increasing hearing loss- conductive type.
- iii. Perforation Usually central
- iv. May have associated polyp.
- v. Patient may have:
 - a. Fever- Due to Acute Mastoiditis, Meningitis, lateral Sinus thrombosis (fever chill and rigors)
 - b. Headache- Due to Meningitis, Extradural abscess
 - c. Vertigo, Tinnitus, Vomiting Due to Labyrinthitis, Brain abscess, Increased intracranial tension, Meningitis
 - d. Facial weakness or paralysis
 - e. Post aural swelling due to Mastoiditis.



Fig 2.1 Showing Sub-total central perforation

3. Complications:

- i. Facial Nerve palsy
- ii. Labyrinthitis
- iii. Meningitis
- iv. Brain abscess Extradural, Subdural abscess
- v. Lateral sinus thrombosis
- vi. Mastoiditis
- vii. Petrositis

4. Investigations:

- i. Oto microscopy
- ii. Aural swab for culture and anti bacterial sensitivity test
- iii. X-ray mastoids / CT-scan / MRI to confirm the destructive pathology and spread of disease. Mastoid is usually sclerotic but may be pneumatic with clouding of air cells.
- iv. Pure tone Audiometry-It gives an assessment of degree of hearing loss and its type
- v. Investigations to assess the co-morbid conditions. (Biochemical & other Lab investigations)

5. Treatment:

5.1 - At Level 1 & Level 2:

- i. Ear toileting to remove all discharge and debris. Can be done by dry mopping or suction clearance under microscope. Irrigation with normal saline (Not forceful with syringe). If unresolved refer to higher centers.
- ii. Ear drops: Antibiotic eardrops containing Neomycin, Polymyxin, Chloromycetin or Gentamycin. Combination with steroids can be used to have local anti inflammatory effect.
- iii. Systemic antibiotics: Are useful in acute exacerbation in chronically infected ear. Otherwise role of antibiotics is limited.
- iv. Precautions: Patients are advised to keep water out of the ear during bathing, hair wash or swimming.

v. Treatment of contributory factors like nasal allergy, infected tonsils, adenoids

5.2 At Level 3:

- Cholesteatoma being aggressive and destructive in nature-definitive management is surgery- in the form of Tympanoplasty OR Modified Radical Mastoidectomy
- Aim: Removal of disease.

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Aims of modified radical surgery are

- 1. To make ear safe to patient by removing the cholesteatoma by surgery.
- 2. To make ear dry and prepare a self cleansing mastoid cavity
- 3. To prevent complications of CSOM.
- 4. Establishing hearing mechanism of the ear (Reconstruction).

3. ACUTE PAROTITIS -VIRAL/BACTERIAL

1. Mumps (Viral Parotitis)

Caused by Paramyxovirus. Disease is contracted by droplet infection and fomites. Children are commonly affected. Incubation period is 2 -3 weeks.

1.1. Symptoms & Sign-

- Parotid swelling Unilateral or Bilateral
- Pain over gland, ear pain
- Low-grade fever
- Arthralgia
- Malaise
- Headache



Fig 3.1 Showing unilateral and bilateral Parotitis

1.2. Complication -

- Orchitis with painful and tender testis,
- Aseptic Meningitis or Meningoencephalitis may occur with or without the salivary gland involvement,
- Unilateral sensorineural hearing loss,
- Other Complications-Thyroiditis, Myocarditis, Nephritis and Arthritis.

1.3. Investigation -

- Haemagglutination inhibition test
- Complement fixation test

1.4. Treatment -

- Supportive Measures Bed rest, adequate hydration, Antipyretics
- Prevention –MMR (Mumps, Measles, Rubella) vaccine at 12-15 months of age

2. Bacterial Parotitis

2.1. Definition –

Infection leading to Mechanical blockage of salivary duct (stasis of salivary secretions coupled with secondary infections), results in Bacterial Parotitis. It is prevalent in debilitated patients.

2.2. Symptoms & signs -

- Severe Pain
- Swelling over parotid
- Tenderness
- Indurations of gland
- Erythema of skin and oral mucosa
- Fever.

2.3. Management-

- Appropriate antibiotics Ampicillin / Amoxycillin 500 mg three to four times daily (25mg/kg/day for pediatric patients for 5 days)
- Adequate hydration
- Oral antiseptic gargles
- Good Oral hygiene
- Warm compression over gland
- Bimanual massage to milky purulent discharge / calculi from the gland
- Stone in the distal duct can be manually expressed & removed by pushing in mouth opening of gland
- Abscess drained out with a small incision.
- Organized abscess to be confirmed by Sialography, CT scan or USG. Needs open surgical intervention.
- Surgical drainage: Elevation of anteriorly based flap over super facial parotid fascia, Parallel to branches of facial nerve with placement of external drain.
- Rule out tumor mass by CT/MRI before surgical excision.



Fig 3.2 Parotid swelling in Acute Parotitis

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4. FURUNCLE

1. Definition:

Infection of root of hair follicle usually caused by Staph aureus. This infection occurs in cartilaginous ear canal.



Fig 4.1 Ear canal showing furuncle

2. Symptoms:

- Severe earache
- Movement of pinna painful
- Blocking sensation

- Tragal cartilage area tenderness
- Otoscopy- swelling in ear canal
- Post auricular lymphadenitis (swelling)

4. Investigation:

Hb, CBC, Blood sugar & culture & sensitivity test of discharge/pus.

5. Treatment:

5.1 Local:

Packing the ear with antibiotic ointment

5.2 Systemic:

- Antibiotics Ampicillin/Amoxicillin 500 mg thrice daily (25mg/kg/day) for pediatric patients for 5 days or Amoxiclav 625 mg BD in adult for 5 days
- Analgesics (Diclofenac 50mg BD for 5 Days)

In case of Recurrent furunculosis: rule out Diabetes

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5. OTO-MYCOSIS

1. Definition:

Fungal infection of ear canal caused by fungus like Candida albicans or Aspergillus Niger.

2. Symptoms & signs:

- Severe irritation and itching in the ear
- Severe earache,
- Whitish wet paper like discharge (Candida),
- Black spores (Aspergillus) or combination of both fungi.

3. Treatment:

• Removal of fungal debris by ear toileting

(mopping or suctioning)

- Antifungal ear drops (Clotrimazole / Flucanozole 2 drops TID for 5 Days)
- Analgesics Tab Diclofenac 50 mg bid for 5 days.
- Application of gentian violet, Treatment of diabetes (If associated)

4. Preventive:

Keep ear dry, Avoid swimming in polluted water, Care in rainy season.



Fig 5.1 Mixed fungal infections over the eardrum

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6. NECK SWELLING

Differential diagnosis depends on swelling occupying particular (Anterior & Posterior) triangle of neck.

1. Classification:

Cervical lymph nodes: classified into 7 groups

Level IA: submental

Level IB: submandibular

Level II: upper jugular

Level III: middle jugular

Level IV: lower jugular

Level V: posterior triangle

Level VI: pretracheal, prelaryngeal

Level VII: mediastinal



Fig 6.1. Showing classification of neck nodes according to levels

2. Symptoms and signs:

- Neck swelling-lymph node consistency:
 - 1. Stony hard: malignancy / metastatic
 - 2. Firm / rubbery: lymphoma

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- 3. Soft: infection /inflammation
- 4. Matted: Tuberculosis
- Pain:
- Present- acute inflammation
- o Absent-granulomatous disease or malignancy
- Size: rapid increase –malignancy

3. Diagnosis:

- Blood investigations: CBC, ESR, HIV
- Cytology: FNAC (Fine Needle Aspiration Cytology) Chest X-ray, Ultrasound
- CT/MRI
- Lymph node diagnostic excision biopsy

4. Treatment:

4.1 Antibiotics course for 7 to 14 days

Ampicillin / Amoxycillin 500mg thrice daily (25mg/kg/day for pediatric patients for 5 days) / Amoxyclav 625 BD 5 to 7 days and follow up evaluation

4.2 Surgical excision of neck swelling

4.3 Malignancy

Search for primary and treat accordingly, as neck nodes appears secondary (Metastatic) to primary malignant focus.

7. ATROPHIC RHINITIS

1. Definition:

Chronic inflammation of nose characterized by atrophy of nasal mucosa and turbinate bones. The nasal cavities become roomy and full of foulsmelling crusts.



Figure 7.1 Atrophic Rhinitis

2. Clinical Symptoms and signs:

- Commonly seen in females and starts around puberty.
- Foul smell form nose
- Feeling of Nasal Obstruction
- Formation of crusts in the nose
- Epistaxis may occur after crusts removal
- Roomy nasal cavity
- Anosmia
- Atrophy of Nasal turbinates, nasal mucosa looks pale and atrophied
- Saddle nose deformity (destruction of cartilage)
- Sometimes nasal septal perforation (erosion of cartilage)

3. Management:

3.1. Conservative:

- Aims at maintaining nasal hygiene by removal of crusts and decreasing putrefied smell due to erosion & destruction.
- Nasal irrigation and removal of crusts by retracted nasal douching with saline, glucose, and glycerin solution.
- Nasal douching The patient must be asked to douche the nose at least daily with Normal Saline or Alkaline solution [Sodium Bicarbonate – 1part, Sodium biborate 1 part and Sodium Chloride – 2 parts (Total 1 spoon powder) mixed in 280 ml of lukewarm water] thrice a day till crust disappears.
 - **25% glucose in glycerin**: After crusts removal nose painted with 25% glucose in glycerin twice a day for 1 month.
 - Potassium iodide can be prescribed orally to the patient in an attempt to increase the nasal secretion.
 - Placental extract injected submucosally in the nose has been attempted with varying degrees of success.

3.2. Surgical management:

- i. Sub mucous injections of paraffin
- ii. Teflon strips, and autogenous cartilage grafting
- iii. Wilson's operation Sub mucosal injection of 50% Teflon in glycerin paste.
- iv. Young's operation or Modified Young's operation This surgery aims at closure of one or both nasal cavities by plastic surgery in rare cases.
- v. Narrowing of nasal cavities by submucosal injection of Teflon paste, insertion of fat, cartilage or bone, or medial shift of the lateral wall of nose.

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8. NASAL MYASIS

Nasal Myasis commonly known as maggots in the nose. It is an infestation of the nose by maggots which are larvae of fly. Maggots are living foreign bodies in the nose.

1. Causes:

- Poor hygiene,
- Leprosy,
- Atrophic rhinitis
- Other nasal diseases which forms crust with foul purulent discharge and loss of nasal sensation attracts flies to the nose.

2. Clinical Features:

i. Maggots are seen crawling in the nose. They come out of the nose.



Fig 8.1 Adult house fly



Fig 8.2. Stages of Development of Housefly

- ii. Foul nasal discharge is present.
- iii. Cellulites of the nose and face may develop.
- iv. Fever and toxemia may be present.

3. Treatment:

- i. Removal of maggots by installing Liquid paraffin may be used to stifle the worm. Liquid paraffin blocks or chokes the lung of the maggots, thereby killing them. The killed worms can easily be removed.
- ii. Turpentine packs can be kept in the nose which irritate the maggots. Nasal saline douching is done for removal.
- iii. Surgical procedure: nasal endoscopic complete removal of the worms & infected parts of nose.



Fig 8.3 Maggots in nasal cavity

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9. FOREIGN BODIES IN NOSE

1. There are two types of foreign bodies in nose

- i. Non-living: Organic like seeds and grams. Inorganic like beads, paper or buttons.
- ii. Living: Maggots.
- iii. Iatrogenic: Cotton wool, pledgets or swabs may be left behind accidentally.
- iv. Commonest occurrence by mishaps in children, age group 0-5 years (self insertion)

2. Clinical Features:

- History of insertion of the foreign body may be available from patient/pain in the nose, bleeding.
- Blocking of the nose.
- Sneezing may be present.
- Unilateral Foul Smelling Blood Stained Discharge – one should suspecte old foreign body in the nose.





3. Investigation:

- i. Anterior Rhinoscopy gives site of foreign body in the nose.
- ii. Nasal endoscopy will locate exact site of the body.
- iii. Xray will detect only radio-opaque foreign body.

4. Treatment:

- (i) Generally Blowing the nose or induction sneezing may expel the foreign body.
- (ii) Removal of foreign body by Eustachian tube catheter, which is inserted behind the foreign body, without destructing nasal mucosa & touching F.B. otherwise bleeding may obscure visibility. It is performed under local anaesthesia.
- (iii) Endoscopy: Under General Anaesthesia



Fig. 9.2 Nasal polyp can be mistaken for foreign body in nose

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10. DEVIATED NASAL SEPTUM

1. Introduction:

The septal deformities are of the following types: -

- i. Deviations are smooth deflections which are upper or lower, anterior or posterior. i.e. Cshaped deviation.
- ii. Spurs are isolated thickenings at the junction of the bone and the cartilage.
- iii. Thickening may result from trauma leading to overriding of the cartilaginous fragments, which grow later in double layers.
- iv. Dislocation: The anterior edge of the septal cartilage may be displaced to one side causing the widening of the columella of the nose.
- v. S Shaped Deformity.
- vi. Bony posterior deformity of vomerine/ ethmoid bone.

2. Clinical features:

- Unilateral or bilateral blocking of the nose.
- Headache due to sinusitis.
- Recurrent Colds.
- Epistaxis.
- Anosmia.
- External deformity associated with deviation of the septum.

3. Investigation:

History, examination of the nose by anterior rhinoscopy & posterior rhinoscopy. X Ray PNS waters view / CT scan PNS.

4. Treatment:

Required only if the patient has persistent or recurrent symptoms due to the deviated septum or blockage of nose. Permanent relief is obtained by the Submucous Resection of the Nasal Septum or Septoplasty sugery.

4.1. Sub mucous resection of nasal septum:

Indications for sub mucous resection of nasal septum:

- Marked septal deviation occurring behind the vertical line passing between the nasal processes of the frontal and maxillary bones. This deviation must be the cause for the patient's symptoms.
- Closure of septal perforations.
- Source of grafting material.
- To obtain surgical access in hypophysectomy, and vidian neurectomy.

4.2. Septoplasty:

Indicated as tailor-made operation without disturbing nasal mucosa of other nasal fossa, where only obstructing part is removed. It can be associated with Rhinoplasty surgery.





Normal Nose

Type C deviated Nasal septum

Type S deviated Nasal septum

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11. NASAL OBSTRUCTION

1. Introduction:

The common causes of Nasal obstruction are DNS, Nasal polyps, Hypertrophied turbinate & Old nasal foreign body like rhinolith.

2. Clinical feature:

- Nasal obstruction, nasal stuffiness, watery discharge if associated with polyp.
- Partial or total loss of sense of smell
- Headache, sinusitis, epistaxis, external nasal deformity

3. Investigations:

- History, examination of the nose by anterior rhinoscopy, posterior rhinoscopy & functional nasal endoscopy (FESS).
- Cottles test used to check the nasal obstruction is due to abnormal nasal valve.



Fig 11.1 Hypertrophied turbinate

• Radiological: X Ray PNS / CT PNS region

4. Management:

4.1. Medical:

- Local nasal decongestants Xylometazoline 0.1 % 2 drops BID for 3 days,
- Local nasal steroid spray like Fluticasone once daily, Antibiotics (Ampicillin / Amoxicillin 500 mg thrice daily / Amoxicillin + Clavulanic Acid 625 mg BD (5 to 10 mg/kg of body weight for Paediatric patients for 5 days), Antihistaminic (Cetirizine 5mg BD for 7 days)

4.2. Surgical:

- Nasal polypectomy
- Turbinoplasty, Turbinectomy
- Septoplasty
- Endoscopic sinus surgery



Fig 11.2 Nasal polyp causing nasal obstruction

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12. TONSILLITIS

1. Acute tonsillitis

1.1 Definition:

It is acute infection/ inflammation of palatine tonsils.

1.2 Pathological types:

- a. Acute parenchymal tonsillitis
- b. Acute follicular tonsillitis
- c. Acute membranous tonsillitis

1.3. Clinical features:

1.3.1 Symptoms-

- Sore throat,
- Difficulty in swallowing,
- Fever, malaise, headache may be present,
- Pain may be referred to the ears.

1.3.2 Signs:

- Patient may look toxic and febrile.
- Tonsils are enlarged and congested; may be studded with follicles or membrane. (Scanty pus may come out)
- Anterior pillars are congested
- Tongue is coated
- Jugulodiagastric lymph nodes are enlarged and tender.

1.4 Treatment:

- Bed rest,
- Soft, bland & warm diet,
- Avoid cold, oily, spicy food,
- Antibiotics for 5-7 days: Amoxicillin 500mg-3 times/day, Amoxicillin with Clavulanic acid 625 mg twice/day x 5 days,
- Anti-inflammatory analgesics to reduce pain, fever and inflammation: Ibuprofen with Paracetamol (200+325) mg 2 times/day x 5 days
- Antiseptic gargles
- Multivitamins orally BD x 5days.

2. Chronic tonsillitis

2.1 Definition:

It is chronic inflammation/ infection of palatine tonsils.

It is characterized by recurrent episodes of acute tonsillitis. A history of 3-4 episodes of acute tonsillitis in a year should be labelled as "Chronic tonsillitis"

2.2. Clinical features:

2.2.1 Symptoms:

- Recurrent history of sore throat, Odynophagia and fever with symptom free interval between the two attacks
- Halitosis
- Change of taste
- Dry cough: Hawking due to irritation in throat.
- Failure to thrive: Seen in children

2.2.2 Signs:

- Tonsils may be enlarged (Parenchymatous type) or Fibrotic and small (Fibrotic type)
- Yellowish cheesy material is seen in tonsillar crypts which oozes out when the tonsils are pressed by tongue depressor.
- Enlarged Jugulodiagastric lymph nodes.
- Occasionally, yellowish cystic swelling (retention cyst) may be seen on the surface of tonsil.
- Redness of the anterior pillar

2.3 Investigation:

Hematological: Hb%, CBC, ESR, BT, CT, PT X-ray neck lateral view for adenoiditis & X-ray chest for preoperative evolution.

2.4 Treatment:

• Tonsillectomy by dissection method under General Anesthesia.



Fig. 12.1 Multiple follicles on the medial surface of both the tonsils and signs of acute inflammation



Fig. 12.2 Acute staphylococcal pseudomembranous tonsillitis with unilateral hypertrophy of the right tonsil

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13. ADENOIDITIS

1. Introduction:

Inflammation of adenoids is called Adenoiditis.

Adenoids are also called as nasopharyngeal tonsils. It is group of lymphoid tissue situated at the junction of the roof and posterior wall of nasopharynx. Adenoids usually atrophy by the age of 13-14 (puberty). However, in some patients it may be hypertrophied and produce symptoms, then treatment is required.

2. Symptoms:

Symptoms and signs occur due to the following:

2.1 Due to nasal obstruction-

- Mouth breathing
- Snoring
- Drooling of saliva
- Difficulty in breathing
- Change of voice
- Rhinitis and sinusitis

2.2 Due to Eustachian tube obstruction:

- Ear pain
- Retracted Tympanic Membrane and conductive hearing Loss
- Recurrent attacks of Acute Otitis Media & Chronic Otitis Media
- Serous Otitis Media in children

2.3 General symptoms and signs

- Recurrent sore throat
- Dysphagia
- Malnutrition
- Mental dullness
- Nocturnal enuresis



Fig 13.1 Adenoids

- Cervical lymphadenopathy
- 2.4 Facial features: (Adenoid Facies)
- Pinched Nose
- Mouth Breathing
- Dribbling of Saliva
- Flat nasal arch
- Malar hypoplasia
- High arched palate
- Crowded and protruding teeth
- Elongated face
- Dull' idiotic' appearance
- Loss of nasolabial fold
- Short protruding upper lip

3. Investigations:

X ray soft tissue nasopharynx: It shows enlarged adenoids and compression of airway.

4. Treatment:4.1 Medical Treatment

Acute infection is treated with:

- Antibiotics: Antibiotics of choice are-Amoxycillin 500mg TID, Amoxycillin with Clavulanic acid 625mg BD for 5 days.
- Anti-inflammatory agents like Ibuprofen 200mg BD for 5 days
- Nasal decongestants: Xylometazoline 0.05% (Adult: 0.1%)
- Antihistaminic: Levocetrizine 5 mg OD for 5 days

4.2 Surgical treatment:

Adenoidectomy under G.A.



Fig 13.2 Adenoid faces



Fig 13.3 Surgical removal of Adenoids

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14. PERITONSILLAR ABSCESS (QUINSY)

1. Introduction:

It is collection of pus in the peritonsillar space which is in between the capsule of tonsil and the superior constrictor muscle.

It usually follows acute tonsillitis.

2. Clinical Features:

These are generally due to septicemia and resemble any acute infection. Symptoms like fever, chills & rigors, general malaise, body aches, headache, nausea and constipation are present. Local symptoms like unilateral severe throat pain, odynophagia, muffled thick speech like hot potato voice. Foul breath, Ipsilateral earache, trismus.

3. Examination findings:

On examination tonsil, pillars & soft palate on the involved side are congested and swollen, uvula is swollen and edematous, bulging of the soft palate and anterior pillars. Mucopus may be seen covering the



Fig 14.1 Site of incision for drainage of peritonsillar abscess.

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Further reading

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tonsillar region. Cervical lymphadenopathy is commonly seen.

4. Investigation:

As mentioned in chronic tonsillectomy section.

5. Treatment:

5.1 Medical management:

IV antibiotics Inj. Ampicillin 500mg 12 hourly for 5 days, IV fluids, and analgesics like Paracetamol 500 mg bid for 5 days, good oral hygiene

5.2 Surgical Management:

- Incision and drainage of abscess (I & D)
- Interval tonsillectomy



Fig 14.2 Inflamed peritonsillar space with abscess

2. Indian Pharmacopoeia Commission. National Formulary of India (NFI). 4th ed. India: Government of India, Ministry of Health and Family Welfare; 2011: pp 314.



15. ACUTE PHARYNGITIS

1. Definition:

It is acute infection / inflammation of pharynx.

2. Etiology:

- a. Infective
- b. Associated with Tonsillitis, Rhinitis, Sinusitis.
- c. Associated with Gastro-oesophageal reflux.

3. Clinical features:

3.1. Symptoms-

- Sore throat or raw sensation in throat with halitosis
- Fever, malaise
- Odynophagia (Pain during swallowing)
- Dry, irritating cough

3.2. Signs-

• Catarrhal pharyngitis: Edema, congestion and inflammation of posterior pharyngeal wall and soft palate

- Ulcerative pharyngitis: Ulceration and sloughing of the posterior pharyngeal wall. It is rare and may occur in immune compromised patients.
- Granular pharyngitis: Red granulations over the posterior pharyngeal wall. (due to hypertrophied lymphoid tissue)

4. Treatment:

- Bed rest and improvement of oral hygiene.
- Soft bland diet with plenty of fluids
- Antibiotics: Antibiotics of choice are. Amoxicillin 500mg 3 times/day x 5, Amoxicillin with Clavulanic acid 625 BD x 5days.
- Antacids: Tab. Ranitidine 150 mg/Cap. Omperazole 20 mg daily in case of reflux.
- Anti-inflammatory agents like Diclofenac and Paracetamol (50+325mg) BD x 5days.
- Antiseptic Gargles like Betadine or Chlorhexidine.

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16. HOARSENESS OF VOICE

1. Introduction:

Hoarseness is term used to describe changes in voice quality. It results from changes in vocal cords.

A hoarse voice is rough and unpleasant, and it results from Lesions of the vocal cords. The causes of hoarseness may range from innocent Acute Laryngitis to Laryngeal Diphtheria and Malignancy.

2. Evaluation of hoarseness:

In the absence of upper respiratory tract infection any patient with hoarseness persisting for more than two weeks needs evaluation.

3. Physical examination:

- i. Thorough head and neck examination.
- ii. Assessment of hearing acuity.
- iii. Upper airway mucosa, tongue mobility. Cranial nerve examination.
- iv. Laryngeal examination.
 - a. Indirect laryngoscopy
 - b. Flexible nasolaryngoscopy
 - c. Strobolaryngoscopy
- v. Examination for signs of systemic disease such as Hypothyroidism or neurologic dysfunction such as Tremors or Parkinson's disease.

4. Treatment:

4.1. For habitual dysphonia

Treatment is by prevention by easing vocal abuse.

4.1.1 Things to do

- Rest to the voice
- Drink plenty of water

4.1.2 Things to avoid

- Tobacco
- Shouting
- Drinking alcohol
- Trying to talk excess when there is cold or laryngitis
- Whispering loudly or for very long
- Trying to change naturally speaking voice

4.1.3 Voice therapy

Maladaptive vocal habit and techniques are replaced with appropriate use of vocal mechanism. If not responded to this then surgery may be advised.

4.2. For organic causes

Treatment of the cause, mostly surgical interventions, followed by speech therapy

4.3. For psychogenic causes

Provide counselling on how to cope with emotional and stressful conditions. Give speech therapy to patient.

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17. STRIDOR

1. Definition:

It is an auditory manifestation of disordered respiratory functions due to airflow changes within larynx, trachea or bronchi.

It may be-

- 1. **Inspiratory** when obstruction is at the level of supraglottis and above.
- 2. **Expiratory** when obstruction is at the level bronchioles.
- 3. **Biphasic** i. e. Both during inspiration and expiration and obstruction is at the level of trachea or sub glottis.

2. Types:

- 1. Congenital
- 2. Acquired

3. General features of stridor:

- 1. Stridor is not a disease but a symptom or sign,
- 2. Noisy breathing which may be wheezing, crowing, whistling, croaking, sighing, rattling or snoring,
- 3. Cough,
- 4. Hoarseness,

- 5. May have difficulty in deglutition,
- 6. Change in voice

4. Investigation:

- Xray neck lateral view,
- X-ray chest,
- Direct laryngoscopy,
- Bronchoscopy &
- C.T.scan to find out cause & level of stridor.

5. Treatment:

5.1. General measures:

- 1. Humidified oxygen
- 2. Steroids (Prednisolone up to 40mg/day x 7 days.
- 3. Antibiotics- Ampicillin 500mg TDS x 5days
- 4. Intubation or tracheostomy is indicated if stridor is severe to relieve airways obstructions

5.2. Surgical

With the help of investigation, cause & level to be find out & depends upon that cause, surgical intervention is needed.

Bibliography

- 1. Dhingra PL, Dhingra S, editors. In: Diseases of ear, nose and throat & head and neck surgery. 6th ed. New Delhi: Elsevier; 2014.
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18. FACIAL PALSY (BELL'S PALSY)

1. Introduction:

It is an Idiopathic condition leading to peripheral facial paralysis or paresis, which is of acute onset.

High Risk cases of Bell's palsy are diabetics and pregnant women.

2. Causes:

Viral infection due to herpes simplex, herpes zoster or E.B. virus & other condition leading to external pressure on facial canal.

3. Clinical Features:

- Sudden onset
- Patient is unable to close his eyes completely
- Dribbling of saliva from angle of mouth (weakness of angular oris muscle)
- Face becomes asymmetrical
- Epiphora Tears flow down from the eye
- Pain in the ear
- Noise intolerance due to stapedial paralysis
- Loss of taste due to involvement of chorda tympani.

4. Investigation & Diagnosis:

• It requires careful history, complete otological and head and neck examination

- X-ray studies, blood test- CBC, ESR, blood sugar and serology
- Nerve conduction & excitability tests to monitor nerve degeneration
- Topodiagnosis localize site of lesion
- 5. Treatment:

5.1 General –

Reassurance, pain relief by analgesics (Tab. Diclofenac 50 mg BD for 7 days), eye care, physiotherapy, massage of facial muscles, use of eye pads.

Acyclovir - 200 - 400 mg 5 times a day for 5-7 days

5.2 Medical –

• Steroid therapy: Prednisolone 1 mg/kg per day in a divided doses for 5 days, if paralysis recovers with the dose of prednisolone, then it is tapered for next 5 days, if paralysis remains the same dose continued for next 10 days and thereafter tapered in next 5 days

Contraindications for Steroid therapy - Pregnancy, Diabetes, Hypertension, Peptic ulcer, Pulmonary Tuberculosis and Glaucoma.

- Ascorbic acid 500 mg Once a day for 7 days
- Multivitamins Vitamin B1, B6, B12 1 b.i.d for 7 days

5.3 Surgical Treatment -

- If there are no signs of recovery within 7 days, then opt for surgical intervention.
- Facial nerve decompression exploring fallopian canal where facial nerve is encased to release pressure.



Fig 18.1 Bell's Facial Palsy

- 1. Dhingra PL, Dhingra S, editors. In: Diseases of ear, nose and throat & head and neck surgery. 6th ed. New Delhi: Elsevier; 2014.
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19. SENSORINEURAL HEARING LOSS AND ITS MANAGEMENT

1. Sensorineural hearing loss (SNHL)

Result from lesions of the cochlea, VIII nerve & central auditory pathways. It may be present at birth (congenital) or start later in life (acquired).

1.1. The characteristics of sensorineural hearing loss are:

- A positive Renne test i.e. AC>BC. (Bone conduction impaired)
- Weber test lateralized to better ear (better hearing ear).
- ABC test: Bone conduction reduced on absolute bone conduction tests.
- More often involving high frequencies.
- No gap between air and bone conduction curve an audiometry.
- Loss may exceed 60 dB.
- Speech discrimination is poor.
- There is difficulty in hearing in the presence of noise.

1.2. Diagnosis:

- History
- Duration of illness sudden or slowly impairment or since birth.
- Severity of deafness
- Type of audiogram, whether loss is high frequency, low frequency, mild Frequency or flat type.
- Site of lesion i.e. cochlear, retro cochlear or central by BERA test.
- Brainstem Evoked Response Audiometry (BERA) helpful in profound sensorineural loss.

1.3. Management:

Early detection of SNHL is important as measures can be taken to stop its progress, reverse it or to start an early rehabilitation & interventions.

2. Specific forms of hearing loss

2.1 Inflammations of labyrinth

- i. Viral labyrinthitis
- ii. Bacterial
- iii. Syphilitic

2.2 Ototoxicity

- i. Amino glycoside antibiotics Neomycin, Kanamycin, Amikacin, Streptomycin, and Dihydrostreptomycin are cochleotoxic drugs with Selective destruction of outer hair cells
- ii. Patients particularly at risk are those having impaired renal function.
- iii. Diuretics- Frusemide and Ethacrynic acid
- iv. Salicylates Symptoms of salicylate ototoxicity are tinnitus and bilateral sensory neural loss. (Hearing loss particularly affecting higher frequencies.)
- v. Quinine Ototoxic symptoms due to Quinine are Tinnitus and Sensorineural hearing loss
- vi. Chloroquine Effect is similar to that of Quinine and permanent deafness can result.
- vii. Cytotoxic drugs Nitrogen mustard, Cisplatin and Carboplatin can cause cochlear damage.
- viii. Miscellaneous Erythromycin, Ampicillin, Chloramphenicol, Ibuprofen, Indomethacin, Phenylbutazone, Tetanus antitoxin, Propranolol, Propylthiouracil, Alcohol, Tobacco & Marijuana also cause damage to the inner ear
- ix. Topical Eardrop Chlorhexidine drop containing amino-glycoside antibiotics. E.g. Neomycin, Framycetin & Gentamycin.

2.3 Noise Trauma –

Hearing loss associated with exposure to noise has been well known in boiler operators, iron & coppersmiths & artillerymen. Lately, noise trauma has assumed greater significance because its being an occupational hazard, the compensation asked for & the responsibilities thrust upon the employer & the employee to conserve hearing. High level of sound pollution, use of speaker walls leads to Noise trauma.

TYPES

1) Acoustic Trauma-Very high sound decibel level for short duration

2) Noise Induced Hearing Loss- Over period of time

2.4 Sudden Hearing Loss

2.4.1 Definition:

It is defined as Sensory neural hearing loss that has developed over a period of hours or a few days. Loss may be partial or complete. Mostly it is unilateral. it may be accompanied by tinnitus or temporary spell of vertigo.

2.4.2 Etiology -

Most often the cause of sudden deafness remains obscure, in which case it is called the idiopathic variety.

- i. Infection Mumps, Herpes zoster, Meningitis, Encephalitis, Syphilis, Otitis media.
- ii. Trauma Head injury, Ear operations, Noise trauma, Barotraumas, Spontaneous rupture of cochlear membrane.

- iii. Vascular Hemorrhage, Embolism or Thrombosis of labyrinthine or Cochlear artery or their vasospasm.
- iv. Ear (otologic) Meniere's disease, Cogan's syndrome
- v. Toxic Ototoxic drugs.
- vi. Neoplastic Acoustic neuroma. Metastases in cerebellopontine angle, carcinomatous neuropathy.

2.4.3 Management -

- Bed rest.
- Steroid therapy- Prednisolone 40-60 mg in a single morning dose for one week & then tailed off in a period of 3 weeks.
- Inhalation of carbogen- (combination of CO2 & O2) It increases cochlear blood flow & improve oxygenation.
- Low molecular weight Dextran
- Hyperbaric Oxygen therapy

2.4.4 Prevention aspects-

- Immunization of child & ANC mothers.
- Avoidance of ototoxic drugs.
- Use of driving safety gadgets.
- Ear infection prevention & treatment in time
- Avoiding sound pollution areas

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20. VERTIGO

1. Definition:

Revolving around or spinning around.

2. Disorder of vestibular system cause vertigo & is divided into:

2.1 Peripheral-

Which involve vestibular end organs & their first order neurons. The cause lies in the internal ear or the 8^{th} nerve. They are responsible for 85% of all cases of vertigo.

2.2 Central –

Which involve central nervous system after the entrance of vestibular nerve in the brainstem and involve vestibulo-ocular, vestibulo-spinal and other central nerves system pathways.

2.2.1 Peripheral Vestibular Disorders

- i. Meniere's disease (endolymphatic hydrops) -It is characterized by trio of vertigo, fluctuating hearing loss, tinnitus with sense of pressure in the involved ear. Vertigo is of sudden onset, lasts for a few minutes to 24 hours
- ii. Benign paroxysmal positional vertigo (BPPV)

It is related to positional changes. Certain provoked position leads to attack of vertigo.

- iii. Vestibular neuronitis
- iv. Labyrinthitis
- v. Vestibulotoxic drugs Drugs like Gentamycin & Amikacin cause ototoxicity by damaging the hair cells of the inner ear.
- vi. Head Trauma Head injury may cause concussion of labyrinth, completely disrupt the bony labyrinth or VIII nerve or cause a perilymph fistula.
- vii. Perilymph fistula
- viii. Syphilis
- ix. Acoustic neuroma It has been classified in peripheral vestibular disorders as it arises from CN VIII within internal acoustic meatus

2.2.2 Central Vestibular Disorder

- i. Vertebrobasilar insufficiency
- ii. Posterior inferior cerebellar artery syndrome
- iii. Basilar migraine
- iv. Cerebellar disease- Cerebellum may be affected by hemorrhage infraction, infection or tumours.
- v. Multiple sclerosis It is a demyelinating disease affecting young adults. Vertigo & dizziness are common complaints.
- vi. Tumours of brainstem & floor of IV ventricle -Gliomas, astrocytomas may arise from pons & midbrain, epidermoid cysts or teratomas may arise from floor of IV ventricle.
- vii. Epilepsy
- viii. Cervical vertigo Vertigo may follow injuries of neck 7-10 days after the accident

3. Investigations:

3.1 Examination:

3.1.1 History:

History of dizziness, giddiness, deafness, tinnitus, otorrhoea, systemic & neurological symptoms. Past history of Giddiness, Injury, Diabetes, Hypertension, Medication and Surgery should be noted. History of tobacco and alcohol consumption should be noted.

3.1.2 Otoscopy:

- For attic perforation with cholesteatoma to be rule out.
- Blood pressure measurement, CVS & CNS exam includes tests for co-ordination, gait, past-pointing test, Romberg's test and corneal reflexes.

3.2 Ear Function Test:

- (i) Hearing test tuning forks test, Audiometry etc.
- (ii) Labyrinthine tests: Caloric test, Electronystagmography (ENG)

3.3 X-Ray

Mastoid, cervical spine and skull

3.4 Lab investigations

- CBC with Hb%
- Glucose tolerance test for Diabetes Mellitus
- Serum cholesterol
- V.D.R.L
- Thyroid Function tests.

3.5 Electrocardiogram

3.6 CT Scan & M.R.I. of the head

4. Management:

4.1 Medical:

- Reassurance regarding the nature of the disease, no jerks, avoidance of provocative posture
- Labyrinthine sedatives like Prochlorperazine 5mg BD for 7 days
- Vasodilators like Cinnarizine 25/75 mg BD for 7 days or vestibular sedatives like Betahistidine 8/16 mg BID for 7 days or for a long time.
- Vitamins like B1, B6 and B12 can be used in supplementary doses
- Diuretics (Frusemide 20/40Mg for 3 to 5 days) and low salt diet for reducing the tension of endolymph.

4.2. Labyrinthine Exercises are helpful in regaining the confidence of the patient:

4.2.1 The manoeuvre consists of five positions.

• *Position 1* - With the head turned **45 degrees**, the patient is made to lie down in head hanging

position. (Dix Hallpike manoeuvre). It will cause vertigo and nystagmus. Wait till vertigo and nystagmus subside.

- *Position 2* Head is now turned so that affected ear is up.
- *Position 3* The whole body and head are now rotated away from the affected ear to a lateral recumbent position in a facedown position.
- *Position 4* Patient is now brought in a sitting position with head still turned to the unaffected side by **45 degrees.**
- *Position 5-* the head is now turned forward and chin brought down **20 degrees.**

There should be a pause at each position till there is no nystagmus or there is slowing of nystagmus, before changing to the next position. After manoeuvre is complete, patient should maintain an upright position for 48 hours. Eighty percent of the patients will be cured by a single manoeuvre. If the patients remain symptomatic, the manoeuvre can be repeated. A bone vibrator placed on the mastoid bone helps to loosen the debris.

4.3 General:

Smoking and alcohol should be avoided.

4.4 Surgical:

- i. Operations, which reduce the tension of endolymph
- ii. Vestibular nerve section.
- iii. Labyrinthectomy

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21. FRACTURE NASAL BONE

1. Introduction:

Two, all pieces of nasal bone present on nasal bridge which usually get fractured in Head and Facial trauma, assault, RTA, sport or industrial accident.

2. Symptoms and Signs:

- History of Trauma
- Nasal deformity with swelling.
- Pain
- Bleeding nose
- Nasal blockage
- Discoloration of nasal skin

3. Signs:

- Epistaxis (active or clots in nasal cavity)
- Crepitus on palpation over nasal bone area.
- Deformity
- Oedema on and around nose
- Nasal obstruction.
- Associated traumatic injury
- Black eye

4. Investigation:

- Imaging study like X-ray nose lateral view, X-ray PNS, CT scan.
- Anterior & Posterior rhinoscopy
- Routine Biochemical & Serological Laboratory Test.
- Preinjury photographs if available.

5. Management:

5.1. Medical:

- Head up position, cold compression to reduce oedema.
- Suturing of open wound
- Anti inflammatory drug (Tab. Diclofenac 50 mg BD x 7 days).
- Antibiotics (Amoxycillin 500mg TID x 5 days or Parenteral Amoxyclav 1.2gm I.V. 12 hourly)

5.2. Surgical:

- Closed reduction under General Anesthesia with septal elevation.
- Open reduction with wiring in case of multiple fractures.

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22. FRACTURE MAXILLA

1. Introduction:

Maxillary bones are present on either side of nose & are part of skull bone. Fracture maxillary bone occurs in Head and Facial trauma, assault, RTA, sport or industrial accident.

2. Symptoms:

- History of trauma
- Nasal deformity with swelling.
- Pain
- Bleeding nose
- Nasal blockage
- Discoloration of nasal skin

3. Signs:

- Epistaxis (active or clots in nasal cavity)
- Crepitus on palpation over nasal bone area.
- Deformity
- Oedema on and around nose
- Nasal obstruction.
- Associated traumatic injury
- Black eye

4. Investigation:

- Imaging study like X-ray nose lateral view, X-ray PNS, CT scan PNS.
- Anterior & Posterior rhinoscopy
- Routine Biochemical & Serological Laboratory Test.

5. Types of Maxillary fracture:

- Le fort I Floating fracture
- Le fort II Midlevel / sub zygomatic fracture
- Le fort III Craniofacial fracture at supra zygomatic, zygomaticomaxillary complex fracture or Tripod fracture.

6. Management:

6.1 Emergency care -

- Airway maintenance,
- Control of oral & nasal bleeding.

6.2 Medical Management -

- Includes emergency care & stabilization of general condition.
- Anti inflammatory drug (Tab. Diclofenac 500mg BD x 7 days.
- Antibiotics (Amoxicillin 500mg t.i.d. x 5 days or Parenteral Amoxiclav 1.2 gm IV 12 hourly.

6.3 Surgical-

Multi disciplinary approach with involvement of ENT surgeon, Maxillofacial surgeon & Neurosurgeon may be needed depending upon types & severity of fracture.



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23. PENETRATING NECK INJURY

1. Causes:

- Gunshot
- Stab
- Penetrating shrapnel with or without speed.

2. Levels:

- Includes skin and platysma.
- Includes important structures like major vessels (carotid & jugular vein), trachea, esophagus, cranial & other nerves.
- Involvement of spine &vertebrae.

3. Look for

- Dysphasia,
- Hoarseness of voice,
- Oropharyngeal bleeding,
- Neurological deficit,
- Hypotension,
- Sub cutaneous emphysema
- Air bubbles through wound & tracheal area.

4. Immediate Treatment:

- Securing Airway by neck stabilization & endotracheal intubation if necessary
- Maintaining Breathing
- Maintaining Circulation
- This requires immediate intervention.

5. Investigations: any of these, which are relevant to the case

- CT neck
- Angiography
- Laryngoscopy
- Bronchoscopy
- GI scopy

6. Treatment:

- Emergency medical intervention to maintain BP through IV fluid, airway maintenance through plastic airway in mouth or intubation.
- Surgical intervention includes Hemostasis of major bleeding vessels & operative management for respective traumatized organ & treatment of head injury component (If associated with).





from the wound site –

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24. BLUNT EXTERNAL LARYNGEAL TRAUMA (FLOW CHART)



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25.EPISTAXIS

1. Definition:

It is defined as bleeding from inside the nose.

2. Types:

2.1 Anterior–

More common, site of bleeding is littles area, in children and young adults.

2.2 Posterior-

Less common, site of bleeding is posterior superior area, seen in adults.

3. Causes:

3.1 Local –

Injuries to nose, Vascular malformation, Fracture of maxilla, Fracture nasal bone, Nasal surgery, Acute sinusitis, Rhinitis, Nasal diphtheria, Atrophic rhinitis, Rhinitis sicca, Tuberculosis, Syphilis, Rhinosporidiosis, Foreign body in nose like maggots, Deviated nasal septum.

3.2 Nasopharyngeal -

Adenoids, Juvenile angiofibroma, Malignant tumors

3.3 General causes -

Hypertension, Leukemia, Hemophilia, Aplastic anemia, Cirrhosis of liver, Nephritis, patient with Anti coagulant therapy, Influenza, Chickenpox, Malaria.

4. Management:

4.1. Enquire about

• Mode of onset

- Duration & frequency
- Site, Side, Type, Any medical disorder
- Drug intake

4.2. First aid -

Littles area compression- Pinch the nose with thumb and index finger for 2 min. & ask the patient to breath orally.

4.3 Medical/Surgical Management

- In Anterior Epistaxis anesthetize the bleeding point and cauterize with bead of silver nitrate or coagulate with electro cautery
- Anterior Nasal packing
- Posterior Nasal Packing
- Endoscopic cauterization
- If above measures fails, then ligation of vessels.
- External carotid- after the origin of superior thyroid artery
- Maxillary artery approached through Caldwell- Luc operation.
- Ethmoid artery ligation

4.4 General Measures

- Sit up with back rest
- Record blood loss in spitting & vomiting
- Reassure
- Mild sedation
- Check vital signs
- Maintain hemodynamic-blood transfusion if required
- Intermittent oxygenation
- Investigate & treat

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26. PREMALIGNANT LESIONS OF THE ORAL CAVITY

1. Introduction:

Oral cavity cancer accounts for approximately 3% of all malignancies and is a significant worldwide health problem. Most oral malignancies occur as squamous cell carcinomas (SCCs); Most of the oral SCCs develop from premalignant conditions of the oral cavity. The early detection of cancer is of critical importance because survival rates markedly improve when the oral lesion is identified at an early stage (early intervention)

2. Types

2.1 Leukoplakia

White patch or plaque

2.2 Erythroplakia

A fiery red patch with a soft, velvety texture

2.3 Proliferative verrucous leukoplakia

Proliferative vertucous leukoplakia (PVL) is a unique form of aggressive disease.

2.4 Palatal lesion of reverse smokers

The palatal lesion of reverse smokers is unique to individuals who place the lit end of a cigarette inside the mouth.

2.5 Oral submucousfibrosis (SMF)

It is a chronic progressive condition. In which normal mucosa with blood supply is replaced by fibrous tissue leading to narrow mouth opening.

2.6 Other forms:

Lichen planus, discoid lupus erythematous, and epidermolysis bullosa.

3. Work-up and the Early Detection of Oral Cancer:

The standard criteria for diagnosis and identification of oral lesions is histopathology analysis via the procurement of a tissue sample by surgical biopsy.

3.1 Oral cavity examination:

- Lip: Inspect and palpate inner and outer surfaces of the upper and lower lip.
- Buccal mucosa
- o Inspect and palpate buccal mucosa and cheek.
- Inspect and palpate parotid duct to express saliva.
- Gingival and alveolar ridge: Inspect and palpate gingival and alveolar ridge on facial and lingual aspects.
- Tongue
- Inspect and palpate dorsal and ventral surfaces with accompanying retraction of the tongue with gauze.
- Inspect and palpate lateral borders from anterior to posterior with manual retraction.
- Floor of the mouth
 - Inspect and palpate floor of the mouth.
 - Inspect and palpate submandibular ducts to express saliva.
- Hard palate: Inspect and palpate.
- Soft palate and oropharynx: Depress the dorsal surface of the tongue and inspect soft palate and anterior oropharynx.
- Salivary glands: Palpate the parotid, submandibular, sublingual, and minor salivary glands. Ensure clear salivary flow.

Extra oral

- Inspect the head and neck.
- Palpate cervical lymph nodes and salivary glands

4. Investigations:

- Biopsy of the lesion and histopathological diagnosis is the gold standard investigation.
- Oral cytology

Oral cytology describes a diagnostic technique used to sample oral tissue for histopathogical analysis. To obtain a tissue sample, the clinician applies a stiff brush to the oral mucosa with enough pressure to induce pinpoint bleeding, which ensures a full-thickness or trans-epithelial tissue sample. These cellular samples can then be analyzed by a variety of unique diagnostic measures including cytomorphometry, DNA cytometry, and immunocytochemical analysis.



Fig. 26.1 Leukoplakia of the buccal mucosa



Fig 26.2 Submucous fibrosis involving the retromolar trigone anterio tonsilar pillars and soft palate on both sides



Fig 26.3 Leucoplakia of tongue



Fig 26.4 Leucoplakia of Lips

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27. ACUTE UPPER AIRWAY OBSTRUCTION

1. Introduction:

Upper airway consists of nose, nasopharynx, pharynx, larynx and trachea up to carina. Different pathologies in this area can lead to acute airway obstruction.

Obstruction here may be partial or complete. This is also called as extra thoracic airway. The airway collapses during inspiration and dilates during expiration.

2. Clinical presentation

May be partial or complete

2.1. Complete upper air way obstruction:

- Universal chocking sign: patient is unable to breath, speak or cough and may hold the throat between the thumb and finger.
- Vigorous attempts at respiration with intercostals and supraclavicular retraction.
- Raised Heart rate and raised blood pressure, Patient becomes rapidly cyanosed.
- This is followed by diminished respiratory efforts, loss of consciousness, bradycardia, and hypotension.
- Cardiac arrest.
- Death is inevitable if obstruction is not relieved within 3-5 minutes.

2.2. Partial airway obstruction

- May be stable or their may be progressive deterioration
- Signs and symptoms may be mild but as they worsen patient develops inspiratory stridor,

dysphonia, aphonia, chocking, drooling gagging,

- Signs of hypoxemia and hypercarbia. Anxiety, confusion, lethargy and cyanosis may be present as the obstruction worsen
- Powerful inspiratory efforts against an obstruction may produce ecchymosis and subcutaneous emphysema.

3. Management:

3.1. General measures:

- Reverse hypoxia- 100% Oxygen or as close as possible.
- IV access as soon as possible.
- Continuous monitoring and observation.

3.2 Airway management techniques

3.2.1 Airway manoeuvres

- i. Jaw thrust,
- ii. Endotracheal intubation,
- iii. Oropharyngeal or nasopharyngeal airway may be useful in the unconscious patient,
- iv. If the patient is not immediately intubated, then give coma position i. e. Semi prone, slightly head down.

3.2.2 Endotracheal intubation.

Surgical airway:

- Indicated when endotracheal intubation not possible.
- Percutaneous transtracheal jet ventilation.
- Cricothyroidectomy.
- Formal tracheostomy under local anaesthesia may be tried before emergency tracheostomy.
- Emergency tracheostomy rarely required.

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28. DEAF MUTISM

1. Definition:

Deaf Mutism is the inability to acquire speech due to profound or high degree hearing loss (congenital or early acquired childhood) of sensory neural type in both ear.

2. Causes:

2.1 Prenatal

- i. Genetic Defect
- ii. Maternal Infection
- iii. Drugs During Pregnancy
 - 1. Amino glycosides
 - 2. Quinine
 - 3. Chloroquine
- iv. Radiation to Mother in first trimester
- v. Other factors: Nutritional deficiency, diabetes, toxaemia & thyroid deficiency

2.2 Perinatal

- i. Anoxia
- ii. Prematurity
- iii. Birth injuries
- iv. Neonatal Jaundice
- v. Ototoxic drugs

2.3 Post natal -

- i. Genetic
- ii. Non-Genetic

3. Identification of Deafness:

- History
- Responses of hearing in child by investigations

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4. Investigation:

- Brainstem Evoked Response Audiometry (BERA) It is of value to find out the threshold of hearing in infants, particularly the high risk group & in the diagnosis of retro- cochlear pathology.
- Oto acoustic emission (OAE)
- Audiometric examination
- Free field audiometry
- Visual reinforce audiometry
- Play audiometry
- Electrocochleography
- Impedance audiometry
- High-resolution CT scan

5. Treatment:

- Bilateral hearing aid
- Surgical interventions including Cochlear implant
- Auditory training
- Language communication

6. Referral:

If deaf mutism is suspected after investigation, the child should be referred to a higher centre for Cochlear implant.

7. Prevention:

Vaccination to child, avoidance of ototoxic drugs, regular antenatal checkup & care.





7. Ophthalmology

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1. LID SWELLING

1. Stye (Hordeolum Externum)

1.1 Definition

It is an acute suppurative inflammation of the Zeis / Moll glands.





1.2 Etiology

Bacterial- Staphylococcus

1.3 Symptoms

- Pain
- Lid swelling
- Watering

1.4 Sign

A painful swelling at the lid margin.

1.5 Treatment

- Hot fomentation 2-3 times per day.
- Ciprofloxacin 0.3 % (Antibiotic) eye drops. 1-2 drops six times per day for 5 days.
- At night time Ciprofloxacin eye ointment for 7 days.
- Tablet Ciprofloxacin 500mg (systemic antibiotics) twice daily and Tab. Diclofenac Sodium 50mg bid (anti-inflammatory) for three or four days.
- When pus points out it should be let out by pulling the affected eye lash Epilation.

2. Hordeolum Internum

2.1 Definition

It is an acute suppurative inflammation of the Meibomian glands.



Figure 1.2 Hordeolum Internum

2.2 Symptoms

Pain, lid swelling and watering.

2.3 Sign

A painful swelling at the lid margin.

2.4 Treatment

- Ciprofloxacin 0.3 % eye drops six times per day for 5 days
- Tablet Ciprofloxacin 500mg (Systemic antibiotics) twice daily and Tab. Diclofenac sodium 50mg BD (anti-inflammatory) for three days.
- The pus should be drained by incising it.

3. Chalazion

3.1 Definition

It is a chronic granulomatous inflammation of the Meibomian gland.



Figure 1.3 Chalazion

3.2 Etiology

- Uncontrolled Diabetes Mellitus in adults.
- Refractive errors.

3.3 Symptoms

Swelling of the lid and Watering.

3.4 Sign

Painless nodular swelling away from the lid margin.

3.5 Course

- Spontaneous resolution.
- If secondarily infected, then it is called as Internal Hordeolum.
- It may burst through the conjunctiva.
- Very rarely a malignant change may occur (Meibomian carcinoma) especially in old age with a history of recurrence.

3.6 Treatment

- Incision and curettage under Proparacaine 0.5 % eyedrop (local anesthesia.)
- In Marginal chalazion intralesional injection of Triamcinolone 40mg/ml (Depot steroids): 0.1 to 0.2 ml once may be helpful.
- Correction of refractive errors, if any.

4. Blepharitis 4.1 Definition

Inflammation of the lid margin is called as Blepharitis.



Figure 1.4 Blepharitis

4.2 Causes

- Seborrhoeic Blepharitis due to dandruff at the base of eye lashes.
- Ulcerative Blepharitis because of infection by Staph. Epidermidis / Streptococci.

4.3 Symptoms

Itching / rubbing of eyelids.

4.4 Signs

Edema of lid margin, dandruff /ulcer at the root of eye lashes.

4.5 Treatment

- Clean the lid margin with warm water 2-3 times a day.
- Chloramphenicol- Polymixin B combination (Ocupol) eye oint. 3 times a day for 3weeks.

If a Chalazion recurs soon after removal or is rapidly growing in size or ulcerates, a biopsy must be done to rule out any malignancy.

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2. DACRYOCYSTITIS

1. Acute Dacryocystitis

1.1 Definition:

Acute Dacryocystitis is a suppurative infection in the lacrimal sac caused by a blocked nasolacrimal duct. (NLD)



Figure 2.1 Acute Dacryocystitis

1.2 Pathogenesis:

When the opening of NLD is blocked, tears and mucous will remain within sac, forming a deposit that gets easily infected.

1.3 Microorganism:

Most commonly Staphylococcal infection and Streptococcal infection.

1.4 Stages:

- i. Stage of cellulitis.
- ii. Stage of lacrimal abscess.
- iii. Stage of fistula formation.

1.5 Clinical features:

Acute onset, painful, red, hot, firm swelling over lacrimal sac area.

In severe cases there may be a pus point, which may burst

There is also Tear Lake, fullness, conjunctival congestion (i.e. watering and redness of eyes.)

1.6 Associated symptoms:

Fever, malaise.

1.7 Treatment:

i. Tab. Ciprofloxacin 500mg twice daily initially for 5 days.

- ii. Tab. Ibuprofen 3 times a day (Non-Steroidal Anti Inflammatory Drug).
- iii. Hot fomentation 3 times a day.
- iv. Ciprofloxacin 0.3% eye drop one drop four times a day and Ciprofloxacin eye ointment at night time for 5 days.
- v. If there is a pus point, drainage of pus is required.

NO SYRINGING IS DONE IN ACUTE CASES OF DACRYOCYSTITIS

1.8 Complications:

Acute conjunctivitis, corneal ulcers, lid abscess, orbital cellulitis, cavernous sinus thrombosis.

1.9 Referral:

When acute attack of Dacryocystitis is subsided, patient is referred to higher centre for sac surgery (DCR) and Fistulectomy, if required.

2. Chronic Dacryocystitis: 2.1 Definition:

Chronic low grade inflammation of lacrimal sac is called as Chronic Dacryocystitis.



Figure 2.2 Chronic Dacryocystitis **2.2 Pathogenesis:**

Stricture formed due to chronic inflammation.

Obstruction of lower end of nasal nasolacrimal duct caused by polyp or hypertrophied inferior turbinate.

2.3 Clinical Picture:

- Essential symptom is watering of eyes.
- There may be swelling at lacrimal sac area (Mucocele).

• Regurgitation test (on pressure over medial canthus of eye i.e. over sac region) is positive (Muco-pus, pus).

2.4 Treatment:

Topical Ciprofloxacin 0.3% eye drops four times a day. Syringing with Ciprofloxacin 0.3% daily once for 5 days.

2.5 Complications:

Chronic intractable conjunctivitis, corneal ulcer, ectropion.

2.6 Referral:

For sac surgery (Dacryocystorhinostomy or Dacryocystectomy).

2.4 Treatment:

Topical Ciprofloxacin 0.3% eye drops four times a day. Syringing with Ciprofloxacin 0.3% daily once for 5 days.

2.5 Complications:

Chronic intractable conjunctivitis, corneal ulcer, ectropion.

2.6 Referral:

For sac surgery (Dacryocystorhinostomy or Dacryocystectomy).

3. Congenital Dacryocystitis 3.1 Definition:

Non canalization of nasolacrimal duct leading to blockage of drainage passage may lead to Congenital Dacryocystitis.



Figure 2.3 Congenital Dacryocystitis

3.2 Symptoms:

Baby is brought with complaints of watering from one/both eyes.

3.3 Signs:

Swelling at sac region, discharge on pressure over swelling.

3.4 Treatment:

Moxifloxacin 0.5 % eye drops 4 times a day.

- Crigler Massage over sac region.
- If the block persists for more than 6 months probing of nasolacrimal duct is done by ophthalmologist under general anaesthesia.

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3. CONJUNCTIVITIS

1. Definition:

Inflammation of conjunctiva is called as conjunctivitis.

2. Types

It can be Infectious or Allergic.

Infectious conjunctivitis can be bacterial or viral.

2.1. Mucopurulent/purulent (Bacterial) conjunctivitis

2.1.1 Definition:

Acute purulent inflammation of conjunctiva.



Figure 3.1 Conjunctivitis

2.1.2 Causes:

Staphylococci, Streptococci, Pneumococci, Gonococci etc.

2.1.3 Symptoms:

Acute redness of eyes, grittiness of eyes with mucopurulent purulent discharge.

2.1.4 Signs:

Conjunctival congestion, mucopurulent / purulent discharge (no diminution of vision, photophobia), matting of eye lashes due to discharge.

2.1.5 Treatment:

- Moxifloxacin 0.5 % eye drops two hourly for 7 days.
- Chloramphenicol 1% eye oint. At bed time for 7 days.
- Dark goggles.

2.1.6 To prevent spread of infection:

- Frequent hand washing.
- Avoid crowded places.
- Avoid sharing of handkerchief, towels, napkins, pillows etc.

2.2 Viral conjunctivitis

2.2.1 Definition:

Acute conjunctival inflammation caused by viruses.



Figure 3.2 Viral Conjunctivitis

2.2.2 Causative organisms:

Herpes simplex, Herpes zoster, adenovirus etc.

2.2.3 Symptoms:

Acute redness of eyes, grittiness of eyes, watering from eyes, photophobia.

2.2.4 Signs:

Local: Conjunctival congestion, follicular hypertrophy with pre-auricular lymphadenopathy.

Systemic: Patient may have fever, Upper respiratory tract infection.

2.2.5 Treatment:

Usually self-limiting course.

- Acyclovir 3% eye oint. 5 times a day for 14 days with,
- Prophylactic Moxifloxacin 0.5% eye drops four times a day for 7 days with,
- Fluorometholone 0.02% eye drops four times a day for 7 days.
- In case of Herpes Zoster Ophthalmicus refer to Ophthalmologist immediately.

To prevent the spread of infection

- Frequent hand washing to be done.
- Avoid crowded places.
- Avoid sharing of handkerchief, towels, napkins, pillows etc.
- AVOID USE OF STEROIDS (BOTH TOPICAL AND SYSTEMIC).

2.3. Allergic conjunctivitis

2.3.1 Definition: Conjunctivitis caused as allergic reaction to pollens, dust, etc.



Figure 3.3 Allergic Conjunctivitis 2.3.2 Symptoms:

Recurrent attacks of redness, ropy discharge, irritation, itching.

2.3.3 Signs:

Conjunctival congestion, mucoid discharge, papillary hypertrophy.

2.3.4 Treatment:

• If there is secondary infection, use combination of Tobramycin 0.3% & Fluorometholone 0.02% eye drops, one drop 4 times a day for 15 days, along with Carboxymethylcellulose 1% eye drops (Lubricating eye drops) one drop 4 times a day for 15 days.

OR

• Olopatadine 0.1% eye drops, One drop twice a day for 1 week.

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Prevention of spread of infection is equally important as that of treatment

3. PTERYGIUM



Figure 3.4 Pterygium

Pterygium -

A Pterygium is a triangular sheet of fibro vascular tissue which invades the cornea. Pterygia typically develop in patients who have been living in hot climates and may represent a response to chronic dryness and exposure to the sun.

Signs –

- The conjunctiva then overgrows the opacities and progressively encroaches onto the cornea in a triangular fashion.
- Early cases show small, grey, corneal opacities near the nasal limbus.

Treatment –

Surgical excision is indicated either for cosmetic reasons or in cases of progression towards the visual axis. The excision of the conjunctival component followed by auto grafting or amniotic membrane grafting

4. CORNEAL ULCER

1. Definition:

Corneal ulcer is defined as breech or discontinuation of corneal epithelium with necrosis of surrounding corneal tissue.



Figure 4.1 Hypopyon Corneal ulcer

2. Etiology of Corneal Ulcer:



Pathogens can invade intact corneal epithelium & can cause ulceration.

Infective agents: N. Gonorrhoea, C. Diptherium, N. Meningitidis.



3. Clinical features

3.1 Symptoms:

- Redness
- Pain
- Watering / discharge
- Photophobia
- Diminution of vision

3.2 Signs:

- Lid Edema
- Conjunctival Congestion
- Ciliary Congestion
- Corneal Ulcer
- Hypopyon

Figure 4.2: Etiology of Corneal ulcer • Anterior Chamber depth –Shallow (Indicates Perforation)

- Iritis
- Posterior synechiae
- Lens normal
- IOP Normal/ Raised
- Sac (Dacryocystitis)

4. Investigations:

- Fluorescein Staining
- Corneal Scrapings
- Gram Staining
- Giemsa Staining
- KOH Mount
- Culture & Sensitivity

- Sac syringing
- Routing Investigation
- Blood Pressure
- Blood Sugar

5. Treatment:

- Cleanliness
- Fortified topical antibiotic eye drops like Amikacin 80mg/2ml.
- Tobramycin with a syringe, Inj 2ml of Tobramycin (40mg/ml) directly into a 5 ml bottle of tobramycin (0.3%) eye drop. Use within 14 days. keep in refrigerator.
- Antibiotics: Topical Moxifloxacin 0.5% eye drops 2hrly.
- Cycloplegics -Atropine sulphate 1% eye oint twice a day.
- Hot fomentation.

- Systemic antibiotic: Tab. Ciprofloxacin 500mg BD for 5 days.
- Diclofenac Sodium 50mg BD (antiinflammatory) for three or four days.
- Refer the patient to Ophthalmologist

6. Complications after perforation:

- Adherent leucoma
- Ant. Staphyloma
- Corneal fistula
- Pseudo cornea
- Spread of infection to other ocular tissues
- Expulsion of the lens / vitreous
- Expulsive choroidal hemorrhage
- Spontaneous evisceration
- Endophthalmitis / Panophthalmitis

• Rest.

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5. SCLERA



Figure 5.1 Episcleritis

1. Episcleritis

It is a common, benign, self-limiting and frequently recurrent disorder which typically affects young adults. It is seldom associated with a systemic disorder and never progresses to a true Scleritis.

2. Types of Episcleritis

2.1 Simple Episcleritis -

It is characterized by sectoral or rarely, diffuse redness. It usually resolves spontaneously within 1-2 weeks.

2.2 Nodular Episcleritis -

It is localized to one area of the globe, forming a nodule with surrounding congestion.

3. Symptoms

- Unilateral mild discomfort
- Tenderness to touch
- Watering

4. Treatment

• Mild cases –

Topical steroids, Ciprofloxacin 0.3% + dexamethasone 0.1% eye drops, one drop three times a day or topical NSAIDs Ketorolac Promethymene eye drops 0.5% one drop three times a day for one week.

• Unresponsive recurrent cases – Systemic Flurbiprofen 100mg three times a day.

5. Scleritis 5.1 Definition

Scleritis is a granulomatous inflammation of the sclera.



Figure 5.2 Scleritis

5.2 Types

5.2.1 Anterior Scleritis

- a. Non-necrotizing diffuse or nodular.
- b. Necrotizing with or without inflammation.
- **5.2.2 Posterior Scleritis**

5.3 Symptoms

- Unilateral mild discomfort
- Tenderness to touch
- Watering

5.4 Associated systemic diseases

About 45% of patients with Scleritis have associated systemic diseases like,

- Rheumatoid Arthritis
- Connective tissue disorders
- Miscellaneous conditions like relapsing Polychondritis, Herpes Zoster.

5.5 Treatment

- Oral NSAIDs Flurbiprofen 100mg three times a day for 5 days.
- Oral Prednisolone 40-80mg a day tapered according to response.
- Combined therapy with a NSAID and low dose steroid.

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6. UVEITIS

1. Definition:

Inflammation of Uveal tract is known as Uveitis.



Figure 6.1 Uveitis

2. Causes:

- Auto immune
- Infective

3. Classification:

Depending on the anatomical part of Uvea involved it is classified as

- 1) Anterior (Iridocylitis)
- 2) Intermediate (Pars-Planitis)
- 3) Posterior (Choroiditis)
- 4) Panuveitis (all parts are involved)

3.1 Acute Anterior Uveitis

3.1.1 Symptoms:

- Redness
- Pain -dull-aching pain, more during night
- Watering
- Photophobia (intolerance to light)
- Decrease in vision (usually slight blurring)

3.1.2 Signs:

Keratic precipitates and aqueous cells/flare can be seen on slit-lamp examination.

3.2 Intermediate uveitis

3.2.1 Symptoms:

- Decrease in vision (usually slight blurring)
- Black spots in front of eyes (floaters)

3.2.2 Signs:

- Ciliary Congestion.
- Tenderness Over Ciliary Body.
- Snow Banking and Vitreous haze seen on Ophthalmoscopy.

3.3 Posterior uveitis

3.3.1 Symptoms:

- No pain, Decrease in vision
- Black spots in front of eyes (floaters)

3.3.2 Signs:

Vitreous haze, focal inflammatory lesions and macular edema detected on ophthalmoscopy.

3.4 Panuveitis:

Symptoms and signs of both anterior and posterior uveitis are present.

4. Complications:

- Complicated cataract
- Secondary Glaucoma
- Cystoid macular edema
- Retinal Detachment
- Phthisis Bulbi (shrunken eye)

5. Evaluation of a Case of Uveitis:

- History
- Detailed history of previous episodes, treatment and response to treatment.
- History of associated systemic conditions like Tuberculosis, Syphilis, HIV infection, Rheumatoid Arthritis, Ankylosing Spondylitis, Malignancies, Diabetes Mellitus etc.

5.1 Ocular examination:

- Visual acuity.
- Pupillary reaction.
- Slit-lamp examination for keratic precipitates, aqueous cells and flare.
- Dilated fundus examination for vitreous haze, snow banking, chorioretinitis lesions, cystoid macular edema.

5.2 Investigations:

Depending on most likely cause in a particular case following investigations may be required,

- Complete Blood Count
- Blood sugar levels

- X-ray chest, ESR and Monteux test to rule out Tuberculosis.
- VDRL / TPHA to rule out Syphilis.
- ELISA for HIV.

5.3 Treatment:

- Topical steroids Prednisolone Acetate 1% eye drops (1hrly initially then tapered according to response to treatment).
- Cycloplegics –Atropine Sulphate 1% eye drops TDS.
- Antibiotics and systemic steroids are usually not required.
- Uveitis is a vision threatening condition. Refer the patient immediately on suspicion of Uveitis.
- Long term use of steroids can cause Cataract and Glaucoma.

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7. GLAUCOMA

1. Introduction: -

- Second major cause of blindness.
- Often asymptomatic in early stage.
- Damage is irreversible.
- Effective treatment is available.
- IOP Depends on the balance between production and removal of aqueous humour.

2. Definition:

Chronic progressive optic neuropathy caused by a group of ocular conditions which lead to damage of the optic nerve with loss of visual function.

Pathogenesis

- Raised IOP (Intra Ocular Pressure)- mechanical changes decreased axoplasmic flow ganglion cell death apoptosis.
- Reduced optic nerve head perfusion Ischemia.

3. Classification 3.1 According to etiology

- Primary
- Secondary
- Congenital- Present at birth. Infantile- present in first year life. Juvenile- present in the late childhood.

3.2 According to Appearance of the Angle

- Open angle Glaucoma
- Closed angle Glaucoma
- Combined mechanism Glaucoma

4. Investigations: -

- IOP with Applanation / Schiotz Tonometer
- SLE (Slit Lamp Examination)
- Fundus Examination
- Gonioscopy
- Visual -field testing
- OCT (Optical Coherence Tomography)



Figure 7.1 Open-Angle Glaucoma

5. Open-angle Glaucoma:

The most common form of Glaucoma, accounting for at least 90% of all Glaucoma cases.

- It is caused by the slow clogging of the drainage canals, resulting in increased intra ocular pressure
- Has a wide and open angle between the iris and cornea.
- Develops slowly and is a lifelong condition.
- Has symptoms and damage that are not noticed.
- It causes SLOW damage to the optic nerve causing gradual and irreversible loss of vision.
- "Open-angle" means that the angle where the iris meets the cornea is as wide and open as it should be. Open-angle Glaucoma is also called primary or chronic glaucoma. It is the most common type of Glaucoma.

a. Risk Factor:

- Age 6th decade.
- Sex –Common in Female.
- Race Common and more severe in Black.
- Family History and Inheritance –First degree relative of patients with POAG (Primary Open Angle Glaucoma) are at increased risk of developing the disease.
- Refractive error- POAG is common in Myopia

b. Clinical features:

- Generally asymptomatic headache.
- Frequent changes in Presbyopic correction.
- Difficulty in dark adaptation.
- Scotoma (especially in inferior field). {Blindness in particular sector}

c. Signs:

- Intraocular pressure > 21 mmHg (Schiotz Tonometer).
- Optic nerve head changes C: D ratio > 0.5.
- Asymmetry between two nerve heads > 0.2.
- Narrowing/notching/pallor of NRR.
- Disc hemorrhages.
- Visual field defects.
- Defects in nerve fiber layer.

d. Management:

- Start Timolol Maleate 0.5% twice daily if patient is not Asthmatic or not Hypertensive, according to response.
- Start Latanoprost 0.005 % /Travoprost 0.004 % (Prostaglandin analogues) one drop once daily in the evening if patient is Asthmatic or Hypertensive, according to response.
- Systemic Tab. Acetazolamide 250-1000 mg /day orally.
- Surgical Treatment Trabeculectomy
- Refer the patient to a Higher Centre for evaluation and management.

6. Primary Angle Closure Glaucoma

a. Introduction: -

Angle-closure glaucoma, a less common form of glaucoma:

- Is caused by blocked drainage canals, resulting in a sudden rise in intraocular pressure.
- Has a closed or narrow angle between the iris and cornea.
- Develops very quickly.
- Has symptoms and damage that are usually very noticeable.
- Demands immediate medical attention.

It is also called acute glaucoma or narrow-angle glaucoma. Unlike open-angle glaucoma, angle-closure glaucoma is a result of the closure of angle between the iris and cornea.

b. Definition:

Spectrum of conditions in which the peripheral iris moves forward to block the openings of the trabecular meshwork at the angle, causing a rise of intraocular pressure.





Figure 7.2 Angle-Closure Glaucoma

c. Predisposing factors:

- Short eye, Hypermetropia.
- Smaller corneal diameter.
- Shallow anterior chamber.
- Relative forward positioning of lens-iris diaphragm.

d. Clinical features:

- Unilateral headache or brow ache.
- Blurring of vision on the same side.
- Unbroken colored halos around lights during the episode.
- Gross diminution of vision.
- Red eye.
- Nausea, vomiting.
- Ciliary and conjunctival congestion, hazy cornea.
- Vertically oval, mid dilated pupil.
- Stony hard eyeball (very high IOP).

e. Treatment:

• Systemic - Intravenous Mannitol 20% 1-2 gm per Kg over ½ an hr.

- Tab. Acetazolamide, 250-1000mg per day in divided doses should be given if the patient is not nauseating or vomiting and can tolerate oral medication.
- One drop of 2% Pilocarpine placed in the eye every 5 min till pupil constricts, then one drop QID.
- One drop of Timolol Maleate 0.5 % twice daily

7. Congenital Glaucoma (Buphthalmos)

a. Definition:

Glaucoma appearing between birth and the ages of 3-4 years.



Figure 7.3 Congenital Glaucoma

b. Pathogenesis:

- Failure / abnormal development of the trabecular meshwork.
- Angle remains closed by persistent embryonic tissue.

c. Clinical features:

- Enlarged eye.
- Hazy cornea frosted glass appearance.
- Intolerance to light.
- Corneal edema.
- Watering.
- Blue sclera.
- Iridodonesis.

d. Management:

Urgent referral to higher center for Goniotomy / Trabeculotomy.

8. Lens Induced Glaucoma

Definition:

It is a type of Optic Neuropathy secondary to raised IOP due to Cataractous lens (Hyper mature Cataract).



Figure 7.4 Lens Induced Glaucoma

a. Classification:

- Phacolytic glaucoma.
- Phacomorphic glaucoma.
- Phacoanaphylactic glaucoma.

b. Symptoms:

- Diminution of vision.
- Pain.
- Redness of eyes.
- Headache.

c. Signs:

- Decreased visual acuity.
- Conjunctival congestion.
- Corneal oedema.
- Anterior Chamber shallow (Phacomorphic glaucoma).
- Flare, cells, sterile Hypopyon.
- Pupil sluggishly reacting to light.
- Lens mature or hyper mature Cataract.
- IOP raised.
- Fundus Absent Glow.

d. Investigations:

- Complete Blood Count.
- Blood Sugar Level estimation.
- Kidney Function Test.
- Serum Electrolytes.
- Serology.
- B-Scan.

e. Aims of Management:

- To relieve pain.
- To minimize optic nerve, insult secondary to raised IOP.
- To remove the cause.
- To reduce the IOP IV Mannitol (20%) 1-2 gm/kg over 30 min.

IV or systemic Acetazolamide – 250-1000 mg/day till IOP reduces.

f. Surgery:

- After controlling IOP.
- Extracapsular Cataract Extraction with PCIOL implantation.
- Post-Operative IOP monitoring.
- Visual damage in Glaucoma is irreversible hence early detection by regular screening is important.
- Once diagnosed, Glaucoma is lifelong, which needs continuous lifelong treatment and follow up.
- Treatment is aimed at preserving the vision.

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8. CATARACT

A cataract is clouding of the lens of the eye, which impedes the passage of light. Most cataracts are related to ageing, although occasionally children may be born with this condition, or cataract may develop after an injury, inflammation or disease.

Risk factors for age-related cataract include Diabetes, prolonged exposure to sunlight, tobacco use and alcohol consumption. Vision Can be restored by surgically removing the affected lens, and replacing it by an artificial one.



A cataract is an opacity of the normally clear lens which may develop as a result of aging, metabolic disorders, trauma or heredity

Figure 8.1 Cataract

1. Definition:

Any opacity of lens or its capsule whether congenital or acquired is known as cataract.

2. Types of cataract:

- **2.1 Senile cataract**
- **2.2 Traumatic cataract**
- **2.3 Complicated cataract**
- 2.4 Metabolic cataract



Figure 8.2 Senile Cataract



Figure 8.3 Traumatic Cataract



Figure 8.4 Complicated Cataract



Figure 8.5

3. Stages of Cataract:

- Immature cataract
- Intumescent
- Mature cataract
- Hyper mature- Morgagnian
- Sclerotic

4. Clinical features:

- Painless progressive diminution of vision, glare, black spots before the eyes, distortion of objects, polyopia.
- Complications of hyper maturity like Uveitis and Lens dislocation, Glaucoma.

5. Investigation: -

- V/A, IOP, Syringing.
- Blood Pressure, Blood sugar, ECG, Urine routine.
- A-scan.
- Xylocaine sensitivity test.
- Physician fitness.

6. Treatment:

6.1 Surgery-

- Conventional Extra Capsular Cataract Extraction with Posterior Chamber Intra Ocular Lens Implant (PCIOL).
- Manual Small Incision Cataract Surgery (SICS) with PCIOL.
- Phacoemulsification with PCIOL

6.1 Post-Operative Management –

- Topical Steroid + antibiotic eye drops Ciprofloxacin 0.3% + Dexamethasone 0.1% eye drops one drop six times a day for one week tapered every week up to six weeks & topical NSAIDs, Ketorolac Promethymene eye drops 0.5% one drop three times a day for two weeks. Tropicamide 1% eye drops one drop at night time for two weeks.
- First follow up at end of the first week & final follow up after 40 days for spectacle correction.

6.2 Complications of cataract surgery:

- Early- Striate Keratitis, corneal edema, prolapse of iris, Hyphema, Anterior Uveitis, delayed formation of anterior chamber, early Endophthalmitis, Toxic Anterior Segment Syndrome(TASS).
- Late- Bullous Keratopathy, Cystoid Macular edema, posterior capsular opacification, secondary Glaucoma, Retinal Detachment, late Endophthalmitis.

It is imperative for a Medical officer to recognize late complications of cataract surgery.

6.2.1 Bullous keratopathy

Corneal oedema due to corneal decompensation. Managed in initial stage, by local hypertonic saline eye drops (four times a day and eye ointment at night) & oral tab Acetazolamide 250mg three times a day. If unresponsive, Keratoplasty is the treatment of choice.

6.2.2 Posterior Capsular Opacification

It is the thick capsular opacification of posterior capsule after extra capsular cataract surgery. It can be managed by Nd: YAG capsulotomy.

6.2.3 Cystoid Macular Edema.

Relatively uncommon in uncomplicated phacoemulsification, occurs more often after the complicated surgery. Peak incidence 6-10 weeks.

6.2.4 Post-Operative Endophthalmitis

a. Definition: -

Refers to intraocular inflammation predominantly involving the vitreous cavity and anterior chamber as a result of post-operative intraocular colonization by microorganisms.

b. Symptoms

• Pain, Diminution of vision, redness and watering of eye.

c. Signs: -

- Decrease visual acuity.
- Lid swelling.
 - Discharge.
 - Corneal edema.
 - Conjunctival Chemosis.
- A/C Reaction, Iritis, Corneal Haze, Hypopyon, Circumcorneal congestion.

d. Risk factors:

- Post-operative Endophthalmitis.
- Environmental factors: Improper OT fumigation, improperly cleaned OT.
- Improper sterilization techniques.
- URTI in Surgeon / staff/ patient.
- Instrumentation with improperly autoclaved instruments.
- Infection of Sac –Blepharitis.
- Open wound leak.
- Poor patient hygiene.
- Poor compliance in putting post-operative medicine.
- Post-operative rubbing of eyes/ Trauma



Complication: Complete loss of eye sight if no timely intervention done.

f. Treatment:

It is an acute ophthalmic emergency

- Start intrusive systemic and topical antibiotics.
- Fortified Gentamycin eye drop every 15min.for 2 hrs. then one hourly three days.
- Moxifloxacin 0.5% eye drops ¹/₂ hourly.
- Cycloplegics Atropine Sulphate 1% eye ointment, eye drops.
- Urgent referral to Ophthalmologist is required.

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9. OPHTHALMITIS

1. Endophthalmitis: 1.1 Definition:

Endophthalmitis is the clinical term used to describe the inflammatory response of the eye to ocular infection.



Figure-9.1: Endopthalmitis

Acc to aetiological agents





1.2 Types:



1.3 Risk Factors

1.3.1 Bacterial

- Defects in sterilization of instruments.
- Contamination of tap water.
- Multiple dose fluids and drugs.

1.3.2 Fungal

- Contaminated irrigating solutions.
- Contaminated IOLs, viscoelastics, poor OT Hygiene, hospital construction activities.

1.4 Symptoms

Patient presents with symptoms most commonly on the second day after surgery.

- Pain
- Red



Figure 9.2 Endophthalmitis with Purulent Hypopyon

- Decreased vision.
- Hazy cornea.
- Hypopyon.
- Post-operative Endophthalmitis is the most common form.
- It comprises 70% of Infective Endophthalmitis.

1.5 Management

- In established Endophthalmitis, antibiotics when given orally or I.V., have poor penetration into the vitreous cavity.
- Hence, intravitreal injections are treatment of choice.
- Intravitreal injections bypass the blood retinal barrier and rapidly achieve therapeutic levels at the sites of infection.
- Large majority follow cataract surgery, most common surgical procedure (approx. prevalence 0.082%-0.1%)
- Post-operative Endophthalmitis is one of the most dreaded complications of cataract surgery and constitutes a true emergency.



Figure 9.3

Late onset Endophthalmitis

May be due to infection by organism of low virulence or fungi. Toxic reaction to Intra Ocular Lens (IOL) may also present as late Endophthalmitis. An urgent referral to ophthalmologist is required.

2. Panophthalmitis

2.1 Definitions: -

Refer to the inflammation of all coats of the eye including intraocular structures. It can also extend with the tissue surrounding the eyeball.

2.2 Symptoms of Panophthalmitis

- Eye pain.
- Burst opening of the eye ball.
- Protruding eyeball.
- Loss of Vision.

2.3 Signs

- Lids show a marked oedema and hyperaemia.
- Eyeball is slightly proptosed, ocular movements are limited and painful.
- Conjunctiva shows marked chemosis and ciliary as well as conjunctival congestion.



Figure 9.4 Acute necrotizing Panophthalmitis



Figure 9.5 Morganella morganii panophthalmitis

- Cornea is cloudy and edematous.
- Anterior chamber is full of pus.
- Vision is completely lost and perception of light is absent.
- Intraocular pressure is markedly raised.
- Globe perforation may occur at limbus, pus comes out and intraocular pressure falls.
- Complications include:
 - Orbital cellulites
 - Cavernous sinus thrombosis
 - Meningitis or encephalitis

2.4 Treatment

There is little hope of saving such an eye and the pain and toxemia lend an urgency to its removal. 1. Anti-inflammatory and analgesics should be started immediately to relieve pain.

2. Broad spectrum antibiotics should be

administered to prevent further spread of infection in the surrounding structures.

3. Evisceration operation should be performed to avoid the risk of intracranial dissemination of infection.

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10. RETINAL DETACHMENT(RD)

It is one of the major ocular disease conditions causing visual loss.

Predisposing factors

- High myopia.
- Trauma.
- Family history of retinal detachment.
- Intraocular Surgery-Aphakia, Pseudophakia
- Inflammation.
- Peripheral Retinal degenerations (Lattice/ Snail track).

1. Definition:

It is a separation of neurosensory retina from retinal pigment epithelium due to accumulation of fluid in the sub-retinal space through a retinal break, hole or a tear.

2. Symptoms:

- Occurrence of flashes of light (Photopsia) more serious in nasal field.
- Floaters in the vision.
- Curtain/ Veil in front of eye in any portion, more appreciated in lower field than upper.
- Sudden significant loss of vision, if macula is involved.
- A large bullous detachment.

3. Types:

- Rhegmatogenous RD.
- Tractional RD.
- Diabetic Retinopathy

- o Trauma
- Post-cataract vitreous incarceration
- Pars Planitis
- Exudative RD

4. Prophylaxis

Indicated in retinal breaks

- In Aphakic eyes.
- More than 1 clock hour of break.
- Family history of RD.
- Any symptomatic tear.

Asymptomatic tears and holes can be observed.

3 Modalities of prophylaxis.

- Laser Photocoagulation on slit lamp or IDO (Indirect Ophthalmoscope) with indentation.
- Cryotherapy at the break to seal it.
- Scleral buckling surgery for large tears(rarely).

5. Treatment:

RD surgery can be done only by tertiary eye care centers with infrastructure and trained personnel.

6. Complications of long standing RD

- Uveitis.
- Complicated cataract.
- Glaucoma.
- Phthisis bulbi.

In cases of high suspicion of Retinal Detachment, patient should be referred to tertiary eye care centre because timely treatment has better chances of saving vision.

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11. REFRACTIVE ERRORS

1.Introduction –

Normally, the rays of light entering the eye are brought to a precise focus on the retina – the light sensitive layer lining the back of the eye. When such a focus is not achieved, a refractive error results and vision is not clear.

When parallel rays of light from a distant object are brought to focus on the retina with the eye at rest, is called as Emmetropia (that is not accommodating).



Figure 11.1 Emmetropia 2. Causes of Refractive Errors

The eye's ability to refract or focus light- sharply on the retina-is primarily based on three features.

- 1. The overall length of the eye.
- 2. The curvature of the cornea.
- 3. The curvature of the lens inside the eye.

3.Types of Refractive Error

- Myopia.
- Hypermetropia.
- Astigmatism.
- Presbyopia.

4. Myopia

4.1. Definition:

A Dioptric condition of eye in which parallel rays of light from infinity come to focus in front of retina when eye is at rest.



Figure 11.2 Myopia

4.2Etiology:

- Axial lengthening of eyeball (1mm=3D).
- Curvature- abnormal curvature of cornea. eg. Keratoconus (0.1=3D).
- Index myopia eg. Old age, Nuclear Sclerosis.
- Acquired Myopia due to trauma.

4.3Types:

- Developmental.
- Simple.
 - Pathological.

4.3.1 Developmental Myopia:

- Abnormally long eyeball Myopia 10D.
- Fundus marked choroidal sclerosis, hyperpigmentation and myopic crescent.
- Usually stationary image.

4.3.2. Simple Myopia:

- Commonest, progressive from childhood to adult, seldom exceeds 5-6D.
- Generally, stop progression by 21 year of age.
- Best corrected vision is 6/6.
- Fundus exam. Shows Myopic crescent, Tigroid fundus and optic nerve degeneration with or without retinal break.

4.3.3. Pathological Myopia:

- Essentially degenerative and progressive condition, manifests in early childhood.
- The defect can reach up to 30D to 40D.
- The defect has strong heredity more common in female than male.

- Rapid increase in Myopia during puberty.
- Retinal evaluation is mandatory /compulsory.

4.4. Clinical feature:

- Inability to see the distant object and holding the book close to the eye in simple Myopia.
- Eye strain and headache.
- Black spot in front of eye.
- In pathological myopia-eye unusually prominent with slightly dilated pupil.
- Ophthalmoscopy reveals vitreous degeneration and opacity.
- Chorio-retinal degeneration patches at posterior Pole, choroidal sclerosis and Foster Fuchs spot at macula, Posterior Staphyloma.

4.5. Complications:

- Retinal hemorrhage due to post vitreous detachment.
- Lattice degeneration with retinal tear.
- Complicated Cataract.

4.6. Treatment:

- Optical lens- concave lenses with slight under correction.
- Contact lens.

4.7. Surgery: -

Refractive surgery like RK, PRK, ICR, LASIK

4.8. Other important aspects

Avoid contact sports where chances of blunt trauma are more

- Low vision aid- in pathological Myopia LVA is helpful. Glasses or contact lens does not improve the vision.
- Genetic counseling.
- General health-nutritional diet.
- Outdoor activity.
- Near work in good illumination.

5.Hypermetropia



Figure 11.3 Hypermetropia

5.1. Definition:

It is an error of refraction wherein parallel rays of light coming from infinity are focused behind the retina with accommodation at rest.

5.2. Etiology:

- Axial
- Curvature
- Index
- Posterior dislocation of lens
- Aphakia

5.3. Types:

- Latent.
- Manifest- Facultative. -Absolute.

5.4. Clinical feature:

- Low degree- no symptoms.
- High degree-eye strain, headache.
- Latent convergent squint in young.
- Presbyopia develops at an early age.
- Predisposed to angle closure glaucoma.
- Fundus may show features of pseudopapillitis.
- Increased chances of acute angle closure (Risk of Angle Closure Glaucoma).

5.5. Treatment:

- Optical lens- convex lens.
- Contact lens.

5.6. Surgery -

Laser, Thermal Keratoplasty, LASIK, Phakic intraocular lenses

6. Astigmatism



Figure 11.4 Astigmatism

6.1. Definition:

Astigmatism is condition wherein refraction varies in different meridian of the eye hence the point of focus cannot be formed on retina.

6.2. Etiology:

- Curvature Astigmatism- most common with corneal curvature.
- Index Astigmatism- inadequacies of refractive index of lens in different sector.

6.3. Types:

- With the rule.
- Against the rule.
- i. Regular
- ii. Irregular

6.4. Clinical features:

- Small Astigmatism Often asymptomatic.
- Severe symptom in hyperopic astigmatism where accommodation brought in play to overcome hyperopia.
- Irregular astigmatism is caused by corneal scar, penetrating injury to eye, Keratoconus.

6.5. Treatment:

- Cylindrical glasses.
- Contact lens.

6.6. Surgery:

LASIK, Photo astigmatic Keratectomy, Incisional correction (LRI), IOL, Conductive Keratoplasty, Corneal transplantation.

7. Presbyopia:



Figure 11.5 Presbyopia

Normal aging process, when near images can't be focused on the retina due to reduced accommodative ability.

The focus is behind the retina as in hyperopia.

If initially Emmetropic: Person begins to hold reading material farther away and distance vision is unaffected.

If initially hyperopic: Presbyopia occurs earlier.

Corrected with a convex lens for reading (bifocal).

All refractive errors should be detected and treated in childhood by regular school health

checkups and should be fully corrected.

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12. DIABETIC RETINOPATHY

1. Introduction:

India has become the Diabetic capital of the world. There is a high incidence of Diabetic Retinopathy (DR), among patients who are suffering from Diabetes Mellitus for duration of more than 15-20 years. Early diagnosis and intervention is crucial in dealing with this malady. Incidence and severity is more in type I (IDDM) than type II (NIDDM).

2. Classification:



Figure 12.1NPDR (Non Proliferative)



Figure 12.2 PDR (Proliferative) Maculopathy (Clinically Significant)

3. Risk Factors:

- 1. Advanced Diabetic Eye disease.
- 2. Vitreous hemorrhage.
- 3. Traction RD.
- 4. Neovascular Glaucoma.

4. History

- Duration of Diabetes.
- Control of Diabetes.
- Other systemic illness.
- F/H/O severe vision loss due to DR.

5. Systemic Evaluation

- Control of systemic risk factors is the cornerstone in management of DR.
- Glycemic control: Besides Blood Sugar Level, HbA1c levels more than 7.0 % is a known risk factor.
- Hypertension more than 130/90mmHg.
- Increased Triglycerides, cholesterol. Decreased HDL.
- Smoking, Sedentary life style.

6. Diagnosis

- Symptoms: decreased vision, floaters.
- On examination.
- Visual acuity.
- IOP.
- Neovascularization of iris.
- Gonioscopy: Neovascularization of the angle.
- Dilated fundus examination.

7. Fundus:

- Veins dilated and tortuous.
- Dot and Blot hemorrhages.
- Hard exudates.
- Cotton wool spots.
- Micro-aneurysms.
- NPDR stage.
- New vessels and traction bands seen.
- PDR stage.
- Vitreous hemorrhage and traction RD complicates the picture.
- Maculopathy runs as a distinct disease entity with maximum visual defect.
- Fundus Fluorescein Examination (FFE).

- Iodine based dye is injected into the antecubital vein and fundus pictures taken and digitally analysed.
- Leaks and ischemic areas can be picked up.
- Optical Coherence Tomography:
- Non-invasive retinal tissue pictures

8. Treatment: 8.1 NPDR :

• Periodic observation.

- Antioxidants.
- Control of Diabetes.

8.2 CSME:

• Focal/ Grid photocoagulation

8.3 PDR:

- Pan Retinal photocoagulation.
- Intra vitreal injection of VEGF inhibitors
- i. Bevacizumab
- ii. Ranibizumab
- Vitrectomy may be needed for Vitreous hemorrhage, Tractional RD.

9. Prevention:

• Intensive glycaemic control immediately after diagnosis of DM leads to lessen the complications.

Today Telemedicine has taken sophisticated technology to the rural masses yet the cornerstone is increasing awareness in public.

- Fundus examination with dilated pupil must be done for all newly diagnosed Diabetic patients and thereafter atleast once a year.
- A tight glucose control from the beginning has more long term benefits as compared to a tight control many years after diagnosis or after an end organ damage.

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13. OCULAR INJURIES

1. Introduction :

Eye can get injured by chemicals, heat, radiation and mechanical trauma. Ocular injuries are emergencies and treatment in first few hours can affect the visual prognosis.

2. Type of Injuries :-

- \checkmark Chemical.
- ✓ Mechanical injuries.

2.1. Chemical injuries :-

Common chemicals causing injuries are,

Alkalis -lime, ammonia, sodium hydroxide, cement, detergent soaps.



Figure 13.1 Alkali burn

Acids –Hydrochloric, Sulphuric, Acetic acids (vinegar), Toilet cleaners.

2.1.1 Alkali burn: Alkalis can penetrate through cornea causing severe inflammation. They cause occlusion of limbal vessels causing ischemia and necrosis of ocular surface. Necrosed tissue is sloughed off causing ulceration and healing by granulation tissue giving rise to abnormal adhesions between lid and ocular surface (symblepharon), severe dry eye, vascularized opaque cornea and even phthisis bulbi.

2.1.2 Acid burn: Coagulates the ocular surface tissue but usually does not penetrate inside the eye. Acids can cause limbal ischemia, severe dry eye, vascularized corneal opacities.

2.1.3 Clinical features:

a. Symptoms:

- Distressing pain.
- Watering from eyes.

- Decrease /Loss of vision.
- Inability to open the eye.

b. Signs:

- Severe Blepharospasm (forceful closure of lids).
- Conjunctival congestion.
- Raised IOP.

2.1.4. Diagnosis:

- History of the chemical causing injury should be elicited.
- Examination is difficult because of severe Blepharospasm.
- Ocular examination should be done under surface anaesthesia and if required sedation (especially in children).

2.1.5 Immediate treatment:

- Removal of any particulate matter (with special attention to lime pest/powder).
- Simple treatment is WASH-WASH-WASH the eye with water.
- Continuous irrigation with one bottle of normal saline over a period of 30 minutes.
- Topical Ciprofloxacin 0.3% + Dexamethasone 0.1% eye drops-one drop 4 times a day.
- Topical Atropine sulphate 1% eye drops one drop twice daily.
- Tab Ibuprofen 400mg twice daily for pain relief.
- Tablet Acetazolamide 250mg. (carbonic anhydrase inhibitor) one tablet four times a day.

After primary care patient should be referred to ophthalmologist for further management.

Prevention: Keep Limestone /Chuna and other sources of Chemical Ocular Injuries away from the reach of children.

2.1.6. Medical management at higher centre includes

- Assessment of limbal ischemia.
- Topical steroids Ciprofloxacin 0.3% + Dexamethasone 0.1% eye drops-one drop 4 times a day first 7 days.
- Topical lubricating eye drops -Carboxymethylcellulose 1% eye drops one drop 4 times a day for 15 days.
- Prevention of symblepharon formation by symblepharon ring.
- Surgical treatment in the form of amniotic membrane transplant, limbal stem cell grafting or Keratoplasty.

2.1.7 Complications:

- Dry eye
- Corneal ulcer
- Lid scarring
- Symblepharon
- Phthisis bulbi (shrunken eye)



Figure 13.2 Symblepharon and corneal opacity

- Immediate Copious irrigation with normal saline minimizes the damage.
- Use protective glasses at work place (Prevention better than cure!)

2.2 Mechanical Injuries :



Figure 13.3 Ocular trauma classification

2.2.1. Superficial foreign bodies

Common extra ocular foreign bodies are particles of dust, coal, emery, steel, lime particle, grain of corn, husk of seeds, wing/ mouth part of insects, caterpillar hair, thorns etc.

Patient gives history of foreign body in the eye.



Figure 13.4 Foreign bodies

2.2.2. Symptoms:

- Redness.
- Pain (pricking).
- Watering.
- Forceful closure of lids.

Anaesthetize the eye with 4% xylocaine eye drops/ proparacaine eye drops (0.5%).

Examine the conjunctiva by everting/double everting the upper lid.

Examine the cornea (stain with sterile fluorescein strip if necessary) to locate the foreign body.

2.2.3. Treatment:

- Removal of foreign body.
- Conjunctival foreign body can be removed by cotton bud or irrigation.
- Corneal foreign body should be removed by cotton bud or 26 G needle (hold the needle tangent to cornea to avoid perforation) under surface anaesthesia of eye with 4% xylocaine or Paracaine eye drop.
- Ciprofloxacin eye drop0.3 (Antibiotic) four times a day for 5 days.
- Homatropine 2% eye drops three times a day for 4 days in case of corneal foreign body.
- Eye pad for 24 hrs.
- Follow up to rule out infection.

If patient gives history of foreign body but it is not found on examination, suspect intraocular foreign body and carefully examine for wound of entry.

- Immediate Copious irrigation with normal saline minimizes the damage.
- Take precaution by using protective glasses at work places. (Prevention is always better than cure).

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14. OCULAR EMERGENCIES

1. Ocular conditions requiring emergency ophthalmological consultation.

- Trauma -Ruptured Globe
- Lid Lacerations.
- Chemical injuries.
- Endophthalmitis.
- Angle Closure Glaucoma.
- Severe Uveitis.
- Corneal Ulceration.
- Acute Vision Loss.
- Optic Neuritis.
- Orbital cellulitis.
- Lens induced Glaucoma.

2. Common causes of sudden diminution of vision:

Table-14.1:Common causes of sudden diminution of vision

Painful diminution of	Painless diminution	
vision	of vision	
Acute congestive	• Vitreous	
Glaucoma	hemorrhage	
• Chemical injuries	• Retinal Detachment	
• Mechanical injuries	• Central retinal	
• Endophthalmitis	artery occlusion	
Panophthalmitis	• Ischemic central	
	retinal vein	
	occlusion	
	• Optic Neuritis	
	• Malingering	

3. Common Causes Of Acute Red Eye



Figure 14.1 Conjunctivitis

- Conjunctivitis (with mucopurulent /purulent discharge)Subconjunctival hemorrhage (painless).
- Corneal ulcer.
- Acute iridocylitis (dull aching pain and slight blurring of vision).
- Endophthalmitis (severe pain with Diminution of vision).
- Panophthalmitis (severe pain, proptosis, with painful ocular movements).
- Orbital cellulitis (severe pain, proptosis, with painful ocular movements).

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15. PAEDIATRIC OCULAR PROBLEMS

1. Introduction :-

Out of 45 million people worldwide who are blind, around 1.4 million are children under 16years of age. The vast majority of childhood blindness happens before the age of five – a period when 75 per cent of learning is through sight.

2. Why is childhood blindness a priority?

There are several reasons why stakeholders believe that eliminating childhood blindness is a priority.

- There are an estimated 500,000 new cases each year of childhood blindness roughly one per minute.
- Blindness in children is often preventable if communities and parents become aware of the causes.
- Without early intervention for cataract blindness, children may go blind permanently.
- Blinding conditions increase child mortality up to 50% of children who become blind die within two years.
- 90% of children who are blind don't go to school.
- Eliminating childhood blindness will lead to a greater reduction in the number of **"blind years"** experienced by adults.

3. Causes of childhood blindness:

• Refractive errors.

(For details please refer to refractive error topic).

- Corneal scarring (scarring of the outer eye because of vitamin A deficiency & trauma).
- Cataract forms 39 per cent of all childhood blindness.
- Infections
 - Ophthalmia neonatorum.
 - Congenital Dacryocystitis.
 - Blepharitis.
- Squint.
- Ocular Trauma.
- Congenital and Developmental Cataracts. Leukocoria.
- Congenital anomalies

3.1 Ophthalmia neonatorum



Figure 15.1 Ophthalmia neonatorum

3.1.1. Definition:

Conjunctivitis occurring during 1st month after birth.

3.1.2. Mode of presentation, differential diagnosis & treatment:

Time of onset	Differential diagnosis	Treatment	
Within the first 48 hrs	Neisseria Gonorrhoeae	Inj. Ceftriaxone IM, Gentamycin / Moxifloxacin eye drops 1hrly (till discharge decreases), followed by 4 times a day for 7 days. Bacitracin eye oint. 4 times a day for 7 days.	
	Chemical	Wash the eyes, Erythromycin eye oint 4 times a	

Table 1: Ophthalmia neonatorum - mode of presentation, differential diagnosis and treatment

Time of onset	Differential diagnosis	Treatment	
		day for 3 days	
48 hrs to 72 hrs	Other bacteria	Gentamycin /Tobramycin eye dops two hourly for 7 days.Neomycin-Bacitracin eye oint 4 times a day for 7 days.	
5-7 days	Herpes Simplex Virus(HSV II)	Acyclovir eye oint 5 times a day for maximum 14 days.	
>1 week	Chlamydia Trachomatis(D-K)	Erythromycin eye oint./Chloramphenicol eye oint. 4 times a day for two weeks.	

3.2. Squint :

It is a condition where the eyes do not look in the same direction. Whilst one eye looks forwards to focus on an object, the other eye turns either inwards, outwards or downwards. A child with a squint may stop using the affected eye to see with. This can lead to visual loss called Amblyopia, which can become permanent unless treated early in childhood. Treatment involves patching the good eye, to force the use of the affected eye. Sometimes surgery is needed to correct the appearance of a squint. Squints are common and affect about 1 in 20 children. Most squints develop before preschool age, usually by the time a child is three years old. Sometimes squints develop in older children, or in adults



Figure 15.2 Esotropia



Figure 15.3 Exotropia

3.2.1 Definition:

Malalignment of visual axes is called as squint. It can be Esotropia/Exotropia or vertical malalignment i.e. hypertropia or hypotropia.

3.2.2 Types of Squint :-

- Esotropia An eye that turns inwards.
- Exotropia An eye that turns outwards
- Hypertropia An eye that turns upwards.
- Hypotropia An eye that turns downwards.
- Constant The squint present all the time.
- Intermittent The squint comes & goes.
- Manifest squint The affected eye turns when the eyes are open and being used.
- Latent squint The eye turns only when it is covered or shut, but looks fine when the eye are open.
- Concomitant squint The angle (degree) of the squint is always the same in every direction that you look.
- Incomitant squint The angle of squint can vary according to direction

3.2.3 Symptoms:

Squint(deviation of eye), diminution of vision

3.2.4 Signs:

Squint, diminution of vision, Amblyopia (lazy eye).

3.2.5 The aims of treatment of squint are :-

- Preserve or restore vision.
- Straighten the eyes.
- Restore binocular vision.

3.2.6 Treatment:

- Refractive correction.
- Amblyopia treatment.
- Exercises and if necessary surgical correction is done. (before 10 yrs. of age to have binocular vision).

Evaluation of the squint by ophthalmologist <u>at the</u> <u>earliest</u> is must.

3.3. Leukocoria:



Figure 15.4 Leukocoria

3.3.1 Definition:

White pupillary reflex is called as Leukocoria.

3.3.2Common Causes:

- Congenital Cataract.
- Retinoblastoma.
- Retinopathy of Prematurity.
- Toxoplasma /Toxocara Endophthalmitis.
- Persistent Hyperplastic Primary Vitreous.

3.4. Congenital Cataract:

It is clouding or opacity of the normally transparent lens inside the eye. Babies are sometimes born with cataracts as a result of an infection, injury, or poor development before they were born, or they may develop during childhood.

It is very important to treat this condition as soon as possible to get the best results. The treatment options and the timing of surgery will be decided by the ophthalmologist, based on the following factors:

- Age of the child.
- Density of cataract.
- Whether cataract involves one eye or both eyes.

3.5 Retinoblastoma:

It is the cancer of the eye in children. It originates from the retina, the light sensitive layer, in eye. It is the commonest tumor of the eye in childhood As the child does not complain of any poor vision, the tumor may remain undetected. The most common way of presentation is a white reflex (Leukocoria) behind the pupil. It may also present as squint (crossed eye), poor vision, painful red eye, inflammation of the tissue surrounding eye, protrusion of the eye ball (proptosis) etc. The aim of treatment in retinoblastoma, in order of priority, is to save the life, eye sight and cosmesis of the child. There are many treatment modalities for this tumor.

3.6 Retinopathy of Prematurity :

The most important determinant of any ROP management program is an effective screening strategy.

3.6.1 Whom to screen for ROP:

- All infants born below 1750 grams birth weight must undergo screening and all infants between 1750 grams and 2000 grams must be screened in case of any risk factor.
- All infants born below 34 weeks of gestation must undergo screening and all infants between 34-36 weeks must be screened in case of any risk factor.

3.6.2. When to start screening:

- All babies must be screened not later than 30 days of life or 4 weeks after birth.
- Babies born < 1200 grams or < 28 weeks must be screened by the 2nd - 3rd week of life.

3.6.3. Treatment:

- Laser Photocoagulation.
- For advanced stages like retinal detachment surgical treatment is required.

3.7 Congenital anomalies:

3.7.1 Ptosis:

Ptosis refers to drooping of an upper eyelid of one or both eyes. The droop may be barely noticeable, or the lid can descend over the entire pupil. Surgery usually is the best treatment for drooping eyelids. Children born with moderate or severe ptosis require treatment in order to get proper vision for subsequent development of child. Failure to treat ptosis can result in Amblyopia.

All these are potentially vision threatening conditions which need immediate referral to ophthalmologist.Every time the when the baby is brought for immunization look for pupillary reflex.

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16. LOW VISION

Low vision is a reduced level of vision that cannot be fully corrected with conventional glasses. It is not the same as blindness. Unlike a person who is blind, a person with low vision has some useful sight. However, low vision usually interferes with the performance of daily activities, such as reading or driving. A person with low vision may not recognize images at a distance nor able to differentiate colours of similar tones.

You are legally blind when your best corrected central acuity is less than 20/200 (perfect visual acuity is 20/20) in your better eye, or your side vision is narrowed to 20 degrees or less in your better eye. People who are legally blind may still have some useful vision. If you are legally blind, you may qualify for certain government benefits. It is estimated that approximately 17 percent of people over the age of 65 are either blind or have low vision.

1. Symptoms

- Difficulty in recognizing objects at a distance (street signs or bus signs).
- Difficulty in differentiating colors (particularly in the green-blue-violet range).
- Difficulty in seeing well up close (reading or cooking).

2. Causes

- Although low vision can occur at any stage in life, most of people develop low vision because of eye diseases.
- Common causes of low vision, particularly with older adults, include Macular Degeneration, Glaucoma, and Diabetic Retinopathy.

3. Tests and Diagnosis

- 1. Refraction (to assess your vision and determine the prescription for your glasses, if glasses may be of any use).
- 2. Visual field (to assess your peripheral vision).

4. Treatment and Drugs

- Optical devices to help you adapt, such as magnifiers, telephones, or closed-circuit televisions.
- Adaptive non-optical devices, such as largeprint cookbooks and talking watches.
- Occupational Therapy.

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Further reading

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17. EYE BANKING

Eye bank collects, evaluates and distributes the eyes donated by the donors. All eyes donated are evaluated using strict medical standards.

1. Functions of eye bank:

Those donated eyes which are found unsuitable for transplantation are used for valuable research and medical education purpose in the eye bank.

- 1. Availability of trained staff round the clock at Eye donation center in all District hospitals & all Eye bank to attend the calls.
- 2. Evaluate and provide quality corneas to corneal surgeons.
- 3. Enable corneal research using eyes unsuitable for grafts to find newer techniques, improve preservation methods and train corneal surgeons.
- 4. Increase public awareness about eye donation and eye banking.

2. Eye donation:

2.1 Important aspects of eye donation :

- 1. Eyes are to be donated only after death.
- 2. Eyes must be removed within 4-6 hours after death.
- 3. In case of death, nearest eye bank should be informed immediately.
- 4. Eyes must be removed only by a trained doctor.
- 5. The eye bank team may remove eyes at home of the deceased or at a hospital.
- Eye removal process is simple and takes only 10 to 15 minutes.
- 7. Eye removal does not lead to any disfigurement in eye.

- 8. Only the transparent section of the eyes called cornea is taken out and not the complete eye ball.
- 9. A small quantity of blood will be drawn to rule out communicable diseases.
- 10. Anyone can pledge eyes.
- 11. The eyes can be pledged to any eye bank preferably the nearest one.
- 12. The identities of both the donor and the recipient remain confidential.
- 13. One pair of eyes gives vision to *TWO* corneal blind people.
- 14. Eyes donated to the Eye-Bank that are not medically suitable for transplant may be used for medical research and education purposes.

2.2. Process of Eye donation

Pledging for eye donation procedure:

2.2.1 Eye donation can be done in either of these two ways.

- 1. She/he can walk in to a nearest Eye Bank and PLEDGE their eyes for donation. For this, a pledge form needs to be filled, signed by a witness (can be your relative/friend) and given back to the eye bank. At the time of donor's death, his/her relative/friend, who was a witness for the pledge form or any other family member/friend who had the knowledge that the person who passed away intended to donate the eyes, should call the nearest eye bank. One important aspect to be noted here that it is not sufficient if a person pledges for donating his/her eyes after the death, his/her relatives and friends should be well informed and be well aware to call the nearest eye bank, for donating the eyes after their death.
- 2. After death, a relative or a friend of the deceased person can inform the eye bank and

tell them that they wish to donate the eyes of their bereaved folk.

2.2.2. Donation procedure:

If there is an unfortunate death occurs of one of your family member/close relative/close friend, pick up the phone and dial to the nearest eye bank. Please note that this should be done WITHIN 6 hours of the death.

Give the eye bank officials the location of the house along with a landmark so that they can come and collect the eyes as quick as possible. These eyes will give a new vision to two blind people.

2.2.3. Important points to be taken care after a person's death, before eye donation:

After the death of a person, call the nearest eye bank within SIX HOURS of the death of the person.

- Switch off fans & keep AC on, if you have one.
- Close the eye lids gently and keep a moist cloth over the eyes.
- Raise head with a pillow.

2.2.4 What happens after eye donation?

- The donor's family receives a certificate of appreciation from the eye bank.
- The eyes are taken to the eye bank and evaluated by trained staff.
- Eyes are preserved in a solution of Optisol GS and refrigerated. This solution contains chemicals and drugs that are needed to keep the living cells of cornea healthy and functional.
- Tests are carried out and the report is sent to the corneal surgeon
- The eyes are used for a corneal transplant operation within 72 hours, but with present day availability of special storage media the eyes can be stored for a longer time before being transplanted.
- The recipient is chosen from the eye bank's waiting list and called for corneal transplant.
- Neither the patient knows whose cornea is used for him, nor the relatives of the donor know who has received the cornea. This information is strictly confidential.
- Periodic follow-up of the recipient is done over the time to ensure that the graft is successful.



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8.	Surgery	,
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1. Abdominal Pain

1. Introduction

Acute is a term used synonymously for a condition that needs immediate intervention



Figure 1.1: Acute causes

Abdominal Pain can be due to various organs involved with underlying causes & patient will present accordingly.

2. Gastric & Duodenal ulcer:

2.1. Presentation:

- Burning pain at the epigastric region referred to back worsened (gastric ulcer) or relieved by intake of food (duodenal ulcer)
- Dyspepsia, nausea/vomiting, occasionally haematemesis/melena.
- Complication with aggravation / progression of illness may cause perforation

2.2. Investigation:

- Routine investigation (Hb, CBC, Urine)
- X-ray shows gas under diaphragm in perforation
- Gastroscopy
- USG Abdomen

2.3. Treatment:

2.3.1 Drugs-

- Ranitidine 150 mg BD or Omeprazole 40 mg BD and alike drugs on empty stomach for 15 to 30 days.
- Intestinal motility regulators Domperidone 5 to 10 mg BD after meals for 15 to 30 days
- Liquid antacids

2.3.2 If perforation is diagnosed

- NBM, RT aspiration
- I.V. fluids RL, DNS, D 5% 5000-6000 cc in 24 hrs. to stabilize the patient
- Antibiotics Cefotaxime 1 gm I.V. BD, Gentamycin 80 mg I.V. BD,
- Metronidazole 500 mg I.V. TDS

• Stabilize the patient & shift the patient for surgical intervention after stabilization patient or after primary treatment.

3. Biliary Tract & Pancreas:

3.1. Presentation:

- Colicky pain in right upper abdomen, dyspepsia, nausea/vomiting
- Occasional jaundice

3.2. Investigation:

- Routine investigation: CBC, ESR
- USG Abdomen
- Serum amylase estimation.
- Serum lipase / LFT

3.3. Treatment:

- Analgesics like Aceclofenac 50 mg BD, Dicyclomine 20 mg BD
- Antibiotics Cefotaxime 1 gm I.V. BD, Gentamycin 80 mg I.V. BD
- Metronidazole 500 mg I.V. BD Stabilize the patient and as per diagnosis- biliary stone/ Pancreatitis patient may be referred for expert management to surgical hospital as early as possible.

3.4. Acute epididymo-orchitis /

Pyocele

Acute epididymo-orchitis is an infection in epididymis, testes & spermatic cord & manifests as acute Pain in Scrotum may be associated with fever. When infection, pus in tunica vaginalis, it is called as Pyocele.

3.4.1 Symptoms

- Pain is scrotal area.
- Erythema, in local area.
- Difficulty is walking due to pain.
- Fever

3.4.2 Investigation

- · USG Scrotum
- CBC shows leukocytosis

3.4.3 Treatment:

- a) Antibiotics
- b) Inj. Cefotaxime 1 gm IV 12 hourly. Inj. Metronidazole 500 mg IV / 8 hourly. Inj. Diclofenac 50 mg /IM 8hourly.
- c) Magnesium sulfate dressing:
- d) Surgical intervention Surgical intervention is required if conservative treatment does not give relief. Pyocele has to be drained for relief.

4. Small & Large Intestine:

4.1. OBSTRUCTION

4.1.1 Presentation:

- Severe pain, vomiting
- Distension of abdomen
- Constipation

4.1.2 Investigation:

- Routine investigation (CBC, KFTs & LFTs)
- X-ray standing Shows multiple gas-fluid levels (> 4 levels significant).

4.1.3 Treatment:

- a) NBM, Ryle's Tube aspiration
- b) I.V. fluids RL, DNS, Dextrose 5% (Adult 5000-6000 cc) in 4 to 6 hrs. to stabilize patient
- c) Antibiotics Cefotaxime 1 gm I.V. BD, Gentamycin
 80 mg I.V. BD,
- d) Metronidazole 500 mg I.V. BD
- e) Stabilize the patient & shift patient for surgical intervention after primary treatment.

4.2. PERFORATION

4.2.1 Presentation:

- Severe pain in abdomen, vomiting
- Distension of abdomen, constipation
- On examination Tender G/R

4.2.2 Investigation:

- Routine investigation
- X-ray standing Shows gas under diaphragm
- USG abdomen

4.2.3 Treatment:

- a) NBM, RT aspiration
- b) IV. fluids RL, DNS, Dextrose 5% (Adult 5000-6000 cc) in 4-6 hrs to stabilize patient
- c) Antibiotics Cefotaxime 1 gm I.V.BD, Gentamycin 80 mg IV BD,
- d) IV Metronidazole 500 mg BD
- e) Stabilize the patient & shift patient for surgical intervention after primary treatment

5. RENAL & URETERIC COLIC

It's type of pain caused due to irritation or obstruction by stone, in kidney or ureter itself. It is repeated spasmodic pain of longer or shorter duration till cause is removed i.e. stone removal or expulsion.

Renal pain most of the time is located in lumbar or back region. Ureteric pain is typical colic, mild or severe in nature radiates from lumbar to scrotal region. It is invariably associated with haematuria (micro/ macroscopic)

5.1. Sign & symptoms

Abdominal pain from loin to groin on one side or both sides in flank & back. Pain is associated with burning micturition, nausea, vomiting, fever & chills. Patient complains of haematuria. Stone size is not associated with severity of pain.

5.2. Investigations

- Haemogram
- Urine- m/e, c/s
- Renal profile (KFT + Serum Calcium)
- X-ray (KUB)
- USG abdomen (KUB)
- IVEU (Intravenous Excretory Urogram)

5.3. Treatment

- Analgesics- Inj. Diclofenac 50 mg/IM
- Antibiotics- Inj. Ampicillin 500 mg IV /6 hourly.
- Inj. Gentamycin 80 mg. IV/12 hourly. > 3 to 4 days
- IV Fluids -Ringer lactate, DNS,

Dextrose 5%

• Catheterization SOS

Give the treatment to stabilize the patient and / or refer the patient to higher surgical unit.

6. Acute Appendicitis

The acute appendicitis is essentially a surgical condition & can be defined as pain of sudden onset in right lower abdomen with fever with or without vomiting, nausea. Obstruction in lumen is cause for Acute appendicitis.

6.1. Presentation:

- Pain at first starts around the umbilicus or epigastrium and later it shifts to the right iliac fossa.
- Sudden disappearance of pain without improvement of general condition means gangrene and perforation.
- Anorexia, Nausea and Vomiting
- Fever
- Tachycardia and it is proportionate to temperature except in cases of gangrene, perforation and generalized peritonitis.
- Tenderness in right iliac fossa /guarding and rigidity

6.2. Investigation:

- Routine Haemogram (CBC & WBC is increased)
- KFT
- Urine examination
- CECT abdomen
- X- ray abdomen, abdominal Sonography
- CT abdomen with contrast

6.3. Treatment:

6.3.1 Conservative Treatment

Appendicitis with signs of localization

- Rest in a hospital
- Nil by mouth
- Analgesics- Inj. Diclofenac 50 mg/IV 8 hourly /SOS.
- Antibiotics- Inj. Ampicillin 500 mg IV/6 hourly. Inj. Gentamycin 80 mg IV/12 hourly. Inj. Metronidazole 400 mg IV/8 hourly.
- IV Fluids Ringer lactate, DNS, Dextrose 5%
- If patient is responding/ improving, give above treatment for 5 to 6 days

6.3.2 Surgical Treatment

If during the conservative treatment there is evidence of gangrene, perforation, generalized peritonitis or abscess formation, operation is indicated. If patient starts deteriorating with conservative line of treatment, then patient may be posted for emergency appendectomy.

Emergency appendectomy is contra indicated when diagnosis is appendicular mass.

7. Obstructed Inguinal Hernia

It is due to obstruction / retention of Abdominal visceral organs in hernia sac.

Patient presents with- non-reducible Inguino-scrotal swelling, pain in, distension of repeated vomiting. In late stages fever, septicemia.

7.1. Investigations:

- Routine Haemogram
- X-ray standing position shows multiple gas fluid levels
- USG pelvis
- No cough impulse.

7.2. Treatment:

- NBM, Ryle's tube aspiration, head low position
- IV Fluids Ringer lactate, DNS, dextrose 5%
- Analgesics- Inj. Diclofenac 50 mg / IV
- Antibiotics- Inj. Ampicillin 500 mg IV/6 hourly. Inj. Gentamycin 80 mg / IV/12 hourly. Inj. Metronidazole 400 mg / IV /8 hourly.
- Try to reduce the hernia, if not possible, then after stabilization of patient within 4 to 6 hours shift the patient for further needful surgical intervention at higher center.

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2. WOUNDS AND ABSCESS MANAGEMENT

From the earliest times the healing of wounds has been the central problem in surgical practice. This applies equally to the wounds of warfare and human assault, to the wounds of accidents, and to the wounds which the surgeon makes deliberately in the course of surgical operations.

The breach in the surface of the body, the skin, exposes the deeper tissues to the danger of bacterial infection (sepsis), and this danger persists until the healing process has restored an intact surface.

1. Commonly occurring injuries can be open or closed.

- Abrasions, bruises
- Contusions
- Lacerated wounds
- Penetrating wounds
- Incised
- Crush wounds
- De-gloving injury- wounds with skin loss
- Vascular injuries

2. Observe for

2.1 Three 'B'

- Bleeding
- Breathing
- Breaks (fracture)

2.2 Wound Contamination

2.3 Concealed bleeding (internal)

3. Management:

- 3.1 Cleaning of wounds
- 3.2 Debridement of dead or devitalized tissue
- 3.3 Suturing of wounds
- Wound healing may be primary intention- this is brought about by joining together the edges of the wounds by sutures, clips, adhesive materials
- Secondary intention when the wound edges are not brought together as there is skin loss, gross

contamination, wound infection, to be done by secondary suturing, skin grafting.

3.3.1 Suture Material:

- Non-absorbable -linen, silk, nylon, proline, ethilone
- Absorbable- cat gut, vicryl

3.3.2 Suture Types:

- Simple single or continues
- Mattress single or continues
- Tension sutures when there is requirement of forced alignment of wound edges

3.4 Supportive treatment to the associated injury/illness as required- fracture site stabilization etc.

4. Abscess

4.1 Clinical features:

An abscess is a tender mass generally surrounded by a colored area from pink to deep red. Abscesses are often easy to feel by touching. The middle of an abscess is full of pus and debris which leads to fluctuation test positive.

Painful and warm to touch, abscesses can show up any place on your body. The most common sites are in your armpits (axillae), areas around your anus and vagina (Bartholin gland abscess), the base of your spine (pilonidal abscess) around a tooth (Dental abscess) and in your groin. Inflammation around a hair follicle can also lead to the formation of an abscess; which is called a boil (furuncle).

4.2 Treatment

Unlike other infections, antibiotics alone will not usually cure an abscess. In general, an abscess must open and drained in order for it to improve. Sometimes draining occurs on its own, but generally it must be opened by a doctor in a procedure called incision and drainage (I&D)

Other risk factors for abscess include exposure to dirty environments, exposure to persons with certain types of skin infections, poor hygiene, and poor circulation.

4.3. Drugs:

• Antibiotics - Cap. Ampicillin 500mg QID 4 to 5 days or

Tab. Ciprofloxacin 500mg BD 4 to 5 days or

Tab. Cefadroxil 200mg BD 4 to 5 days

- Symptomatic treatment by analgesic, antiinflammatory drugs
- Supplementary/supportive Vitamin C 500 OD 3 to 4 days

4.4. Prevention

Maintain good personal hygiene by washing your skin with soap and water regularly

Take care to avoid nicking yourself when shaving your underarms or pubic area

Seek immediate medical attention for any puncture wounds, especially if associated with some debris in it

4.5. Important points

Once treated, the abscess should heal

Many people do not require antibiotics

The pain often improves immediately and subsides more each day

Wound care includes wound repacking, soaking, washing, or bandaging for about 7 to 10 days. This usually depends on the size and severity of the abscess

After the first two days, drainage from the abscess should be minimal to none. All sores should heal in 10-14 days.

Synonyms and Keywords

Abscess, abscesses, boils, carbuncles, furuncles, hiler adenitis, suppurative, pilonidal abscess, pustules, and white heads.

wounds, associated medical conditions of patients

On steroids or chemotherapy

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3. HEAD INJURY

Head injuries comprises of injuries to skin, skull, intracranial organs with vessels, in combination or singly.

Skull is the protective bony cage/vault for brain. Though it is not a perfect fit considering weight & volume of brain which may in turn leads to injury to the brain by inclusive of skin & vault or singly by mere inertia to brain & its membrane. The brain is suspended by superior cerebral veins from above, and antero - posteriorly to the membranes and laterally by attachments of dural process causing direct impact injuries to site - coupi injuries or indirectly to the opposite side of skull called counter coupe injuries.

1. Injuries can be classified grossly as

- Concussion
- Contusion
- Laceration

1.1 Cerebral Concussion:

It is distortion and displacement of brain substance minimally. Producing sudden loss of consciousness on sensory & motor paralysis with variable duration from seconds to minutes. There is a complete recovery in this injury.

1.2 Cerebral Contusion:

In this injury distortion, displacement is of severe nature along with injury to brain matter- neural fibers, this additional sliding movement between gray & white matter in to producing damage to nerves & axons.

1.3 Cerebral Laceration:

In this injury internal changes are same but the brain surface is actually torn. Clinically causing prolonged unconsciousness which leads Intracranial Hemorrhage.

2. Intracranial Hemorrhages:

There are four anatomical types of intracranial hemorrhages

- 2.1 Intra-cerebral hemorrhage in the brain substance it is usually associated with lacerations in brain.
- 2.2 Subarachnoid- in the pre arachnoids mesh.
- 2.3 Extradural Hemorrhage:

Extradural - Outside the dura between dura and periosteum of inner table of skull associated with fracture of skull.

It is an injury causing blood accumulation between skull & dural membrane. This causes pressure effects on brain & its functioning. It causes phase of unconsciousness with period of apparent recovery between two periods of unconsciousness, which is known as lucid interval.

Treatment: Craniotomy and immediate surgical evacuation

2.4 Subdural Hemorrhage:

Subdural under the dura, but outside the arachnoids matter: It is usually from the veins causing blood accumulation beneath the dural membrane.

Classification: Acute < 3 days, Sub-acute 4 -21 days, chronic >21 days

Treatment: Craniotomy and immediate surgical evacuation.

3. Signs & Symptoms:

- Vomiting
- Bleeding ENT
- Convulsions
- loss of consciousness, disorientation

4. Clinical Examination

- Examine level of consciousness, reaction and size of pupils: Cranial nerve examination, Spine examination DTR/Planters
- Blood pressure, pulse, respiratory rate

5. Investigations:

- Routine investigations
- Skull X-ray AP & Lateral
- CT-Scan Plain and CECT
- MRI

6. Management:

It depends upon the site, severity of injury, general condition of the patient & other relevant complications.

- Nil by mouth: RTA
- IV fluids, RL, D-10% 4000-5000 ml
- Drugs
 - IV Mannitol 100 cc / BD
 - Inj. Phenytoin 200-600 mg / day or
- If convulsions than Inj. Sodium Valproate 400-2000 mg / day
- Inj. Cefotaxime 1gm / BD
- Inj. Gentamycin 80 mg / BD

- Inj Diclofenac 3cc IM slowly
- Catheterization
- Maintenance of air way, throat & nasal suction
- Control of bleeding
- Care of associated injuries
- Oxygen inhalation with mask, SOS intubation / tracheostomy
- Management of shock
- After stabilization, patient to be shifted to higher center for expert neurosurgical management as early as possible.

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4. CHEST INJURY

Chest injury is common in road accident, fall from height, stab injury

1. Chest wall

- Fractured ribs
- Flail chest
- Fractured sternum

2. Pleura:

- Pneumothorax
- Tension Pneumothorax
- Traumatic haemothorax

3. Lung:

- Pulmonary contusion
- Pulmonary laceration

4. Mediastinal structures:

- Ruptured thoracic aorta
- Ruptured bronchus
- Ruptured diaphragm
- Blunt injury to the heart
- Haematopericardium

5. Penetration injuries:

- Bullet
- Stab wounds to the chest

6. Early Treatment:

- Relief of pain analgesics Inj. Diclofenac stat TDS
- Stabilization of the chest wall- strapping
- Removal of pleural blood or air -Simple aspiration, air tapping by needle, ICD
- Removal of tracheo-bronchial secretions
- Tracheostomy
- Oxygen administration by mask
- NBM
- IV Fluids Ringer lactate, DNS, dextrose 5%
- Antibiotics-
 - Inj. Ampicillin 500 mg. IV/6 hourly.
 - Inj. Gentamycin 80 mg. IV/12 hourly.
 - Inj. Metronidazole 500 mg IV/8 hourly.
 - Blood transfusion in massive haemothorax
- After stabilization, patient to be shifted to higher center for expert management within 4-8hrs

7. Common injuries:

Common injuries we come across which need primary treatment to stabilize the patient are:

7.1. Fracture Ribs:

7.1.1 Clinical presentation

- May be single or multiple, simple or compound. with or without Pneumothorax. Patient may present with - pain, difficulty in breathing in a later stage sometimes inability to take breath secondary to Pneumothorax,
- On examination: Tachycardia, Tachypnea

7.1.2 Diagnosis & management

Patient needs to be investigated by X-ray chest

- Analgesia
- Breathing
- Strapping

7.2. Flail Chest

7.2.1 Clinical presentation

This type of injury is commonly seen in accidents due to multiple rib fractures causing paradoxical movement of chest wall. Resulting in to respiratory insufficiency & retention of pulmonary secretions.

7.2.2 Diagnosis & management

To be diagnosed by X-ray Chest

Patient needs following treatment.

- Propped up position
- Oxygenation by mask
- Chest wall stabilization by strapping
- Tracheostomy SOS
- IPPV

7.3. Traumatic Pneumothorax:

7.3.1 Clinical presentation

It is a trapped air in pleural cavity due to penetrating wounds to chest wall, pleura and or lungs. It may be associated with haemothorax.

7.3.2 Diagnosis & management

- a) To be diagnosed by X-ray Chest evidence of fracture ribs, lung collapse black radiolucent chest cavity area on injured sides. Sometimes air fluid level in Haemo/ Pneumothorax.
- b) Patient needs ICD as above / IPPV

7.4. Tension Pneumothorax:

7.4.1 Clinical presentation

A tension Pneumothorax develops when there is a oneway valvular leak of air into the pleural cavity. Air rapidly accumulates, compressing the lung, and urgent treatment is required, as it is a life threatening emergency.

7.4.2 Diagnosis & management

X-ray chest

To be diagnosed and treated as above in addition patient needs.

- a) Simple needle aspiration to release tension
- b) ICD with under water seal

7.5. Haemothorax:

7.5.1 Clinical presentation

An accumulation of blood in the pleural cavity can occur from injury to the muscles in the chest wall, lung, heart or great vessels. There may also be a Pneumothorax in association.

Patient may be presented with pain, breathlessness, shock. On examination there are signs of pleural collection with lack of breath sounds & dull percussion note.

7.5.2 Diagnosis & management

- a) To be diagnosed by X-ray Chest of fluid level
- b) Patient needs following treatment with ICD
- Blood transfusion SOS
- After stabilization of the patient as and where required as per condition of injury & patient to be shifted to higher center in ALS transport vehicle.

8. INTERCOSTAL DRAINAGE (ICD)

8.1. Indications for Chest Tube Insertion:

- Pneumothorax
- Hydropneumothorax
- Haemothorax
- Empyema

8.2. PREPARATION

- 8.2.1. Requirements for Chest Tube Insertion procedure:
 - Chlorhexidine or povidone-iodine solution
 - Sterile towels and drapes
 - Sterile sponges
 - 2% Lidocaine without epinephrine (40 ml)
 - 10-ml syringe
 - 18-, 21-, and 25-gauge needles
 - Mayo scissors
 - Standard tissue forceps
 - Towel forceps
 - Needle holder
 - 0-Silk suture with cutting needle
 - Scalpel handle and no. 10 blade
 - Chest tubes (24, 28, 32, and 36 French)
 - Chest tube drainage system with NS
 - 2-in. adhesive tape
 - Sterile gowns and gloves, masks, caps



Figure 4.1: Inter-coastal Drainage tube

8.3. Pneumothorax / Haemothorax ICD insertion procedure:

8.3.1 Part Preparation

- Parts shaved and prepared
- Consent taken which should be valid and in written
- Parts painted and draped
- **Position:** Semi recumbent (45%) on a backrest Turn the patient's head on the opposite side

8.3.2. Anesthesia: LA

8.3.3. Site:

- 2nd inter costal space, mid-axillary line for pneumothorax.
- 7th inter costal space, in the mid-axillary line for Haemothorax.

Cut skin, Subcutaneous tissue. Split the muscles with artery forceps, till the pleura is reached. Keep the inter costal tube ready - Malecot's catheter or Portex ICD tube. Keep the underwater drain and drainage tube ready. Plunge the artery forceps into the pleura. The ICD tube mounted on another long & blunt artery forceps in thrust into the pleura along the direction of opposite shoulder.

8.3.3. Position the ICD tube:

All the holes of the tube should be in the pleural cavity

8.3.4. Fix the ICD tube with a skin stitch:

- Air bubbles are seen coming out and a column of fluid moving with respiration. Blood is seen coming out and a column of fluid moving with respiration in Haemothorax.
- **Clinically, the patient improves:** The tachypnoea settle. Respiratory sounds are heard. Chest X-ray shows lung expansion.
- 8.3.5 Inter costal drain is removed when the lung is expanded completely and air column movement absent

X-ray Chest shows full expansion, Column of fluid in the IC drain stops moving.

8.3.6 Removal:

Cut the skin stitch, pull the IC drainage tube out and give immediate strapping (Vaseline gauze airtight)



Figure 4.2: Site for Lidocaine Infiltration



Figure 4.3: Intercostal Tube insertion



Figure 4.4: Intercostal Tube fixation

8.4. POST INSERTION:

8.4.1 X-ray chest watch for complications:

- Not draining (check for kinking)
- Organ injury (lung, liver, spleen, heart, vessel) careful insertion
- Indication for Surgery
- Surgical emphysema (small hole and good suturing)
- Infection (sterile technique)

8.5. Safety features:

- First tube connecting drain to drainage bottles must be wide to decrease resistance
- Volume capacity of this tube should exceed ½ of patient's maximum inspiratory volume (otherwise water may enter chest)
- Volume of water in bottle should exceed ¹/₂ of patient's maximum inspiratory volume to prevent in drawing of air during inspiration
- Drain should always stay at least 45cm below heart level (prevention of removed fluid or water refluxing into patient)
- Clamp drain when moving
- Water level above tube in another bottle determines the amount of suction applied before air drain through tube (safety suction limiting device).

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5. ABDOMINAL INJURIES

It has only muscle layers protecting the internal organs anteriorly and laterally.

1. Blunt Trauma

Occurs when the offending agent is blunt and wide area of contract occurs at the time of impact. Severity of the injury depends on the force of the offending agent. Most commonly it effects to injury of spleen

2. Penetrating trauma

Occurs when injured by sharp instruments and the depth and direction of the wound is more important than the size of the wound. Most commonly it effects to Small bowel.

2.1. Cause:

- Accidents (Traffic, industrial and disasters)
- Falls
- Assaults
- In children (child abuse, bicycling, swimming, etc)

2.2. Investigation

- The history of the traumatic event is important in determining the nature and severity of the intra-abdominal organ injury.
- X-ray abdomen standing
- USG abdomen and CECT

3. Presentation & Management

3.1 Life saving measures such as ABCDE

- Airway patency
- Bleeding control
- Circulation maintenance
- Breathlessness
- Deformity (cervical stabilization and fracture immobilization) should be done first before transport to the hospital.
- Examine head to toe thoroughly on arrival of the trauma patient to identify the entire injuries. Categorize patient immediately into hemodynamically stable or unstable.
- Haemodynamically unstable patient will have the following features

- Cold clammy extremities
- Restlessness
- Breathlessness
- Sweating
- Altered level of consciousness
- External injuries
- **3.2. Stabilize the hemodynamically unstable patient first, investigate later.**
- 3.3. Cardiothoracic, head and spinal injuries get priority over Abdominal and other skeletal and surface injuries.

Refer patient immediately for emergency surgery & expert managements.

3.4 Blood transfusion if blood loss or in shock.

3.5 Treatment

- NBM, RT aspiration
- IV Fluids Ringer lactate, DNS, Dextrose 5%
- Analgesics Inj. Diclofenac 50 mg/IM.
- Antibiotics-
- Inj. Ampicillin 500 mg IV /6 hourly OR Inj. Cefotaxime 1 gm/BD.
- Inj. Gentamycin 80 mg IV / 12 hourly.
- Inj. Metronidazole 500 mg IV/ 8 hourly.
- 4. Proceed in the following order for haemodynamically stable patient:
 - Complete clinical examination
 - Plain X-ray Erect or left lateral position
 - Diagnostic peritoneal tapping
- Ultra sound (CECT)
- Abdominal CT

If there is progression in illness/detected internal injury, patient to be shifted for further expert treatment to higher center.

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6. Gangrene

1. Definition:

- Macroscopic death of the tissues with superadded putrefaction.
- Rest pain is an ominous symptom and usually requires revascularization because this form of advanced ischemia generally progresses to tissue loss. Patients with Diabetes or Renal failure are more susceptible to the development of ischemic pedal ulcers.

Two Types of Gangrene

- **Dry gangrene** usually results in patients with chronic critical limb ischemia without infection or without any other complicating factors like diabetes, osteomyelitis etc. It is characterized by dry shrivelled appearance, with clear line of demarcation and no foul smell.
- Wet gangrene is characterized by no clear line of demarcation, foul smell, purulent discharge, evidence of proximal spread in form of skip areas



Figure 6.1: Dry Gangrene Figure 6.2: Wet Gangrene

2. Treatment:

- Analgesics- Inj. Diclofenac 50 mg/IM
- Antibiotics- Inj. Ampicillin 500 mg.

IV/6 hourly. OR

Inj. Cefotaxime 1gm. IV/12 hourly

Inj. Gentamycin 80 mg. IV/12 hourly.

Inj. Metronidazole 500 mg. IV/8 hourly.

• IV fluids -Ringer lactate, DNS, dextrose 5% 5000-6000 ml

≻3- 4 days

Dry gangrene: Treat the cause

Wet gangrene:

- Stabilize the patient hemodynamically.
- Requires urgent assessment and shift the patient as required for further surgical treatment.
- Exclude Diabetes by repeated Blood Sugar estimation.

3. Fournier's Gangrene

Idiopathic Gangrene of scrotal skin

3.1 Clinical features:

- Common in young apparently healthy individuals.
- Sudden appearance of scrotal inflammation red swollen, very painful. Patient is toxic with fever, prostration
- Within one/two days, extensive gangrene of the scrotal skin occurs resulting in sloughing of the scrotal skin exposing the testicles. In few, the gangrene can involve skin of the penis, anterior abdominal wall, medial side of thigh, perianal region. In such situations, it is described as perianal phlegm
- Luckily, the testis does not get involved in Fournier's gangrene because of thick tunica albuginea.

3.2Treatment

- Antibiotics- -Inj. Cefotaxime 1 gm/BD.
 -Inj. Gentamycin 80 mg. IV/12 hourly
 -Inj. Metronidazole 500 mg. IV/8 hourly.
- IV Fluids RL, DNS 5000-6000 ml

If there is no regression in ischemic changes and when extensive debridement is required, shift patient to higher surgical center.

* Exclude Diabetes by blood sugar levels. When granulations occur, skin grafting is needed if raw area is large, if small wound can be sutured by approximating the skin edges of the wound.

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7. Burns

1. Introduction:

A common surgical emergency. Children and females are more vulnerable in India. Commonest are accidental scalds due to hot water or oil in children and stove burst or suicidal/ homicidal burns in females.

2. Clinical features:

Clinically burns can be classified as follows:

- Burns >10% in children and >25% in adults require Intravenous fluid therapy. In addition, certain specific cases as shown in figure 1 preferably need to be managed in Burn Centres.
- In adults, Rule of Wallace or rule of 9 is applicable.
- In Paediatrics, head is large hence rule of 9 is not applicable.

3. Treatment:

3.1. Medical:

- Reassure the patient.
- Burn surface calculation using **Wallace rule of '9'** and Calculate fluid requirement according to Parkland Formula and administer. Strictly monitor patient's intake and urine output after catheterization. If the burn surface area exceeds 35%, consider keeping the patient NBM and put Ryle's tube anticipating paralytic ileus to avoid aspiration.
- <u>Investigations:</u>
 - CBC
 - ABG
 - BSL
 - KFTs

3.2 Dressing:

3.2.1 Open –

Wounds to be cleaned with distilled water and antiseptic lotion, apply antiseptic ointment/creams – Furacin, Silver sulfadiazine

+Chlorhexidine etc.

3.2.2 Close –

Deep burns need close dressing by autoclaved bandage after completion of procedure mentioned above or use **Tulle Gras** (Bactigras or Sofra Tulle) of Silver sulfadiazine + Chlorhexidine, Povidone-Iodine etc.



Figure 7.1: Calculation of Burns percentage



Figure 7.2: Burn Surface Area = Burn percentage

3.3 Proper antibiotics coverage

- Burn patients should be given antibiotics like Inj. Cefotaxime 1gm BD or 3rd generation Cephalosporins.
- Inj. Gentamycin 80 mg BD
- IV Metronidazole 500 mg 8 hourly etc. for 7 to 8 days keeping in view the condition of the patient.

3.4 Other

• Venesection may be required in case if peripheral lines are all collapsed. Preferably Venesection is done at the ante cubital fossa. But if not possible due to extensive burns, then long Saphenous vein can be cannulated for administering fluids.

- Endotracheal intubation SOS Tracheostomy in patients with airway and breathing difficulty HNF.
- Escharotomy incisions may be required for circumferential deep burns involving the extremities or the chest.
- Daily needful dressing under aseptic precaution
- Adequate oral hydration

4. When to refer:

- All patients requiring special burn centre care as listed earlier.
- Patients in septicemia shock

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8. Dressings

1. Aim of wound dressing:

In an open wound which is being allowed to heal by secondary intention, or is being prepared for a delayed reconstructive procedure, the requirements are prevention of desiccation of the surface, removal of excess wound exudates, protection from physical damage and reduction of risk of infection

2. Types of wound dressing:

2.1 Occlusive (covered)

In most cases, occlusive wound dressings are preferable since they prevent bacterial contamination, keep the wound moist and provide a cover over the wound allowing normal activities.

2.2 Non occlusive (open)

Non occlusive wound dressing may be used in special cases of clean granulating wounds, burns, dry escharotic where the patient is well informed and educated regarding keeping the wound clean and local hygiene.

3. Indications for wound dressing:

- The primary determinant of the need for a dressing is the depth and degree of bacterial contamination of the wound.
- Superficial clean wounds are best managed by infrequent dressing.
- Deep clean wounds are best managed by immediate suturing and light dressing (for physical protection only)
- Contaminated / Dirty wounds are best managed by surgical debridement / wash followed by regular wound dressing
- All electively sutured surgical wounds DO NOT require regular wound dressing. At most they require a light dressing for physical wound protection.

4. Dressing components:

- 4.1 Antiseptics and wound cleaning solutions
 - Povidone iodine
 - Cetrimide
 - Glacial Acetic acid 10% infected greenish wounds (pseudomonas)

- Normal saline
- Hydrogen peroxide
- EUSOL

4.2 Dressing material

- Cotton gauze / pads / bandage
- Hydrocolloid dressings
- Paraffin / non adhesive gauze (BACTIGRAS)
- Collagen granules
- Adhesive strips

5. Types of wounds and dressings:

5.1 Clean open wounds:

Such wounds require simple cleaning with normal saline followed by a light dressing with non-adherent gauze such as Bactigras / plain paraffin gauze. Adherent dressings such as plain cotton gauze should be avoided as they tend to damage the granulation tissue on dressing change.

5.2 Clean sutured wounds:

Electively sutured clean wounds require only a light dressing of cotton gauze. The treating doctor may or may not place a layer of antiseptic ointment over the wound (e.g. povidone iodine, soframycin). Repeat dressings are not required.

5.3 Contaminated sutured wounds:

wounds require Contaminated thorough cleaning with copious amounts of normal saline. If the degree of contamination is high, this should be followed by hydrogen peroxide. The wound should then be irrigated with an antiseptic solution and covered with cotton gauze. Dressing change may be performed at 24 hours and is primarily to assess the presence of a collection or infection in the wound. The presence of either should prompt removal of some or all the sutures converting the wound into an open contaminated wound. Such wounds may also require systemic cover of antibiotics depending on the degree of contamination.

5.4 Contaminated open wounds:

Such wounds require regular (daily) dressing. Irrigation with hydrogen peroxide should be performed followed by normal saline to decrease the physical contamination of the wound. Antiseptic solution irrigation should then be performed. Such wounds tend to discharge, so a bulkier dressing with more absorbent gauze may be required.

5.5Dirty wounds:

Such wounds are characterized by presence of slough, necrotic tissue, eschar or frank gangrene. These wounds require initial surgical debridement for removal of gross necrotic tissue and debris. Debridement may have to be repeated. Depending on the degree of slough, this may be performed bedside or may require an OT and anesthesia. The dressing should be performed with hydrogen peroxide, followed by normal saline and antiseptic solution. The covering cotton gauze should be soaked in normal saline or EUSOL. Dressing changes need to be frequent and may have to be repeated thrice daily. The covering material should ideally be kept moist with saline / EUSOL. The wounds with surrounding cellulitis, edema should be covered with Magnesium sulfate dressings. It absorbs the fluids, reduce edema and pain. Large granulating wounds should be sent for Skin grafting.

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9. BREAST

1. Common presentations

1.1. Lump:

- Any new discrete lump (Mass) in breast or axillae
- New lump (Mass) in pre-existing Modularity
- Asymmetrical Modularity that persist after one or two menstrual cycle
- Abscess or breast inflammation which does not settle after treatment
- Recurrent mass/swelling

1.2. Pain:

- If associated with a lump (mass)
- Unilateral persisting pain in postmenopausal women

1.3. Nipple Discharge:

- Blood stained discharge
- Persistent single duct discharge
- 1.4. Nipple retraction or distortion / nipple eczema

1.5. Skin retraction of long duration



Figure 9.1: Signs of Breast Cancer

2. Examination of breast:

Some key steps:

- The patient (naked to the waist) is examined in the presence of a female attendant
- With patient sitting note change in contour, skin tethering, nipple tethering, arms above her head
- Tethering of lesion to the chest wall
- Examination of axillary tail with flat of hand
- Lump should be assessed for size (measured by calipers) shapes, consistency, tenderness, mobility and fixity to the overlying skin/ underlying chest wall, skin changes in the form of edema (peau d'orange

- Both axillae should be examined for palpable lymph nodes ensuring the anterior, medical posterior and apical nodes. The size, numbers and fixity should be recorded
- Note that clinical assessment of axillary nodes may not reflect their pathology status. For TNM staging it is important.
- The supraclavicular and infra-clavicular fossa and cervical region should also be examined for palpable lymph nodes
- Any other symptomatic regions should be examined as required for signs for metastatic diseases
- All examination findings must be recorded in sketch or written form, as part of good clinical practice and for medico legal reasons
- Initial assessment, investigations and staging of breast symptoms should be notified.

3. Benign Breast Conditions

- Fibro-adenoma
- Cyst
- Duct Papilloma
- Fibroadenosis or Fibrocystic disease of Breast.

4. Inflammatory conditions

- Abscess (Acute Mastitis)
- Fistula

5. Other conditions (Nonmalignant)

5.1. Fat necrosis

Fat necrosis following trauma can occur. Small cancer may clinically and radiologically mimic fat necrosis. Treatment is by reassurance or excisional biopsy.

5.2. Mastalgia (Breast pain):

- Can be symptom in malignancy of breast in less than 10%
- Chest wall disease should be excluded
- Intra-abdominal (gall stones) intra-thoracic (cardiac, pleural) pain may all present as breast pain.
- Exclusion of significant breast pathology, reassurance and non-steroidal drugs can provide symptomatic relief.

5.3. Gynaecomastia:

- Pubertal Gynaecomastia
- Gynaecomastia secondary to drugs therapy liver disease testicular tumors hyperthyroidism renal disease should be excluded.

6. Malignant lump (cancer):6.1. Definition:

The risk of developing distant metastasis increase with tumor size. Early breast cancer is defined as the condition in which tumor is confined to the breast, size 2 cm with obvious no metastasis.



Figure 9.2: Metastatic spread of Ca Breast

6.2 T.N.M. classification

T-primary tumor N-nodes M-metastasis

6.2.1 T – Primary tumor mass

This classification is internationally accepted and widely used to describe the extent of the tumor, regional nodes and metastasis.

- T0 No demonstrable tumor
- Tis Carcinoma in situ
- T1 Tumor diameter 2cm or less without fixation
- $T2-2\ cm$ to 5 cm without fixation
- T3 Tumor greater than 5cm
- T4 Extension of a tumor of any size to skin or chest wall.

6.2.2 N-regional lymph nodes

- N0- No palpable homolateral axillary nodes
- N1 Mobile homolateral axillary nodes
- N2- fixed homolateral axillary nodes or matted
- N3- Homolateral supraclavicular and infra-clavicular nodes + internal mammary and/or edema of arm.

6.2.3 M – Metastasis

- M0 No distant metastasis
- M1 Distant metastasis

6.3. Screening for breast cancer:

- Beneficial in women over 30 + years age
- Can detect smaller tumor with lower mass of lymph node, metastasis
- Early detection of breast cancer. By examination and mammography reduces morbidity in screened patients.



Figure 9.3: Breast Self-Examination

6.4. High Risk factors:

- Strong family history of Breast or Endometrial cancer
- Carcinoma of Contra-lateral Breast
- A history of intra-ductal papilloma
- Childless women and those conceiving after 30 years of age
- Breasts with dense parenchyma with prominent duct patterns (fatty breasts with minimal connective tissue elements are at risk)
6.5. Diagnosis of breast cancer:

6.5.1 Physical examination of breast (Clinicalexamination of breast fortnightly)



Figure 9.4: Symptoms of breast cancer 6.5.2 FNAC (fine needle aspiration cytology) by 23G needle:



Figure 9.5: FNAC Aspiration cytology

6.5.3 Imaging methods/ mammography



Figure 9.6: Mammography

6.6. Investigation:

- Biopsy, Tru-cut needle biopsy of the lesion for histopathological evidence
- Estrogen, Progesterone / Hormonal receptor status
- MRI spine for Metastasis in the vertebras.
- USG Liver for secondaries.

6.7. Treatment:

6.7.1 Aim:

- To reduce morbidity and extend 5-year and 10-year survival rates.
- To conserve the breast, if possible
- To achieve the control of disease.

6.7.2 Treatment options:

- Surgical management (MRM, BCT, QUART)
- Radiotherapy
- Chemotherapy
- Hormonal therapy

7. Surgical management:

7.1 Surgical management includes

- Modified radical mastectomy (Patey's)
- Radical mastectomy (Halstead) not done commonly
- Breast conservative procedure (Quadrantectomy, lumpectomy, wide local excision)
- Simple mastectomy for ulcerated, foul smelling lesion of breast (toilet mastectomy)
- Breast reconstruction cosmetic surgeries-after complete removal

7.2 Radiotherapy:

- Pre-operative for tumor mass depletion/regression
- Post-operative as advised by Surgeon

7.3 Hormonal therapy:

• Tab. Tamoxifen 100 mg BD

7.4 Chemotherapy: CMF regime, will achieve 25% reduction in the risk of relapse over 10 -15 years.

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10. Congenital Anomalies of G.I.T

1. Most common anomalies are:

- i. Cleft Lip or Cleft Palate
- ii. Hernias
- iii. Esophageal Fistula
- iv. Anorectal malformations

1.1 Cleft Lip (CL) or Cleft Palate (CP)

CL and CP are facial malformations that occur during embryonic development and they are the most common congenital deformities of the head and neck. They may appear separately or more often together.

1.1.1 Definition:

a) Cleft Lip: It is a congenital fissure in the upper lip.b) Cleft Palate: It is a midline fissure of the palate that results from failure of the two sides to fuse.



Figure 10.1: (A) Unilateral cleft lip, (B) Unilateral cleft lip and jaw, (C) Bilateral cleft lip with cleft palate and (D) Isolated cleft palate

1.1.2 Important feature of Cleft Lip & Cleft Palate

- Small notch
- Extending into the base of the nose.
- Unilateral or bilateral.
- CL/CP may involve the soft and hard palate.
- One or both sides.
- Unilateral clefts are nine times more common than bilateral clefts.
- 60% to 80% of children born with cleft lip and palate are male.

1.1.3. Diagnostic evaluations:

- Apparent at birth.
- Examining the palate
- Prenatal diagnosis after 13-14 weeks when the soft tissue of the fetal face can be visualized.

1.1.4. Why CP more serious than CL?

• It interferes more with feeding and breathing, more difficult to repair.

1.1.5. Therapeutic Management

The management of CL involves the cooperative efforts of a multidisciplinary health care team.

- Pediatrics
- Plastic surgery
- Speech/ language pathology
- Audiology
- Nursing and social work

1.1.6. Management is directed toward:

- Closure of the clefts, Surgery can be done after 6 months of age with Hb more than 10 gm and baby weight more than 10 kg.
- Prevention of complications.
- Facilitation of normal growth and development in the child.

1.1.7. Prognosis:

Even with good anatomic closure most children have:

- Some degree of speech impairment.
- Inefficient functioning of the muscles of the soft palate and nasopharynx.
- Improper tooth alignment.
- Varying degree of hear loss.
- Improper drainage of the middle ear.
- Recurrent Otitis media with scarring of the tympanic membrane.

1.1.8. Nursing consideration

It is important for nurse to emphasis not only the infant's physical needs but also on the parent's emotional needs.

- CL or CP reduces the infant's ability to suck, interferes with compression of the areola.
- Feeding is best with the infant's head in upright position.
- Large, soft nipple with large holes have been advised and used.
- Large syringe with soft rubber tubing can be used.
- With some infant spoon feeding works best
- Breast pump may be useful to stimulate the letdown reflex.

1.1.9. Postoperative care for CL:

- Elbow restrains are used to prevent the infant from rubbing or disturbing the suture line.
- Older infants who roll over will require a jacket restraint to prevent rolling on the bed and rubbing the face on the sheet.
- Adequate analgesia is required to relieve pain.
- Clear liquids are offered when the infant has fully recovered from anesthesia.
- The suture site is carefully cleansed.
- A thin layer of antibiotic ointment may be applied.
- Gentle aspiration of mouth and nasopharyngeal secretions.
- An upright infant position.

1.1.10. Postoperative care for CP:

- Lie on the.
- Feeding by bottle, breast or cup.
- Oral packing, it is usually removed after 2 to 3 days.
- Elbow restrains.
- Instruct the parents to keep the restrains at home until palate is healed (2 to 6 weeks).
- Mild sedatives may be prescribed.
- Soft food after discharging the patient as per advice of surgeon.

1.2. Hernias

It is a protrusion of a portion of a visceral organ through an abnormal or normal opening. Commonly seen hernias are:

- The diaphragm,
- The Abdominal wall, or
- The inguinal canal.

1.2.1. Diaphragmatic Hernias

Protrusion of Abdominal organs through opening in diaphragm.



Figure 10.2: Congenital Diaphragmatic Hernia

a) Symptoms

- Mild to severe respiratory distress
- Tachypnea, cyanosis, dyspnea
- Absent breath sound.
- Shock
- b) Diagnosis:
 - Symptomatic
- X ray chest
- c) Treatment
 - Supportive treatment of respiratory distress (Use of endo-tracheal intubation, oxygenation).
 - Prophylactic antibiotics (Ampicillin 50 mg/kg)
 - Surgical reduction of hernia and repair of defect.

d) Nursing care:

Preoperative:

- Resuscitation, maintain suction, oxygen.
- IV fluids
- Positioning head up.
- Administer medication

Post-operative:

• Carry out routine postoperative care and observation.

1.2.2 Hiatus Hernia:

Protrusion of stomach through esophageal hiatus.

a) Symptoms:

- Dysphagia, vomiting,
- Bleeding (haematemesis)

b) Management:

- Conservative management maintaining Posture.
- Surgical repair



Figure 10.3: Hiatal Hernia

1.2.3 Umbilical Hernia

Weakness in Abdominal wall around umbilicus, incomplete closure of Abdominal wall allowing intestinal contents to protrude through opening.

a) Investigation:

- USG abdomen
- b) Symptoms:
 - Noted by inspection and palpation.
 - High incidence in premature.

c) Management

Surgical repair



Figure 10.4: Umbilical Hernia

1.3 Esophageal Fistula

The esophagus instead of being an open tube from the throat to the stomach is closed at some point. A fistula is common between the trachea and esophagus.

1.3.1. Clinical manifestation:

- Excessive salivation.
- Coughing, choking, cyanosis,
- Apnea
- Abdominal distention.



Figure 10.4: Types of Tracheoesophageal Fistula (TEF)

1.3.2. Nursing alert:1

Any infant who has an excessive amount of frothy saliva in the mouth or difficulty with secretions and unexplained episodes of cyanosis should be suspected of having an EA / TEF and referred immediately for medical evaluation.

1.3.3. Therapeutic Management:

- Maintenance of patent airway.
- Prevention of pneumonia.
- Surgical repair of the anomaly.
- Avoid oral intake, start IV fluids.
- Removal of mouth secretion by suction.
- Broad spectrum antibiotic therapy.
- Gastrostomy and repair of the TEF.

1.3.4. Nursing role:

- Nursing responsibility for detection of this malformation immediately after birth.
- After feeding the infant swallows, but suddenly coughs and the fluid return through the nose or mouth, report immediately.
- The infant placed in incubator oxygen is administered.
- Intermittent or continuous suction of nose.
- Oral fluids are withheld, the fluid intake met by IV fluids or Gastrostomy.

1.3.5. Postoperative care:

- The infant is returned to radiant warmer or incubator.
- The Gastrostomy tube is connected to gravity drainage until the infant can tolerate feeding.
- Tracheal suction with extreme caution to avoid injury to the suture line.
- The initial attempt at oral feeding to make sure that the infant can swallow without choking.
- Oral feedings are begun with sterile water, followed by frequent small feeding of formula.
- Infants are usually not discharged until they are taking oral fluids well and the Gastrostomy tube is removed.

1.4. Anorectal malformations

- Abnormal development of genitourinary and pelvic organs.
- The rectum and urinary tract separate completely by the seventh week of gestation.

a) Imperforate anus

Is the most common congenital anomaly of GIT in newborn.



Figure 10.5: Imperforate anus

Investigation:

• Invertogram will detect type of anomaly: Low or High.

Diagnosis:

- There is no anal opening.
- The nurse is unable to insert the thermometer.
- No passage of meconium.
- Later on abdominal distention and pain occur.

Treatment:

High anomaly will require immediate colostomy while for low anomaly Local incision and Dilatation will be required.

Postoperative care:

- The anal area should be kept dry and clean.
- If a colostomy has been done the skin around the wound must be clean and apply colostomy clean dressing.
- The excoriations of skin occur around Colostomy area due to digestive enzymes from intestinal juices leaking from the side of bag. To prevent this aluminum paint should be painted on the skin around the Colostomy site.



Figure 10.6: Imperforate anus repair

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11. Deep Vein Thrombosis

1. Introduction:

Despite increased awareness and use of prophylactic modalities, DVT and pulmonary embolism (PE) remain important preventable sources of morbidity and mortality. The incidence of DVT ranges between 5 and 9 per 10,000 person-years in the general population, and the incidence of VTE (DVT and PE combined) is approximately 14 per 10,000 person-years.

The more common acquired risk factors include advanced age, hospitalization and immobilization, hormone replacement and oral contraceptive therapy, pregnancy and the recently postpartum state, prior VTE, malignancy, major surgery, obesity, nephrotic syndrome, trauma and spinal cord injury, long-hour travel (>6 hours), varicose veins, antiphospholipid syndrome. myeloproliferative disorders, and polycythemia. Heritable risk factors include factor V Leiden: Prothrombin 20210A gene variant: antithrombin, protein C, and protein S deficiencies; and dysfibrinogenemias. Patients are at risk of developing a post thrombotic limb and venous ulceration.



Figure 11.1: Deep Vein Thrombosis

2. Clinical Evaluation

Early in the course of DVT development, venous thrombosis is thought to begin in an area of relative stasis, such as a soleal sinus vein or immediately downstream of the cusps of a venous valve in the axial calf veins. Isolated proximal DVT without tibial vein thrombosis is unusual. Early in the course of a DVT, there may be no or few clinical findings such as pain or swelling. Even extensive DVT may sometimes be present without signs or symptoms. History and physical examination are therefore unreliable in the diagnosis of DVT. In addition, symptoms and signs generally associated with DVT, such as extremity pain and/or swelling, are nonspecific. DVT of the leg is complicated by the immediate risk of pulmonary embolus and sudden death.

3. Investigations:

Color Doppler will confirm the diagnosis.

4. Treatment:

4.1 Antithrombotic Therapy:

Antithrombotic therapy may be initiated with IV or SC Unfractionated Heparin, SC Low molecular weight Heparin, or SC Fondaparinux (a synthetic pentasaccharide). This initial therapy usually is continued for at least 5 days, while oral vitamin K antagonists are being simultaneously administered. The initial therapy typically is discontinued when the international normalized ratio (INR) is \geq 2.0 for 24 hours

Hemorrhage is the primary complication of UFH therapy. The rate of major hemorrhage (fatal, intracranial, retroperitoneal, or requiring transfusion of >2 units of packed red blood cells) is approximately 5% in hospitalized patients undergoing UFH therapy (1% in medical patients and 8% in surgical patients). For patients with UFH-related bleeding complications, cessation of UFH is required, and anticoagulation may be reversed with protamine sulfate. Protamine sulfate binds to UFH and forms an inactive salt compound. Each milligram of protamine neutralizes 90 to 115 units of heparin, and the dosage should not exceed 50 mg IV over any 10-minute period. Side effects of Protamine sulfate include Hypotension, Pulmonary edema, and anaphylaxis.

Most patients who receive therapeutic LMWH do not require monitoring. Patients who do require monitoring include those with significant renal insufficiency or failure, pediatric patients, obese patients of >120 kg, and patients who are pregnant.

4.2 Vitamin K antagonists:

Includes Warfarin and other Coumarin derivatives, are the mainstay of long-term antithrombotic therapy in patients with VTE. Warfarin therapy usually is monitored by measuring the INR, Vitamin K antagonist may be started on the same day as the initial paraenteral anticoagulant, usually at doses ranging from 5 to 10 mg. Smaller initial doses may be needed in older and malnourished patients, in those with liver disease or congestive heart failure, and in those who have recently undergone major surgery and depends on liver function. diet, age, and concomitant medications.

Virchow's Triad

The factors described by Virchow are important in development of venous thrombosis these are:

- Endothelial injury (Vascular injury)
- Stasis or turbulence of blood flow
- Hyper- coagulability of blood.

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12. Varicose Vein



Figure 12.1: Varicose veins

1. Introduction:

Varicose veins are dilated, tortuous and elongated superficial veins of the lower limbs. Most develop in the tributaries of the greater and lesser saphenous veins, which are usually dilated but rarely varicose themselves.

- Age old disease known since Hippocrates, Mentioned in Ebers Papyrus in 1550.
- Incidence F>M, Prevalence 10–23% in men 30–40% in women
- Skin changes 5%, Active ulcers 0.5%.

1.2 Types:

- Primary Varicose Vein: (More Common, congenital predisposition, decreased number of defective valves)
- Secondary Varicose vein: Less common, dysfunction of valves, trauma, DVT, AV fistula.

1.3 Risk Factors: Female sex, pregnancy, family history, prolonged standing & obesity

2. Clinical features:

Patient usually has Cosmetic concerns

Symptoms of tiredness & heaviness in legs and itching and even leg ulcer

3. Etiology and Risk Factors

Female sex, Pregnancy (especially multiparity), pelvic tumors, family history,

4. Complications:

- Ankle edema, Ankle flare, Pigmentation, Venous eczema, Venous ulcer, Profuse Hemorrhage, Superficial Thrombophlebitis, Equinous deformity, Calcification.
- Deep vein thrombosis:

One of the important etiology as well as complication of varicose veins. It presents clinically as swollen woody calf, pain, ankle edema and redness. Patient may have fever.

5. Investigations:

Doppler ultrasound is most ideal for complete evaluation.

6. Treatment:

6.1. Management options at glance

- Bisguard's exercise- Heel raises at least 4 times every day.
- **Compression stockings** For early varicosities without complications. Types of stocking are
 - Below/Above knee
 - Mid-thigh

6.2. Surgical treatment

- **Flush ligation** at sapheno-femoral junction with stripping of long Saphenous Vein.
- **Perforator ligation** Sub facial.

6.3. Others

- Sclerotherapy Ultrasound-guided foam Sclerotherapy for short segments or recurrent varicosity.
- Radiofrequency ablation (Closure®),
- Endo Venous Laser Ablation.
- Ambulatory phlebectomy or a Combination of these treatments.

7. Do's and Don'ts:

• In case the varicosity starts bleeding do not panic, immediately lie down flat on the ground and raise the bleeding limb above the heart level and apply Compression.

8. Instructions to patient:

• Life style changes, follow Bisguard's exercises daily, use of compression stocking through the day can be removed at night. While sleeping ask the patient to raise the foot end of the bed to aid venous return

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13. GENITO-URINARY DISORDERS

1. PARAPHIMOSIS

1.1 Clinical features:

It is a condition in which the contracted foreskin cannot be retracted over the glans (phimosis). It is forcibly retracted back off the glans and cannot be pulled forwards again. The tight band causes obstruction of venous returns followed by swelling of the distal foreskin and glans. It can occur at any age. Condition is very painful & if not taken care may cause Ischemic/Pre-gangrenous changes.

Clinical features:

- Difficulty in micturition.
- Ballooning of prepuce during micturition
- Edema, Erythema and tenderness of the prepuce and presence of purulent discharge
- Inability to retract the foreskin

1.2 Treatment:

Technique of Reduction- Parts shaved & prepared Patient in OT Parts painted & draped. Local anesthesia 2% Xylocaine infiltrated at the root of the Penis.

- Manual compression and reduction
- By giving multiple needles punctures over swollen & edematous foreskin of penis and squeezing it to drive out the edema fluid followed by mechanical reduction by pulling it to normal position.
- Dorsal slitting at 12' O clock position and reduction.

Reduction of the paraphimosis is followed by a course of antibiotics. And after 8-10 days perform Circumcision (at surgical center).

2. TORSION TESTIS:

It is a condition caused by the twist of suspended testis.

2.1. Patient presents with

Sever scrotal pain of sudden onset referred to lumbar region Vomiting, fever, Scrotal swelling with gross tenderness up to the cord

2.2. Investigation:

- Routine investigation
- USG Scrotum Doppler study shows decreased blood flow & also viability of the testis.

2.3. Treatment:

- I.V. fluids Ringer lactate, DNS, dextrose 5%
- Analgesics- Inj. Diclofenac 50 mg/I.M.Antibiotics-Inj. Ampicillin 500 mg
- I.V/6 hourly. Ini Gentamycin 80 mg I V /12 hourly
- Inj. Gentamycin 80 mg I.V./12 hourly.
- Inj. Metronidazole 400 mg. I.V./8 hourly

If not relived after stabilization of patient to be referred to surgical center.

3. EPIDIDYMO-ORCHITIS

It is an inflammatory & infective condition affecting epididymis and testis

3.1. Patient present with:

- Scrotal pain
- Testicular swelling
- Low grade fever with chills

3.2. Investigation:

- Routine investigation
- USG Scrotum Doppler study

3.3. Treatment:

- Analgesics tablet Acelofenac 10-20 mg/ BD OR Dicyclomine 50 mg/ BD
- Antibiotics- Ofloxacin 400 mg/ BD for 5 to 7 days OR Cefodoxime 200 mg/ BD 5 to 7 days.
- Anti-inflammatory- Serratiopeptidase -5 to 10 mg/ TDS 5 to 7 days
- Ibuprofen 200-400 mg/ TDS 5 to 7 days
- Scrotal support for 2-3 weeks

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14. ANO-RECTAL DISORDERS

1. Hemorrhoids

Hemorrhoids are clumps dilated veins occurring in relation to the ano-rectal junction (commonly known as piles)

1.1. Classification:

1.1.1. By descent

Grade 1- Bleeding

Grade 2- Protrusions with spontaneous reduction

Grade3- Protrusions regressing with manual reduction

Grade4- Irreducible protrusions

1.1.2. By Location

- External: arise from inferior hemorrhoidal plexus, and are covered by modified squamous epithelium occurs below pectinate line.
- Internal: arise from superior hemorrhoidal plexus, also above pectinate line.
- Interno-external- when both are present.

1.2 Clinical Features

1.2.1 Symptoms:

- Bleeding, bright red and painless, frequent bleeding may lead to anemia
- Prolapse
- Pain on prolapse
- Mucus discharge
- Pruritus / itching

1.3 Investigations:

- Direct Visualization
- Digital Rectal Examination
- Proctoscopy
- Basic investigation (Hb, CBC, Urine)
- Blood grouping and RH typing
- USG (Associated portal hypertension)
- Colonoscopy

1.4 Complications: -

- Profuse hemorrhage
- Fibrosis
- Thrombosis
- Infection
- Pyelophlebitis
- Ulceration

- Gangrene
- Suppuration
- Portal pyemia

1.5 Treatment: -

1.5.1 Conservative management:

- Dietary improvement high fiber diet
- Avoid straining at defecation

1.5.2 Active Surgical Treatment

- Sclerotherapy
- Banding
- Photo coagulation
- Hemorrhoidectomy

1.6 Postoperative care

Table 1: Post-Operative Care and Complication

Post-Operative care	Post-Operative Complication	
 Watch for bleeding Sitz bath twice daily in the postoperative period Analgesics Stool softeners and laxatives Avoids digital rectal examination in the early postoperative periods 	 Early Pain acute retention of urine Reactionary Hemorrhage (24 hrs) Constipation Late Secondary hemorrhage (24 hrs) Anal stricture Anal fissure Fecal incontinence 	

1.7 Important note:-

- Surgery is for failure in conservative management or in severe cases
- Colonic carcinoma to be ruled out (colonoscopy)
- Anemia to be corrected pre-operatively (Blood transfusion if required)

- Muco-cutaneous bridges should be preserved during surgery to prevent anal stenosis
- Conservation treatment preferred in pregnant ladies and elder.

2. Anal Fissure

2.1. Definition:

Anal fissure is an ulcer or a crack in the canal or anal verge that may extend from muco-cutaneous junction to dentate line. Anal fissure is of particular concern if it is acute because the degree of patient discomfort, pain and disability are extremely high.

The type of fissure is acute and chronic.

- Acute anal fissure is a deep tear through the skin of the anal margin extending into the anal canal. There is little inflammatory indurations or edema of its edges. There is accompanying spasm of the anal sphincter muscles.
- Chronic anal fissure is characterized by inflamed indurate margins, and a base consisting of either scar tissue or the lower border of the internal sphincter muscle. Chronic anal fissure does not heal with conservative measures.

2.2. Symptoms: -

- Pain during the defecation and / or after defecation
- Slight bleeding / streaking

2.3. Examination: -

- Acute fissure Pain during per rectal examination with or without palpatory mucosal abrasion
- Chronic fissure Skin tag fibrosed ano-rectal mucosa

2.4. Investigations: -

Basic investigation - Hb, CBC, Urine

Colonoscopy

2.5. Treatment: -

2.5.1 Conservative Management

Laxatives – Cremaffin 30ml at bed time / stool bulking agents -

Ointment to relax anal sphincter and improve blood flow like Diltiazem / Nifedipine cream / glyceryl trinitrate ointment

- Sitz bath
- High fiber diet

2.5.2 Surgery: -

- Anal dilatation under GA
- Fissurectomy

3. Pilonidal Sinus

3.1 Clinical features:

A pilonidal sinus is a depression in the skin or small pit that occurs at the bottom of the tailbone (coccyx) and can become infected and filled with pus. Once infected, the technical term is pilonidal abscess. Pilonidal abscesses look like a boil at the bottom of the tailbone, just above the crack of the buttocks. It is more common in men than in women. It usually happens in young people up into the fourth decade of life.

3.2 Treatment:

3.2.1 Medical treatment

- Antibiotics (Cap. Ampicillin 500 mg TDS x 5 days)
- Analgesics (Tab. Diclofenac 50 mg BD x 5 days)

3.2.2 Surgery

- Anal dilatation under GA.
- Fissurectomy

4. Anorectal abscess

4.1 Definition:

Anorectal abscess is a collection of pus in the area of the anus and rectum.

4.2 Causes

- Common causes of anorectal abscess include:
- Blocked glands in the anal area
- Infection of an anal fissure
- Sexually transmitted infection
- Deep rectal abscess may be caused by intestinal disorders such as Crohn's disease or diverticulitis.

4.3. Risk factors

Anal sex

- Chemotherapy drugs used to treat cancer
- Diabetes
- Inflammatory bowel disease (Crohn's disease and ulcerative colitis)
- Use of medications such as Prednisolone.
- Weakened immune system (such as from HIV/AIDS)

The condition may occur in infants and toddlers who are still in diapers and who have a history of anal fissures.

4.4. Symptoms

- Swelling around anus and constant throbbing pain, fever are the most common symptoms. Pain with bowel movements may be severe. Other symptoms may include: -
 - Constipation
 - Discharge of pus from the rectum
 - Fatigue and general malaise
 - Fever night sweats, and chills
 - Lump or nodule, swelling, redness, tenderness at edge of anus
 - Painful, hardened tissue

In infants, the abscess often appears as a swollen, red, tender lump at the edge of the anus. The infant may be fussy and irritable from discomfort, but there are usually no other symptoms.

4.5 Investigations

A rectal examination may confirm an anorectal abscess.

A procto sigmoidoscopy may be done to rule out other diseases.

Rarely, you may need a CT scan, MRI or ultrasound to determine where the pus collection is located

4.6. Treatment: -

4.6.1 Analgesics-

Inj. Diclofenac 50 mg/IM BD/ SOS

4.6.2 Antibiotics-

Inj. Ampicillin 500 mg. I.V. / 6 hourly. 5 days

Inj. Gentamycin 80 mg. I.V. / 12 hourly.

Inj. Metronidazole 400 mg. I.V. /8 hourly.

4.6.3 Treatment involves surgery, open and drain the abscess, patient may be shifted at surgical center.

4.6.4 Post-operative care

After surgery patient needs warm sitz baths sitting in a tub of warm water) this may help to relieve pain, reduce swelling and make the abscess easier to drain.

Drained abscesses are usually left open and have no stitches.

Patient should be advised to take stool softeners, practice good hygiene and eat a soft or liquid diet until the abscess has healed.

4.7. Possible complications

- Anal fistula
- Sepsis
- Continuing pain
- Problem keeps coming back (recurrence)
- Scars

4.8. Prevention: -

- Prevention or prompt treatment of sexually transmitted diseases may prevent this cause of anorectal abscesses. Use condoms during intercourse, including anal sex, to prevent such infections.
- Frequent diaper changes and proper cleaning during diaper changing will help to prevent both fissures and perianal abscesses in infants and toddlers.

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15. Tracheostomy

1. Introduction

Tracheostomy is one of the most common surgical procedures performed in the ICU and is the airway of choice for patients requiring mechanical ventilation for more than 2 weeks.

2. Procedure

Patient's neck is extended and the surgical field is exposed from the chin to several inches below the clavicle. This area is prepared and draped and prophylactic antibiotics are administered at the discretion of the surgeon. A vertical or horizontal incision may be used; however, a horizontal incision will provide a better cosmetic result. The platysma muscle is divided in line with the incision and the strap muscles are separated in the midline. The thyroid isthmus is then mobilized superiorly or is divided as needed to access the trachea. In the event of a low-lying cricoids cartilage, dissection on the anterior wall of the trachea helps to mobilize the trachea out of the mediastinum, and also the use of a cricoids hook will elevate the trachea to expose the second or third tracheal ring. Following identification of the second or third tracheal ring, a vertical tracheostomy is created or a tracheal flap (Bjork flap) is fashioned to create a fistulous tract by suturing the tracheal mucosal flap to the skin in the incision

3. Postoperative Care

The care of a tracheostomy tube after surgery is important. Highlighted below are some specific issues that all intensivists need to know when caring for patients with tracheostomies.

4. Wound and Dressing Care

Daily examinations of the stoma are important in identifying infections or excoriations of the skin at the tracheostomy site. In addition, keeping the wound clean and free of blood and secretions is very important, especially in the immediate post tracheostomy period. Dressing changes should be performed at least twice a day and when the dressings are soiled. Some authors recommend cleaning the stoma with 1:1 mixture of Hydrogen Peroxide and sterile saline. When changing dressings and tapes, special care is needed to avoid accidental dislodging of the tracheostomy tube.

5. Inner Cannulas

The inner Cannulas should be used at all times in the ICU. They serve to extend the life of the tracheostomy tubes by preventing the buildup secretions within the tracheostomy. The inner cannulas can be easily removed and either cleaned or replaced with a sterile, disposable one. Disposable inner cannulas have the advantage of quick and efficient changing, a decrease in nursing time, decrease risk of cross-contamination, and guaranteed sterility. The obturator should be kept at the bedside at all times in the event that reinsertion of the tracheostomy is necessary.

6. Humidification

One of the functions of the upper airway is to moisten and humidify inspired air. Since tracheostomy bypass the upper airway, it is vital to provide patients who have tracheostomy with warm, humidified air. Humidification of inspired gases will prevent complications in patients with tracheostomy Failure to humidify the inspired gases can obstruct the tube by inspisated secretions, impair mucociliary clearance, and decrease cough.

7. Suctioning

Patients with tracheostomy frequently have increased amounts of airway secretions coupled with decreased ability to clear them effectively. Keeping the airways clear of excess secretions is important in decreasing the risk of lung infection and airway plugging. Suctioning is frequently required in patients with poor or ineffective cough. Suction techniques should remove the maximal amount of secretions while causing the least amount of airway trauma. Routine suctioning, however, is not recommended.

8. Tracheostomy Tube Changes

Tracheostomy tubes do not require routine changing. In general, the tube only needs to be changed under the following conditions: (a) there is a functional problem with it, such as an air leak in the balloon; (b) when the lumen is narrowed due to the buildup of dried secretions; (c) when switching to a new type of tube; or (d) when downsizing the tube prior to decannulation. Ideally, a tracheostomy tube should not be changed until 7 to 10 days after its initial placement. The reason for this is to allow the tracheal stoma and the tract to mature. Patients who have their tracheostomy tube changed before the tract is fully mature risk having the tube misplaced into the soft tissue of the neck. If the tracheostomy tube needs to be replaced before the tract has had time to mature, the tube should be changed over a guide, such as a suction catheter or tube changer.

9. Complications of Tracheotomies

9.1. Immediate Complications (24 hours)

- Tube displacement
- Arrhythmia
- Hypotension
- Hypoxia/hypercapnia
- Loss of airway control
- Pneumothorax
- Pneumomediastinum
- Acute surgical emphysema
- Major hemorrhage
- Bacteremia
- Esophageal injury (uncommon)

- Cardio respiratory arrest (uncommon)
- Tracheo-laryngeal injury (uncommon)
- Crushed airway from dilatational tracheostomyuncommon

9.2. Intermediate Complications (from day 1 to day 7)

- Persistent bleeding
- Tube displacement
- Tube obstruction (mucus, blood)
- Major atelectasis
- Wound infection/cellulitis

9.3. Late Complications (>day 7)

- Tracheo innominate artery fistula
- Tracheomalacia
- Tracheal stenosis
- Necrosis and loss of anterior tracheal cartilage
- Tracheoesophageal fistula
- Major aspiration
- Chronic speech and swallowing deficit
- Tracheo cutaneous fistula

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16. URINERY CATHETERZATION

1. Indication:

Urinary catheterization is done when a person is unable to urinate using a toilet, bedpan, urinal, bedside commode or when accurate urinary output is required.



Figure 16.1: Diagrammatic presentation of Urinary Catheter assembly

2. Types of catheters:

2.1 Condom catheter:

Consists of a soft plastic or rubber sheath, tubing, and a collection bag for the urine. The sheath is placed over the penis and the collection bag is attached to the leg. Collects urine when there is no need for catheter insertion.

2.2 Straight catheter:

Is used when the catheter is to be inserted and removed immediately.

2.3 An indwelling catheter:

Also known as Foley catheter, is left inside the bladder to provide continuous urine drainage.

2.4 Suprapubic catheter:

It is a type of indwelling catheter. The supra-pubic catheter is inserted into the bladder through a surgical incision made in the Abdominal wall, right above the pubic bone.

2.5 3-way catheter:

For continuous bladder irrigation (CBI) is a type of indwelling catheter. Irrigate the bladder to prevent obstruction (i.e. bleeding)



Red rubber



Latex



Silicon



Figure 16.3: Catheterisation





3. Sizes & Subtypes:



Figure 16.4: Balloon retainer 3.1 Most common sizes are 8 F to 28 F.

3.2 1 F is equivalent to 0.33 mm = .013'' = 1/77'' of diameter.

Indwelling

3.3 Coude (French for elbowed) catheters have a 45° bend at the tip to allow easier passage through an enlarged prostate

"Council tip" catheters have a small hole at the tip which allows them to be passed over a wire

4. Caring for a Person with an Indwelling Urinary Catheter:

- The catheter tubing is secured loosely to the person's body near the insertion site using a catheter strap or adhesive tape
- Securing the tubing to the person's body prevents the catheter from being accidentally pulled out during repositioning
- The drainage bag is then secured to the bed frame at a level lower than the person's bladder.

5. Emptying Urine Drainage 7.3. Important aspects **Bags:**

- Urine measured at the end of each shift or every 4 • hourly in ward, every hourly in ICU.
- Urine drainage bags should also be emptied if they • are full.
- Leg bags need to be emptied frequently because they are smaller, and hold less urine.

6. Quantity:

- Be sure to monitor urine output
- Amount
- Characteristics (color. sediment. clarity, haematuria, odor)
- Less than **30 ml/hr** of urine indicates a problem

7. Catheter Insertion:

7.1. Equipment:

(check packages and expiry dates)

- Catheter tray (with drapes, fenestrated drape, cotton balls, swab holder, bowl)
- Catheter 14-16 Fr (for women) 12 Fr for young girls
- 16-18 Fr (for men) •
- Sterile drainage tubing with collection bag
- Correct size syringe (check catheter balloon)
- Sterile water •
- Cleansing solution: Chlorhexidine 4%, Povidone • Iodine 10%
- Lubricant anaesthetic jelly •
- Sterile gloves
- Specimen container
- Tape to anchor tubing

7.2. Assess

- Review physician's order and understand purpose • of inserting catheter
- Assess client (last urination, level of awareness, • understanding)
- Palpate bladder .
- Identify meatus and assess skin integrity .
- Identify potential difficulties (i.e. enlarged • prostate)

- Arrange equipment
- Wash hands
- Provide privacy
- Raise bed, stand on left side of bed if right handed (right side if left handed)
- Water proof pad under client
- Position & drape client



Figure 16.5



Figure 16.6

7.4 Position

7.4.1 Female: dorsal recumbent (supine with knees flexed) or Sims position (side-lying with upper leg flexed at knee and hip)



Figure 16.7

7.4.2 Male: supine position

With disposable gloves, wash perineal areas • Anatomy:



Figure 16.8: Male urinary tract



Figure 16.9: Female External Reproductive tract

7.5 **Procedure:**

- 7.5.1 Anaesthetic Lubricant jelly
- 7.5.2 Apply sterile drapes keep gloves sterile women: under buttocks and fenestrated over perineum
 - men: over thighs and fenestrated over penis
- 7.5.3 Place sterile tray and contents between legs
- 7.5.4 Cleanse meatus:





Figure 16.10: Cleaning of part Figure 16.11: Male Urethra Figure 16.12: Catheter in situ

7.4.5 Men: retract foreskin, hold penis below glans, maintain position of hand, with forceps clean in a circular motion from meatus down to base of glans, repeat three more times





Catheter in situ Figure 16.15

7.4.6 Women:

With non-dominant hand, expose meatus, maintain Position of hand, clean with forceps, wipe from front to back, each time take new cotton ball for each swipe, far labial fold, near and directly over meatus

7.6 Insertion:

- Hold end of catheter loosely coiled in dominant hand, place end of catheter in tray
- Insert catheter:

Women: Ask client to bear down as if to void, insert 5 to 7.5 cm or until urine flows, then advance another 2.5 to 5 cm

Men: Hold penis perpendicular, ask client to bear down, insert 17 to 22.5 cm or until urine flows, then advance to bifurcation

- Collect specimen if indicated
- Allow bladder to empty unless policy restricts (800 to 1000 ml) Pelvic floor blood vessels may become engorged from the sudden release of pressure,

leading to possible hypotensive episode. This may also cause painful bladder spasms.

- Inflate balloon with amount indicated (10 ml)
- If client complains of pain, aspirate solution and advance catheter further and inflate
- Gently pull to feel resistance
- Attach catheter to collection bag and attach to bed frame below bladder
- Anchor catheter (thigh if appropriate and coil tubing on bed and attach to mattress)

7.8 Evaluate:

- Palpate bladder
- Assess comfort
- Characteristics and amount of urine

7.9. Document:

- Report and record type and size of catheter
- Amount of fluid used to inflate balloon
- Characteristics of urine, amount, reason for catheter, specimens, client's response

8. Procedure for removal:

- Deflate the bulb completely with syringe
- If bulb cannot be deflated, then
- Cut the one-way valve
- Puncture the channel with needle
- Go on puncturing from distal most end to most proximal end
- USG guided suprapubic needle puncturing of the bulb
- Inflate further till it gets busted.

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17. TRIAGE

1. Introduction:

It is the method of quickly identifying the victims who have immediately life threatened injuries & have the best chance of surviving.

The process in which: The right patient in the right place at right time to receive the right level of care.

Triage involve a dynamic equilibrium between needs and resources

Needs= Number of wounded and types of wounds

Resource = Infrastructure and equipment at hand & competent personnel present

2. The Triage Team

- Triage team leader coordinator
- Clinical triage officer
- Head nurse, matron; chief organizer
- Nursing groups

3. Triage Documentation:

3.1 General information

Name, age, gender, chief complaints, history of past illness, mechanism of injuries, past medical and surgical history, allergy to food or drug and current medication, date of last tetanus immunization, last menstrual period for female age 11 to 60 years

3.2 Vital sign- temperature, Pulse, B.P & respiration etc.

- **3.3 Level of consciousness**
- 3.4 Visual inspection for deformities, laceration, bruising, rashes etc.

4. Triage Process

- Gathering information at point of triage.
- Perform initial assessments at point of triage.
- Triage process should be completed within five minutes.

4.1 Important steps

4.1.1 Start System:

Classification is based on

- Respiration
- Perfusion
- Mental Status

4.1.2 Shift

- Identify and remove
- Select those severely injured
- The dead
- The slightly injured
- The uninjured

4.1.3 Sort:

Categorize the most severely injured based on:

- Life-threatening conditions (ABC)
- Anatomic site of injury
- Red Cross Wound Score
- Treatment available in terms
- Serious wounds: resuscitation and immediate surgery
- Second priority: need surgery but can wait
- Superficial wounds: ambulatory management
- Severe wounds: supportive treatment

5. Categorization:

5.1 Category i) (<u>Red</u>): Resuscitation and immediate surgery

Patients who need urgent surgery lifesaving and have a good chance of recovery. (E.g. Airway, Breathing, Circulation: tracheostomy, haemothorax, hemorrhaging abdominal injuries, peripheral blood vessels)



Figure 17.1



Figure 17.2

5.2 Category ii (Yellow): Need surgery but can wait

Patients who require surgery but not on an urgent basis.

A large number of patients will fall into this group. (e.g. non-haemorrhaging Abdominal injuries, wounds of limbs with fractures and/or major soft tissue wounds, penetrating head wounds GCS > 8.)



Figure 17.4



Figure 17.5

5.3 Category iii (Green): Superficial wounds (no surgery, ambulatory treatment)

Patients with wounds requiring little or no surgery. In practice, this is a large group, including superficial wounds managed under local anesthesia in the emergency room or with simple first aid measures.



Figure 17.3





5.4 Category iv (Black): Very severe wounds (no surgery, supportive treatment)

Patients with such severe injuries that they are unlikely to survive or would have a poor quality of survival.

The moribund or those with multiple major injuries whose management could be considered wasteful of scarce resources in a mass casualty situation.



Figure 7

6. Hospital Triage Setting:

Doctor performs daily triage on a routine basis every day in emergency department.

6.1 Aim:

It is to identify those patients who have the highest degree of compromise for the purpose of providing rapid care to sickest patient first.

6.2 Hospital Triage System

- i. Emergent
- ii. Urgent
- iii. Non Urgent
- iv. Deceased

6.2.1 Emergent: Category 1 Colour Code RED

Those patients that are life threatening but likely to be amenable to rapid intervention.

Time required treatment immediately or within 15 to 30 minutes.

Example:

- Cardiac Arrest
- Airway Obstruction
- Hemorrhage with shock
- Complicated Delivery
- Seizures
- Asthma
- Acute Bleeding or Acute Pain
- Depressed Level of Consciousness

6.2.3 Urgent: Category 2 Colour Code YELLOW

Those conditions that are serious and if not treated in timely manner are likely to become critical. Time needed serious illness or injury that must be attended to, but wait upto two hrs. Example:

- Complex long bone fractures
- Bleeding Controlled with a pressure dressing
- Acute psychiatry problems
- High fever with another vital signs stable

6.2.4 Not Urgent: Category 3 colour code GREEN

Condition that can wait without likelihood of deterioration. Time needed, condition can wait for more than 2 hrs.

Example:

- Minor lacerations which requires sutures
- Minor joint trauma requires x-ray for diagnosis
- Chronic conditions that are stable such as preexisting skin rashes.

6.2.5 Category 4 colour code Black

Dead to be honored and to be taken care as marked in end session, if not possible by assigned agency earlier.

7. Conclusions:

- Triage is method of quickly identifying victims who have immediately life threatening injuries AND who have the best chance of surviving.
- Key elements of the START Triage Systems are: Respiration, Perfusion and medication.
- Reverse Triage is used for mass casualty incidents.

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9. Orthopaedics

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1. INFECTIONS IN ORTHOPEDIC

Infections of bone are difficult to treat due to precarious blood supply. Most common is Haematogenous spread.

1. Osteomyelitis

1.1 Classification:

Based on duration and mechanism of Infection **Duration:**

- Acute
- Sub-Acute
- Chronic

Mechanism:

- Exogenous Open fracture, Surgery or contagious
- Haematogenous

1.2 Factors increasing susceptibility to Infection:

- Patient dependent factors are nutritional and immunological status.
- Surgeon dependent factors are skin preparation, operation room, environment and prophylactic antibiotic therapy

1.3 Most Common Organism:

• Staphylococcus aureus

1.4 Type:

- 1) Acute
- 2) Sub-Acute
- 3) Chronic

1.5 Most Common Site:

Around Knee and Hip (in Paediatric cases)

1.6 Clinical Features:

- Pain and swelling
- Constitutional symptoms
- Warm to touch
- Restricted movements

1.7 Laboratory studies:

- HB, TLC, DLC & ESR
- Aspiration of fluid (>50000wbc/cu.mm.)
- Gram Stain sensitivity (30%)
- Culture sensitivity (90%)
- Blood Culture



Fig. 1.1: X-RAY SHOWING OSTEOMYELITIS OF TIBIA



Fig. 1.2: INFLAMMATION OF SKIN

1.8 Imaging studies:

- X-Ray
- Computed Tomography (CT)
- Radionuclide scanning
- MRI

1.9 Treatment:

1.9.1 Medical Therapy(Conservative)-

Immobilize the joint with splint for pain relief Antibiotic therapy of Osteomyelitis. Treat with intravenous Antibiotics for 3 to 6 weeks Cephalosporin and Aminoglycosides.

Paediatric dosage: -

- Inj. Cefotaxime 750 mg twice a day for 3 to 6 weeks.
- Inj. Amikacin 250 mg/ Inj. Gentamicin 20 mg IV twice a day for 3 to 6 weeks.

Adult Dosage: -

- Inj. Cefotaxime 1500 mg IV twice a day for 3 to 6 weeks.
- Inj. Amikacin 500mg / Inj. Gentamicin 40mg IV twice a day for 3 to 6 weeks.

Treat for at least 3 weeks with IV Antibiotics initially and then shift to oral for another 3 weeks.

Antibiotics should be used in accordance with Antibiotic protocol of Institute.

NSAIDs such as Tab. Diclofenac sodium 50 mg thrice a day along with Antacids until Inflammation subsides and supportive treatment such as multivitamins.

1.9.2. Surgical therapy-

- Debridement
- Sequestrectomy
- Saucerization
- Sinus tract excision

2. Tuberculosis of Bone

Bone is most common extra pulmonary site of Infection of Mycobacterium Tuberculosis.

2.1 Source:

Always secondary.

2.2 Site:

Most common site is spine followed by hip and Knee. Commonly occurs in first three decades of life

2.3 Agent:

Mycobacterium Tuberculosis



Fig. 1.3: PICTURE OF DESTRUCTION OF SPINE



Fig. 1.4: CROSS SECTION OF KOCH SPINE

2.4 Clinical features:

- Mono articular
- History of Night cries, Loss of weight, Anorexia
- Decreased joint movement
- Wasting of muscles
- Pain and Tenderness
- Cold abscess

2.5 Investigations:

- HB, TLC, DLC & ESR
- X-ray early De-calcification & Late Joint destruction

- Confirm diagnosis by biopsy or culture and **2.6.2 Operative**start treatment.
- MRI

2.6 Principles of Treatment:

- 1) General Support Protein rich diet & Haematinics.
- 2) Chemotherapy-Anti-tuberculous treatment as per RNTCP CAT-II.

2.6.1 Local Treatment-

Bed rest and Traction in acute and early stages, Splints and braces.

- 1) Capsulotomy
- 2) Synovectomy
- 3) Curettage
- 4) Excision

2.6.3 Abscess-

1) Incision and drainage.

2.7 Complication of Bone Infections:

- Pathological fracture
- Non-union / Mal union
- Para-paresis in spinal tuberculosis

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2.ARTHRITIS (JOINT DISEASES)

1.Osteoarthritis

It is degenerative disease of joint which commonly involves Knee, Hip and Ankle joint.

Primary Osteoarthritis is degenerative process without any precipitating factors.

Secondary Osteoarthritis is mostly due to Trauma, Infection and avascular necrosis.

1.1 Clinical Features:

- Pain
- Swelling
- Restricted range of movements

1.2 Investigations:

• X-ray of involved joint



Fig. 2.1: X-RAY SHOWING SEVERE OSTEOARTHRITIS CHANGES

1.3Treatment:

- a) NSAIDS -
 - Tab. Diclofenac sodium 50 mg Thrice a day for 1 week and SOS later Or
 - 2) Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
 - 3) Tab. Ibuprofen 400 mg Thrice a day for 1 week and SOS later

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.

 Local application of Diclofenac Sodium Gel 4-5 times per day.

- b) Local Hydrocortisone Acetate Injection 40 mg Intra articular once. Dosage can be repeated after 2-3 months as and when needed (under aseptic precautions).
- c) Surgery (joint replacement).

2. Ankylosing Spondylitis

Chronic progressive inflammatory disease of sacroiliac joints and the axial skeleton, having insidious onset affecting young patients age less than 40 years.

2.1 Clinical Features:

- Backache
- Restricted movements of spine and hip
- Morning stiffness, improvement with exercise, persistence for more than 3 months

2.2 Extra Articular Manifestations:

- Acute iritis
- Pericarditis
- Aortic incompetence
- Subluxation of atlanto-axial joints

2.3 Investigations:

- Haemogram
- HLA B27
- X-ray, Pelvis and Spine

2.4 Diagnostic Criteria:

HLA B27 positive and X-ray picture showing bamboo spine and ankylosed SI and hip joints.

2.5 Treatment:

- a) Conservative Management-
 - 1) Rest.
 - 2) NSAIDS.

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.

- 3) Physiotherapy / back exercise.
- 4) Occupational therapy.
- 5) General preventive measures like genetic counseling.
- b) Physiotherapy -



Fig. 2.2: X- RAY SHOWING BAMBOO SPINE IN ANKYLOSING SPONDYLITIS

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3. CONGENITAL DISORDERS

1. Congenital Torticollis (Wry neck)

- Usually due to contracted sternocleidomastoid muscle.
- Head tilted towards and chin rotated away from affected side.
- A firm swelling at junction of mid and distal 3rd of the muscle may be felt.
- Associated with congenital anomalies in cervical spine.
- Treatment includes stretching exercises, if deformity persists for 2 years surgical correction lengthening of sternocleidomastoid muscle.
- Early identification and immediate implementation of treatment gives good results. Earlier the treatment, better are the results.

2. Congenital Dislocation of Hip (CDH)

If displacement of femoral head from its normal position within the Acetabulum is found at birth. It is regarded as CDH or Developmental Dysplasia of the Hip (DDH).

2.1 Clinical Features:

- More common in females
- Asymmetric groin folds
- Shortening of limb
- Ortoloni and Barlow tests Performed to test the vulnerability of the Hip to dislocate and relocate within the acetabulum.
- Telescopy test Performed with Hip and Knee flexed in 90 degree and axial force applied to the thigh and the greater trochanter movements in relation to the ASIS (Anterior Superior Iliac Spine) are seen.
- X-ray shows broken Shenton's line
- Head lying outside the acetabulum
- Dysplastic acetabulum / femur
- Early identification and immediate implementation of treatment gives good results – Good physical examination
 - Plane Radiography
 - Ultrasonography

2.2 Various splints include:

- Von Rosen splint
- Pavlik harness
- Triple diapers
- Craig splint

2.3 Treatment:

2.3.1 Age 1 - 3 Years-

Initially closed reduction is tried. If it doesn't give the needed result, then surgical correction is opted. Surgery is planned after a period of traction. If the child is more than 18 months only surgical procedures can help.

The methods are

- 1) Removal of the interposed soft tissue from the joint.
- 2) Innominate osteotomy.
- 3) Femoral osteotomy.

2.3.2 More than 3 Years-

Preliminary traction followed by open reduction.

The methods are

- 1) Removal of limbus.
- 2) Salter innominate osteotomy.
- 3) Pemberton pericapsular osteotomy of ilium.
- 4) Rotation osteotomy of femur.

2.4 Complications:

- Avascular necrosis of femoral head.
- Neglected cases develop painful instability of hip and degenerative arthritis in later life.
- Persistent dislocations presenting in adulthood with OA can be treated with total hip replacement Arthroplasty.

3. Congenital Talipes Equino Varus (CTEV)

3.1 Etiology:

Unknown but mostly attributed to mechanical causes.

- Germ plasm defect and primary soft tissue abnormalities.
- Features are usually obvious at birth.
- Foot is twisted and turned inwards so that it faces postero-medially.
- Foot is plantar flexed, inverted and adducted at forefoot. The leg muscles are smaller and heel cord is tight and associated internal tibial torsion may be present.



Fig. 3.1: CLINICAL APPEARANCE OF BILATERAL IDIOPATHIC CTEV BEFORE TREATMENT

3.2 Treatment:

3.2.1 Conservative-

Aim of treatment is to produce and maintain a painless supple plantigrade foot. Preferably should be started within a day or two after birth. Manipulative correction and maintenance in cast.

Methods

- 1) French
- 2) Ponseti method produces best results which comprises correction of deformity in order of CAVE (Cavus, Adduction, Varus & Equinus) with serial castings followed by Tendo-Achillestenotomy and retention of correction in Steinbeck shoes till walking age.

3.2.2 Surgical correction-

Should be opted for in cases that have failed conservative line of management.

a) Soft tissue procedure

- 1) Closed Tendo Achilles Tenotomy.
- 2) Lengthening of medial and posterior structures.
- 3) Talonavicular reduction and maintenance.

Methods

- 1) Turco
- 2) Carroll
- 3) External fixator correction
- 4) JESS: Less than 7 years
- 5) Ilizarov: More than 7 years upto 12 years

b) Bone procedures

Done in case of deformity that has persisted for more than 3 yrs.

- In addition to posteromedial release lateral column shortening and / or medial column lengthening procedures can be done.
- Late / relapsed cases.

Options

- Soft tissue and bone procedures with tendon transfers, gradual correction using Ilizarov method.
- Neglected cases can be treated by triple arthrodesis.

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4. METABOLIC DISORDERS

1. Osteoporosis

Osteoporosis is defined as abnormally porous bone and its strength is less than normal for a person of that age and sex.

1.1 Clinical features:

- Bone pain
- Fractures
- Kyphosis
- Weakness
- Fatigue

1.2 Investigations:

- X-ray
- Special investigations (if needed) include
- DEXA Scan

1.3 Diagnosis:

SD > 2.5 below the average for premenopausal women for that population group.

1.3.1 X-ray features:

- Loss of trabecular definitions.
- Thinning of cortices.

1.4 Primary osteoporosis:

Physiological bone depletion that normally accompanies ageing and loss of gonadal activity.

1.4.1 Post-Menopausal Osteoporosis

• Complaints of back pain

• Increased thoracic kyphosis

1.5 Treatment:

- a) Physiotherapy.
- b) Bisphosphonate,
 - Alendronate 70 mg once weekly given with full glass of water before breakfast (in severe cases) for 3 months.
- c) Vitamin D 1000IU/ day for 3 months.
- d) Calcium both diet and drug Tab. Calcium carbonate 500 mg twice a day for 2-3 months and can be extended later.
- e) Hormone replacement therapy in selected cases.
- f) Calcitonin Nasal spray (200 IU/day) once a week for 3 months minimum, Injectable Calcitonin for limited use.
- g) If and when combination of Calcium and Vitamin D is available, it should be used.

1.6 Secondary Osteoporosis:

- Nutritional
- Endocrine
- Drug induced
- Malignant disease
- Identify the cause and treat it accordingly
- Correct the nutritional deficiency
- Correction of hormonal imbalance

1.7 Surgery:

• Vertebroplasty & Kyphoplasty –in presence of neurodeficit.

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5. REGIONAL CONDITIONS

1. Peri Arthritis Shoulder (Frozen Shoulder)

Significant restriction of both active and passive shoulder motion.

1.1 Clinical Features:

Night pain, inability to reach overhead and reach away from the body.

1.2 Investigations:

- X-ray
- Blood rule out metabolic causes
- Ultrasound (Dynamic ultra sound)
- MRI (Thickness of the capsule and synovium >4mm) to rule out other pathology

1.3 Treatment:

- a) Supportive Diathermy, Ultrasound, TENS.
- b) Medications
 - 1) Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
 - 2) Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
 - 3) Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.

- c) Intra- articular steroids. Inj. Hydrocortisone acetate 40 mg once every 3 months.
- d) Physiotherapy -Stretching exercises.
- e) Manipulation under general anaesthesia (if symptomatic > 6 months).
- f) Surgical Release (If not response to above treatment).

Open or Arthroscopic release at specialized centers.

2. Cubitus Valgus

- Outward deviation of forearm at elbow.
- Normal carrying angle is 100 in male and 150 in females.

2.1 Causes:

- Nonunion fracture of lateral condyle of humerus.
- Destruction of lateral condyle due to sepsis.

- Tardy-ulnar nerve palsy may be present treated with anterior transposition of ulnar nerve.
- Gross deformity supracondylar osteotomy.

3. Cubitus Varus

- Inward deviation of forearm at elbow.
- Most common cause is Malunion of supracondylar fracture.
- Change in arc of motion with hyper extension and restricted flexion.
- Deformity needs supracondylar osteotomy.

4. De Quervain's Disease

Chronic constrictive tenosynovitis of abductor pollicis longus and extensor pollicis brevis.

4.1 Clinical features:

Diffuse pain at distal end of radius laterally, tenderness and painful movements of thumb.

• Finkelstein's test positive.

4.2 Treatment:

- a) Early splint wrist and thumb in full extension.
- b) Anti-inflammatory drugs.
 - 1) Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
 - Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
 - 3) Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later.

NSAIDs cause hyperacidity hence antacids such as Cap Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.

- c) Local administration of Inj. Hydrocortisone 40 mg once every 3 months.
- d) Later soft tissue release can be done.

5. Carpel Tunnel Syndrome

Most commonly found in IT professionals and students. Median nerve is involved.

5.1 Clinical features:

It is caused by entrapment of medial nerve in carpel tunnel. Also seen in malunited lower end radius fractures and in pregnant women. Tinel's sign – It is reproduction of tingling sensation in palm and wrist by tapping over the course of nerve.

5.2Treatment:

5.2.1 Conservative-

NSAIDs

- Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
- 2) Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
- 3) Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later.

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.

Or

5.2.2 Surgical Release

6. Tennis Elbow

- Lateral epicondylitis due to repetitive stress Injury
- Commonly seen in house wife's and players:

6.1 Treatment:

6.1.1 Medical treatment-

- 1) Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
- Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
- Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later. NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.
- Local Steroids. Inj. Hydrocortisone acetate 40 mg once every 3 months.

Supportive splints.

6.1.2 Surgical release-

7. Golfers Elbow

7.1 Clinical features:

Medial epicondylitis which is due to repetitive stress. Injury commonly seen in house Wife's and Players.

7.2 Treatment:

7.2.1 Medical treatment-

- 1) Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
- 2) Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
- Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given.

 Local Steroids. – Inj. Hydrocortisone acetate 40 mg once every 3 months.

7.2.2 Surgical release

8. Avascular Necrosis of Femoral Head

Death of bone due to gradual vascular impairment or sudden infarction. Clinically there is pain in groin, radiates to thigh and knee, local tenderness, limitation of movements, loss of abduction and internal rotation, preserved flexion.

8.1 Radiological Features (X-rays):

- Stage1- Pre radiologic stage no radiologic findings.
- Stage 2- Osteoporosis, sclerotic cystic areas.
- Stage 3- Partial collapse, flattening of femur head, increased density and deformity of head.
- Stage 4 -Secondary deterioration of the articular cartilage.

8.2 Other Investigations:

- Radio-isotope scanning
- MRI most sensitive to detect ischemic necrosis
- Bone-marrow pressure measurements
- Intramedullary venography
- Core biopsy of femur head

8.3 Treatment:

- a) Early Bed rest, traction, weight reduction, withdrawal of steroid and alcohol.
- b) Stage 2 Core decompression of head, muscle pedicle bone grafting.
- c) Stages 3 & 4 Osteotomy and replacement arthroplasty.



Fig. 5.1: BILATERAL GRADE 4 AVASCULAR NECROSIS OF FEMUR HEAD

2)

3)

given.

c) Local Steroids-

1) Tab. Diclofenac sodium 50 mg thrice a day

Tab. Indomethacin 25 mg thrice a day for 1

Tab. Ibuprofen 400 mg thrice a day for 1

NSAIDs cause hyperacidity hence antacids

such as Cap. Omeprazole 20 mg or Tab.

Pantoprazole 40 mg once a day should be

1) Inj. Hydrocortisone acetate 40 mg once

for 1 week and SOS later Or

week and SOS later Or

week and SOS later.

Every 3 months.

2) Surgical release.

d) Ultrasound therapy

9. Plantar Fasciitis / Calcaneal Spur

9.1 Clinical Features:

- Pain in both heels
- Unable to bear weight
- Tenderness over medial tuberosity of calcaneum X-ray lateral view of calcaneum may show spur

9.2 Investigation:

X-ray Calcaneum lateral view to rule out spur Blood sugar to rule out diabetes.

9.3 Treatment:

- a) Soft sole pad in the heel
- b) Medical-

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6. DISORDERS OF SPINE

1. Lumbar Spondylosis

1.1 Clinical Features:

Low back ache is a very common problem faced in day to day life. Not all of them, are caused by disc pathology. Clinical features suggestive of disc pathology include backache associated with para spinal spasm, stretch pain and leg pain.

1.2 Investigation:

1) X-rays lumbosacral spine AP

1.3 Treatment:

- a) Rest.
- b) Medical management.
 - 1) Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
 - 2) Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
 - 3) Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later.

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20mg or Tab. Pantoprazole 40mg once a day should be given.

- 4) Local Application of Muscle relaxant gel such as Diclofenac sodium gel.
- c) Education about proper back posture maintenance of lumbar lordosis with erect posture or lumbar pillow.
- d) Physiotherapy interferential therapy, traction
- e) Epidural steroid Injections.

1.4 Operative Indications:

- Cauda Equina syndrome especially with bowel and bladder involvement.
- Patients with persistent pain not relieved by conservative measures for 6 8 weeks.
- Unilateral leg pain extending below knee that has lasted at least 6 weeks.
- Discectomy is performed usually by posterior approach and patients will have good post-Operative pain relief.

1.5 Watch for Red Flags Signs:

- Bowel-bladder involvement.
- Hypoesthesia around anus.

If you see any of the red flags refer urgently to higher center with surgical facility.

2. Cervical Spondylosis

It is a degenerative condition of the cervical spine occurring over 50 years of age. Involves the intervertebral discs, posterior intervertebral joints. Commonest at C5 - C6.

2.1 Clinical Features:

- Pain and stiffness.
- Radiating pain to shoulder or downwards on the outer aspect of the forearm and hand.
- Giddiness on and off.
- On examination there is loss of normal cervical lordosis and limitation in neck movements.
- Tenderness over the lower cervical spine or in the muscles of the paravertebral region.

2.2 Investigations:

X-ray cervical spine AP and Lateral

2.3 Treatment:

- a) Rest.
- b) Medical management.
 - 1) Tab. Diclofenac sodium 50 mg thrice a day for 1 week and SOS later Or
 - 2) Tab. Indomethacin 25 mg thrice a day for 1 week and SOS later Or
 - 3) Tab. Ibuprofen 400 mg thrice a day for 1 week and SOS later.

NSAIDs cause hyperacidity hence antacids such as Cap. Omeprazole 20 mg or Tab. Pantoprazole 40 mg once a day should be given

Local Application of Muscle relaxant gel such as diclofenac sodium gel.

- c) Education about proper Neck Posture.
- d) Physiotherapy, interferential therapy, Cervical traction.

3. Kyphosis

It is a posterior curvature of the spine.

Types

- i. Smooth rounded kyphosis
 - Scheuermann's disease
- ii. Angular kyphosis
 - Tuberculosis spine
 - Traumatic

4. Scoliosis

4.1 Definition:

Scoliosis is defined as lateral curvature of the spine. Scoliosis is named according to the level and side to which the main convexity of the curve is directed.

The common patterns are,

- i. Thoracic scoliosis
- ii. Thoraco-lumbar
- iii. Cervico-thoracic scoliosis (depending upon the site of primary curve)

4.2 Clinical Features:

Rib hump, Prominence of posterior chest wall. Test done to decide whether deformity is mobile or rigid. Make the patient lie in the lateral position on the concave side. Curvature is diminished in mobile cases.

4.3 Investigations:

- Radiography of the whole spine
- Degree of the curve is measured by the Cobb's angle
- Skeletal maturity by the fusion of the iliac apophysis (Risser sign) is an indicator of end of growth of spine

4.4 Treatment:

- a) Mild cases Conservative with serial custom made braces.
- b) Moderate cases Spinal and breathing exercise with correction by Milwaukee Brace.
- c) Severe cases Cobb's angle > 40 deg. surgical correction with instrumentation and Fusion carried out with cancellous bone graft.



Fig. 6.1: SCOLIOSIS OF DORSO – LUMBER SPINE

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7. GENERAL FRACTURE MANAGEMENT

1. Definition

Disruptions of bone tissue are called fracture; visible disruption of articular cartilage is also called fracture (Chondral fracture).

2. Types of fractures

- i. Closed fracture and Open fracture
- ii. Incomplete fracture or Greenstick fracture and Complete fracture
- iii. Linear fractures Transverse, Oblique
- iv. Spiral, Comminuted (more than two fragments).
- v. Segmental fracture, fracture with bone loss and Impacted fracture
- vi. Stress fracture
- vii. Pathological fracture

3. Gustilo-Anderson classification of open fracture

- Type I Open fracture with wound < 1cm Bone is not exposed.
- Type II Wound > 1cm without extensive soft tissue damage / skin flaps avulsion.
- Type III A Open fracture with extensive soft tissue damage, but with adequate soft tissue coverage of bone. It also includes segmental fracture, comminuted fracture with laceration < 1cm.
- Type III B Extensive soft tissue loss with periosteal stripping with bony exposure.
- Type III C Open fracture with an arterial Injury that require immediate repair regardless of size of the wound.

4. Clinical features

- Pain
- Swelling
- Inability to use the affected limb
- Deformity
- Crepitus
- Abnormal mobility
- Loss of transmitted movement

5. Investigations

- Routine X-ray AP and Lateral view
- Special views if necessary

6. Management includes

- a) Conservative management for closed fracture and Type I - open fracture for which closed manual reduction tried and patient put on appropriate Plaster of Paris splint for 3 - 4 weeks.
- b) Surgical management.

6.1 Initial management of open Injuries:

- Record vitals, start lifeline, I.V. fluids, I.V. Antibiotics, Analgesics.
- Immediately debride the wound of contaminated wound, devitalized tissue. Look for 4 C's, Consistency, Colour, Contractility, Circulation.
- Irrigate copiously with saline: Type I 3L saline, Type II 6 L saline, Type III 9 L saline.
- Enlarge the wound if necessary for adequate debridement.
- Remove contamination in the medullary canal.
- If wound can be closed, suture the surgically created wound, put loose stitch for other wounds over a drain if necessary. If closure is not possible, leave the wound open.

7. Complications

7.1 Acute:

- Shock
- ARDS
- Thrombo-embolism
- Neuro-vascular Injuries
- Crush syndrome
- DVT

7.2 Chronic:

- Delayed union, Non-union, Malunion
- Growth disturbances
- Joint stiffness
- Volkmann's ischemic contracture
- Myositis Ossificans
- Post-traumatic arthritis
- Avascular necrosis

7.3 Peculiar to Open fractures are:

• Infection

- Tetanus
- Gas gangrene
- Osteomyelitis
- Hypovolemic shock

8. Crush Syndrome

Severe crush Injury of limbs and muscles results in release of myoglobin leading to renal failure. Treatment is managing acute renal failure and Maintain hydration.

9. Compartment Syndrome

- It is due to ischaemic necrosis of structures of anterior compartment of fore-arm / legs.
- It is defined by the following signs (6 P's).
- a. Pain
- b. Pallor
- c. Paraesthesia
- d. Paralysis
- e. Pulselessness
- f. Positive Passive stretch

9.1 Treatment:

Emergency surgical decompression by fasciotomy.

10. Delayed union

Union is considered delayed, when healing has not advanced at the average rate for the location and the type of fracture, usually 3 - 6 months. Conservative management with functional cast for additional 4 - 12 weeks or ORIF with appropriate implant with or without bone grafting.

11. Non-union

A diagnosis of Non-union is justified either by clinical or X-ray findings which show healing has ceased, minimum of 9 months elapsed since Injury and the fracture shows no visible progressive signs of union for 3 months.

Time period -

- Fracture of long bone 6 to 9 months
- Fracture of neck of femur 3 months

12. Malunion

A malunited fracture is one that has healed with the fragments in a non - anatomical position. Corrective osteotomy with internal or external fixation with or without bone grafting.

13. Joint stiffness

Due to inappropriate fracture immobilization, intra articular fractures, periarticular adhesion of soft tissues, capsules and muscle contractures.

13.1 Treatment:

- a) Physiotherapy.
- b) Exercises.
- c) Manipulation under anesthesia
- d) Surgical excision and lengthening of contractures.

14. Myositis Ossificans

It is a reactive lesion (ossification) occurring in soft tissues and in stripped periosteum followed by trauma and ill-advised massage.

14.1 Treatment:

- a) Drugs:
 - Low dose Indomethacin (25 mg TDS) for 1 month.
- b) Mobilization and ROM excision till pain free joint range.

15. Fractures of Necessity

For these fracture, surgery is always necessary.

- Lateral humeral condyle fracture
- Femoral neck fracture
- Distal tibial epiphysis fracture

16. Physeal Injuries

Salter and Harris classification of Physeal Injuries.

- **Type–I** Epiphyseal separation through physis only with or without displacement.
- **Type–II** Triangular metaphyseal spike attached to separated epiphysis. (Thurston Holland sign.)
- **Type–III** Physeal separation with fracture through the epiphysis into the joint. If there is displacement joint incongruity occurs.
- **Type–IV** Fracture through the metaphysis, physis, epiphysis.
- **Type–V** Compression fracture of the physis producing growth arrest, diagnosed retrospectively.
- **Type–VI** Bruise or contusion to the periphery of the physis producing growth disturbances.



Fig. 7.1: EPIPHYSEAL INJURIES

16.1 Management:

Majority of children's fracture are treated conservatively. Few fractures require open reduction and internal fixation or closed pinning.

17. Open fractures in Children

Frequent and vigorous debridement with irrigation and adequate stabilization of fracture will reduce the rate of non-union and secondary infection. Rarely after closed reduction Haematogenous osteomyelitis occurs at the fracture site.

18. Splintage and Transport of Trauma Patient

18.1 Guidelines:

- 1) Immobilize one joint above and below.
- 2) Usage of readily available materials such as umbrella, Plaster of Paris, folded paper, wooden stick.
- 3) Splintage should neither be too tight or too loose.
- 4) Do Not transfer patient unless his vitals are stabilized.

Details of splinting and transport of trauma patient are mentioned in treatment of individual fractures.

18.2 Preparation of Cast/Slab Application:

It is essential to prepare the patient and all necessary material and equipment before beginning the processes of fracture reduction and cast application.

Required materials

- 1) Examination couch or Table
- 2) 2-4 rolls 150 mm padding
- 3) 8-9 Plaster of Paris(POP) rolls, 150 mm wide
- 4) Bucket with cool water
- 5) Pillows to support casted leg
- 6) Aprons to protect team members and patient
- 7) Paper to cover the floor

Everything must be assembled and ready before beginning the procedure.



Fig. 7.2: MATERIALS FOR APPLYING SLAB/CAST

18.3 Method:

Slab is prepared by overlaying of Plaster of Paris rolls of adequate length and layers to splint the fracture along with adjacent joints in anatomical position and applied over dorsal or ventral surface with cotton padding and bandage.

Cast is cylindrical application of Plaster of Paris rolls over adequate cotton padding over the fractured limb in anatomical position.

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8. POLYTRAUMA

1. Definition

- Injury severity score (ISS) 16 or above
- Systolic blood pressure below 80 mm Hg
- Glasgow coma score less than 15
- Higher fluid resuscitation requirements
- Chest, head, abdominal organ Injuries
- Fractures of more than one long bone

2. Stage of Care

2.1 Acute Resuscitation Period: (1-3 hours)

- From the first point of contact with medical service to the control of acute life threatening conditions.
- Rapid systemic assessment to identify life threatening conditions.
- Then Airway, Breathing, Circulatory (ABC) support should be made. This involves airway control, Thoracocentesis, rapid control of external bleedings, vigorous fluid and blood replacement therapy.
- Then complete diagnostic checkup if there is no acute life threatening situation.

2.2 Primary Stabilization Period: (upto 48 hours)

- From the control of acute life threatening situation and complete stability of Respiratory, Haemodynamic and Neurologic symptoms.
- Here acute management of fractures associated with arterial injuries and acute compartment syndrome are managed.
- Fractures are temporarily stabilized with external fixators and compartments.

2.3 Secondary Regeneration Period: (2 - 10 days)

- In this, general condition of the patient is stabilized and monitored.
- Systemic inflammation and multiple organ dysfunction syndrome are managed.
- Tertiary reconstruction and rehabilitation period (weeks to months) after trauma.
- Necessary surgical procedures.
- Definitive treatment of complete mid-phase fractures.
- Specialized procedures to achieve fracture correction or joint reconstruction.

2.4 Golden Hour:

First one hour after injury, with three-fold increase in mortality for every 30 minutes.

3. Follows ABCDE

- Airway
- Breathing
- Circulation
- Disability
- Exposure

4. Initial Management of patient in Shock

- a) Direct control of obvious bleeding by direct pressure (preferable), tourniquet clamping of blood vessels
- b) Large-bore intravenous access
 - 1) Ringer lactate infusion
- 2) Blood replacement as indicated, by serial haematocrit estimation and blood pressure.
- c) Traction with Thomas Splints or extremity splints to limit hemorrhage from unstable fractures.
- d) Consideration of angiography or immediate operative intervention for Hemorrhage control.

4.1 Haemorrhagic Shock:

- Diagnosed by Hypotension, Tachycardia, seen in patients with large open wounds, active bleeding, Pelvic and / Femoral fractures and Abdominal or Thoracic trauma.
- In the absence of open hemorrhage bleeding into voluminous space (chest, abdomen, pelvis, thigh) must be ruled out.
- This may require Peritoneal lavage, Angiography, CT, MRI.
- Managed by aggressive fluid replacement, blood transfusion, Angiographic embolization operative intervention, fracture stabilization, etc.

4.2 Blood Replacement:

- Fully cross matched blood is preferable
- In case of life threatening situations 'O'negative blood can be used.
- Warming of blood prior to administrations prevents Hypothermia

• Monitor coagulation factors, Platelets and Calcium levels.

5. Indications for Immediate Surgery

- Hemorrhages secondary to Liver, Splenic renal parenchymal injury –Laparotomy.
- Aortic, Caval or Pulmonary vessel tears Thoracotomy.
- Depressed skull fracture or acute intracranial hemorrhage Craniotomy.
- Disability (Neurologic Assessment).
 - a. Assess level of consciousness by GCS.
 - b. Pupillary response sensation.
 - c. Motor response in all extremities.

d. Rectal tone and sensation.

- GCS = Eye opening score + Verbal (intubated or non-intubated) score + Motor score.
- GCS if < 13, systolic BP < 90, RR< 10 / min or > 30 / min warrants Emergency trauma care.

6. Radiographic Evaluation

- X-rays of Skull, pelvis, spine and Extremities
- Ultra sound abdomen
- CT, MRI

Assess the concomitant Injuries such as head Injuries, thoracic Injuries, and genitourinary Injuries and refer the patient to higher centers for tertiary care management.

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- 3. Chapman MW, Williams L, Wilkins. Chapman's Orthopaedic surgery. 3rd edition



9. PELVIC INJURIES

1. Introduction

- It is an emergency in Orthopaedics which involves multispecialty intervention.
- Mode of Injury.
 - a. High energy injuries
 - b. Crush injuries
- c. Impact injuries
- Associated with complications and other fractures.

2. Clinical Features

- Pain and Tenderness at affected site •
- Range of movements painful
- Shock •
- Intra-abdominal / Urethral / Vascular injuries •

3. Management

- a) Evaluate ABCDE and stabilize the patient.
- b) Evaluate for other associated injuries Head, Chest, Abdomen and Spine.
- c) If Haemodynamically stable. assess radiologically with X-ray pelvis AP view, inlet and outlet view. If there is no or minimal displacement, treat conservatively with strict bed rest and analgesics.
- d) If there is displacement with anterior opening type (unstable Fracture) then assess the displacement.
- e) If it is < 2.5 cm treat conservatively.

Bibliography

- f) If it is > 2.5 cm external fixation or Open reduction and Internal fixation.
- g) If there is unstable fracture with vertical displacement, then treat with ORIF.
- h) If Haemodynamically unstable, then stabilize the Pelvis with external fixator at the casualty itself without shifting the patient and follow the above steps.

4. Associated Complications and Treatment

- a) Shock-blood transfusion and fluid replacement.
- b) Embolisation of bleeding Pelvic vessels. (intervention radiology)
- c) Urethral rupture diagnosed by blood in Urethra, Perineal hematoma, Distended bladder, managed by Urologist.
- d) Bladder rupture-extravasation of urine-Urologist and Surgeon.
- e) Bowel and intra-abdominal injuries are managed by General surgeon.
- f) Thrombosis DVT prophylaxis, vascular opinion must be obtained.
- g) Post-operative-
 - 1) Infection rate (0-25%) managed accordingly
 - 2) Thrombo-embolism
 - 3) Pin tract infection
 - 4) Death inevitable in certain situations

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10. FRACTURES OF UPPER LIMB

1. Mode of Injury

- a. Fall on outstretched hand
- b. Road Traffic Accident
- c. Associated Poly Trauma

2. Type of Fracture

1. Fracture of Hand:

- i. Fracture of the base of the first Metacarpal
- ii. Fractures of the other Metacarpal bones
- iii. Fractures of the Phalanges

2 Fractures around Wrist:

- i. Fracture of the Scaphoid bone
- ii. Fractures of other Carpal bones
- iii. Dislocations of the Carpal bones
- iv. Fracture of the lower end of the Radius (Colles' and Smith's)
- v. Galeazzi fracture dislocation
- vi. Fracture separation of the lower Radial epiphysis
- vii. Fracture of the shafts of the forearm bones

3 Fractures around Elbow:

- i. Fracture of the head and neck of the Radius
- ii. Fracture of the upper end of the Ulna with dislocation of the head of the radius (Monteggia fracture dislocation)
- iii. Fracture of the Coronoid process
- iv. Fracture of the Olecranon process
- v. Fractures of the Epicondyles and Condyles
- vi. Supracondylar fracture

4 Fracture of the Humerus:

i. Fracture of the Shaft

- ii. Fracture of the Greater tuberosity
- iii. Common Fracture of Proximal Humerus/ Fracture of greater tuberosity

5 Fractures of the Shoulder Girdle:

- i. Fractures of the Clavicle
- ii. Fractures of the Scapula

2.1 Fracture of Hand:

2.1.1 Fractures of base of the first metacarpal-Bennett's Fracture

Intra-articular fracture through base of first Metacarpal bone in which the fractured shaft is displaced laterally due to unopposed pull of APL. (Abductor Pollicis Longus)

a) Investigation -

• X- ray of hand Antero posterior and oblique view

b) Treatment -

- 1) Primary Stabilization in anatomical position with the help of available splinting material.
- 2) Closed or open reduction and internal fixation with 'K' wire.

A. Rolando Fracture

It is a common extra articular fracture of base of first metacarpal bone.

a) Investigation -

- X-ray of hand Antero-posterior, Lateral and Oblique views.
- b) Treatment -
 - 1) Primary stabilization in anatomical position with the help of available splinting material.
 - 2) Closed or open reduction and internal Fixation with 'K' wire ORIF by K wire.
 - 3) External fixation
 - 4) Mini fragment 'T' plate fixation

2.1.2 Fracture of the other Metacarpal Bones

a) Investigation -

• X-ray of hand Antero-posterior and Oblique views.

b) Treatment -

- 1) Primary stabilization in anatomical position with the help of available splinting material.
- 2) Conservative Cock up splint.
- 3) Surgical Close or Open reduction and internal fixation with K wire or mini plate.
- 4) External fixation for compound (open) fracture.

2.1.3 Fractures of the Phalanges

a) Symptoms -

Pain & Swelling and bony deformity with loss of active movements.

b) Signs -

Tenderness at Fracture site along with Crepitus and Abnormal Mobility.

c) Investigation -X- ray of hand AP and Oblique views.

d) Treatment-

- 1) Primary stabilization in anatomical position with the help of available splinting material.
- 2) Conservative Buddy strapping, POP slab, finger splints



Fig. 10.1: FINGER SPLINT

 Surgical – K (Kirschner) wire or JESS (Joshi's External Stabilization System) fixator.

2.2 Fractures Around Wrist:

2.2.1 Fracture of the Scaphoid bone-

It is common in young adults usually caused due to fall on outstretch hand, often overlooked.

- a) Symptoms Pain, Swelling and Restriction of wrist movement.
- **b)** Signs Tenderness in anatomical snuff box and impairment of wrist movement.
- c) **Investigations** X-ray of wrist in AP, lateral and two oblique projections.
- **d) Treatment** Primary stabilization in anatomical (glass holding) position with the help of available splinting material.
 - 1) **Conservative** Plaster immobilization usually till 2 to 3 months.
- 2) **Surgical** Closed or open reduction and internal fixation with a special compression screw.

e) Complications:

- The incidence of complications is high.
 - Delayed or non-union
 - Avascular necrosis
 - Osteoarthritis

2.2.2 Fractures of other carpal bones-

These are less common serious fractures. The treatment is conservative management with plaster immobilization.

2.2.3 Dislocations of the carpal bones-

Complete dislocation of the wrist is very uncommon and incomplete carpal dislocation involve one or more of the carpal bones and are as follows:

- 1) Dislocation of lunate bone.
- 2) Perilunar dislocation of the carpus.
- a) **Symptoms** Pain, Swelling, Deformity and restriction of movement of the wrist joint.
- **b)** Signs Tenderness and Loss of anatomical position of wrist and hand.

- c) Investigations -
 - X-ray of the wrist in AP, lateral and oblique views.

CT Scan

- d) Treatment -
 - 1) Primary stabilization in anatomical (glass holding) position with the help of available splinting material.
 - Conservative Manipulative reduction under anaesthesia and plaster immobilization.
 - 3) Surgical operative replacement of carpal bones.
- e) Complications -
 - Avascular necrosis
 - Osteoarthritis
 - Injury to median nerve

2.2.4 Fracture of the lower end of the radius

A. Colles' fracture - It is a fracture of the lower end of Radius at metaphyseal region which manifests in bent fork deformity due to dorsal and radial displacement of the distal fragment.

B. Smith's fracture – It is reverse of the Colles' fracture with ventral displacement.

a) Symptoms -

Pain, Swelling and bony deformity of the wrist with loss of active movements

b) Signs -

Tenderness at fracture site along with or without Crepitus and abnormal mobility

c) Investigation -

- X-ray of forearm with wrist AP and Oblique views.
- X-ray Wrist in Ulnar deviation.

d) Treatment -

- 1) Primary stabilization in anatomical (glass holding) position with the help of available splinting material.
- 2) Close reduction and plaster immobilization.
- 3) Close reduction with K wire fixation.
- 4) Close reduction with K. wire fixation and external distractor application.
- 5) Open reduction and internal fixation with multiple K wires, plate and screws.

2.2.5 Galeazzi fracture – dislocation-

It is the fracture of radial diaphysis at the junction of middle and distal third of the shaft with associated disruption of distal radio ulnar joint.

a) Symptoms -

Pain, Swelling, Deformity and Shortening of the forearm.

b) Signs -

Bony Deformity & Tenderness, Crepitus & Pain aggravated by passive stretching of wrist.

c) Investigation -

X- ray forearm with Elbow and Wrist in AP and lateral projection.

- d) Treatment -
 - Primary stabilization in anatomical position by applying gentle axial traction and splinting with the help of available material. (moulded splints, folded newspaper, card board or wooden plank)
 - 2) Surgical
 - Mostly needed for prevention of Malunion
 - Open reduction and internal fixation with plate and screws

2.2.6 Fracture – separation of the lower radial epiphysis-

It is seen in children before Physeal fusion in which the epiphysis is fractured and separated from the metaphysis and results into gross deformity, if not treated adequately. The clinical features and management are same as that of fracture of lower end of radius in adults except surgical intervention which is rarely needed.

2.2.7 Fracture of the shafts of the forearm bones-

The forearm bones, radius and ulna get fractured either single or both and are very common Injuries which result into gross deformity and functional restriction, if not treated.

- a) Symptoms-Pain, Swelling over forearm and bony deformity.
- b) Signs -Tenderness, Crepitus, Abnormal mobility with or without signs of compartment syndrome.
- c) **Investigation** -X-ray of the forearm with wrist and elbow in AP and lateral projections

d) Treatment:

- Primary stabilization in anatomical position by applying gentle axial traction and splinting with the help of available material. (moulded splints, folded newspaper, card board or wooden plank)
- 2) Splintage.



Fig. 10.2: SPLINTAGE FOR FRACTURE RADIUS/ULNA AND LOWER END OF HUMERUS

- 3) **Conservative** Closed reduction and plaster application under anaesthesia, mostly in children.
- 4) Surgical -
 - Closed reduction and internal fixation with elastic (in children) or rigid intramedullary nails.
 - Open reduction and internal fixation with plates and screws.
 - External fixator application in case of open fractures.

2.3 Fractures Around Elbow:

2.3.1 Fracture of the head and neck of the radius-

One of the commonest fractures of the upper limb in young adults.

a) Symptoms -

b) Signs -

Sharp local Tenderness and impaired movements. (Flexion of elbow and pronation and supination)

 c) Investigations – X-ray of elbow joint with forearm in AP and lateral projections.

Pain and Swelling on lateral aspect of Elbow and restriction of movement.

d) Treatment -

- Primary stabilization in anatomical position (flexion at elbow and mid prone position of forearm).
- Conservative in case of slight damage to the radial head and neck with plaster immobilization.
- 3) Surgical -
 - Open reduction and internal fixation in case of severe damage to the radial head and neck.
 - Excision of radial head in severely damaged and displaced fractures.

2.3.2 Fracture of the upper end of the ulna with dislocation of the head of the radius (Monteggia fracture – dislocation)-

This is uncommon Injury with characteristic displacement, the ulna is angled forwards and the head of radius is dislocated forwards which is obviously seen on clinical and X-ray examinations. Clinical features are same as the forearm fractures.

a) Treatment -

- 1) Accurate fracture reduction is essential. It is seldom possible to reduce both the dislocation and fracture by closed method.
- 2) Open reduction and internal fixation with plate and screws is essential.

2.3.3 Fracture of the coronoid process-

The Coronoid process is seldom fractured unless in association with posterior dislocation of the Elbow.

2.3.4 Fracture of the olecranon process-

The Olecranon process is fractured by a fall on the point of the elbow usually in adults.

Clinical feature same as fractures above with distinct disruption of three-point bony relation of the Elbow. (It is a triangle formed by the tip of Olecranon and the two epicondyles)

a) Treatment -

The treatment depends upon the type of fracture i.e. plaster immobilization in case of crack fracture, internal fixation for fracture separation and excision in case of comminuted fracture.

2.3.5 Fractures of the condyles and epicondyles

Condylar fractures are relatively uncommon, but often troublesome and occur mainly in children.

- a) Symptoms Pain, Swelling and Restriction of movement of the elbow.
- b) Signs -
 - Marked tenderness and widening of the Elbow.
 - Disruption of the three-point bony relation.

Complications -

- Injury to the Brachial artery leading to Volkmann's ischaemic contracture in delayed or neglected cases.
- Injury to median nerve
- Malunion

2.3.6 Supracondylar fracture-

a) Clinical features -

Pain, Swelling and Deformity.

- **b) Investigation -**X-ray Elbow, AP and lateral views.
- c) Treatment -
 - 1) Conservative plaster immobilization.
 - 2) Surgical Closed or open reduction and internal fixation with K wires or cancellous screws.

2.4 Fracture of Humerus:

2.4.1 Fracture of the shaft-

a) Clinical Finding -

Pain and Swelling over arm abnormal mobility, crepitus.

- b) Investigation -
 - X-ray of arm with shoulder & elbow AP and Lat.
- c) Treatment
 - 1) Splintage



Fig. 10.3: SPLINTAGE OF FRACTURE OF HUMERUS

- 2) Surgical Closed nailing by
- Rush nail
 - Ender's nail

Children

- Flexible nail
- Humerus Inter locking Nail.
- Open reduction and internal fixation with plate & screws.
- External fixator

Watch for wrist drop. (radial nerve injury pre and post reduction)

2.4.2 Fracture of Surgical Neck Humerus

a) Clinical Finding -

- Pain and Swelling over Shoulder
- Painful movements of Shoulder
- Crepitus, Tenderness

b) Investigation -

X-ray shoulder AP and Lateral view (special views if needed)

- c) Treatment -
- 1) Conservative immobilization
- 2) **Surgical** K wire fixation Buttress plating

2.4.3 Common Fracture of Proximal Hume b) Investigation -

Fracture of greater tuberosity -

a) Clinical Finding -

- Pain and Swelling over Shoulder.
- Painful movements of Shoulder.
- Crepitus, Tenderness.
- b) Investigation -X-ray Shoulder AP & CT Scan
- c) Treatment -
- 1) Conservative Arm to chest strapping.
- 2) Surgical –Cancellous screws or plate fixation.
- 3) Primary shoulder replacement.

2.5 Fractures of the Shoulder Girdle:

2.5.1 Fracture of Clavicle -

a) Clinical Finding -

- Pain and Swelling over shoulder.
- Bony Deformity
- Crepitus, Tenderness

Fig. 10.4: FIGURE OF 8 BANDAGE

- X-ray Shoulder AP
- c) Treatment -
 - Conservative Figure of '8' bandage. (Clavicle brace)
 - Surgical Closed reduction and K wire fixation for fracture of lateral end and open reduction and internal fixation with plate and screws for mid shaft fractures.

2.5.2 Fracture of Scapula -

a) Clinical -

Pain and Swelling over shoulder painful movements of shoulder.

b) Investigation -

- X-ray Shoulder AP
- c) Treatment Shoulder immobilization

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11. FRACTURE OF LOWER LIMB

1. Mode of Injury

- 1) Road Traffic Accident
- 2) Associated Poly Trauma

2. Type of Fracture

- i. Fracture Upper End Femur
 - Intra capsular
 - Extra capsular
 - Femur head fracture
- ii. Fracture Shaft Femur.
- iii. Fracture Lower End Femur.
 - Intra articular
 - Extra articular
- iv. Fracture Patella
- v. Fracture Upper End Tibia
- vi. Fracture shaft Tibia
- vii. Fracture around Ankle joint
- viii. Fracture of bones of foot

Splinting of Lower Limb Injuries



Fig. 11.1: STEP 1 TRACTION



Fig. 11.2: STEP 2 LIMB SPLINTING



Fig. 11.3: STEP 3 BUDDY STRAPPING



Fig. 11.4: ALTERNATE METHOD OF SPLINTING

3. Fracture Neck of Femur (Intra-capsular)

Introduction-

Fracture neck of Femur more common in old age due to Slip and fall.

3.1 Classification According to the site of fracture:

- Subcapital
- Transcervical
- Basicervical

Usually caused by trivial fall in the elderly due to presence of Osteoporosis, however metastasis from malignancies can also lead to the pathologic fractures.

The aim of treatment is to achieve union of the fracture and a durable hip joint afterwards

3.2. Principles of management include:

3.2.1 Osteosynthesis

- i. Screws, Moore's Pins etc.
- ii. DHS, Blade Plate.
- iii. Internal Fixation + Fibular Grafting.
- iv. Muscle pedicle Graft + Internal Fixation.

3.2.2 Osteotomy

- i) Pauwels valgus osteotomy
- ii) Mc Murray's osteotomy

3.2.3 Arthroplasty

- Hemiarthroplasty
- Total Hip Replacement
- Excision Arthroplasty

4. Trochanteric Fractures (Extra-Capsular Fracture Neck Femur)

4.1 Fracture of Greater Trochanter

This is isolated fracture of greater trochanter due to fall on the side of Pelvis, usually minimally displaced and managed with pelvic stripping and non-weight bearing.

4.2 Fracture of Intertrochanteric

Region:

Intertrochanteric Hip fractures are more common in road traffic accidents in young adults and domestic fall in elderly.

4.3 Fracture of Sub Trochanteric Region:

Occur between lesser trochanter and a point 5 cm distally and are seen as independent entities or as an extension of intertrochanteric fractures.

4.4. Classification

4.4.1 Trochanteric fractures are classified according to Boyd and Griffin classification

4.4.2 Sub trochanteric fractures are classified according to Seinsheimer classification

A. Clinical features:

- **Symptoms** Pain, Swelling, Bruising, shortening of the limb and inability to stand or walk.
- Signs -

- i) Severe Tenderness and Swelling around hip region.
- ii) Shortened and externally rotated lower limb.
- iii) Inability to do straight leg raising.

• Investigations:

- i) X-ray of pelvis with both hips in AP view and affected Hip in Axial view
- ii) CT Scan

• Treatment:

- i) Conservative Initial traction and immobilization in NRB (Non Rotatory Boot) in Undisplaced fracture
- ii) Surgical -
 - Closed reduction and internal fixation with Dynamic Hip Screw and plate (DHS)
 - Proximal Femoral Nail (PFN)
 - o Intramedullary Hip Screw
 - Dynamic Condylar Screw (DCS)

For sub trochanteric fractures a DCS is the implant of choice after closed or open reduction. Other modalities like PFN, Angled blade plate, Reconstruction nail or Modified Kuntscher nail may also be used as per the preference of the surgeon.

5. Fracture Shaft of Femur

Fractures of the shaft of the femur are mostly due to high-energy trauma.

5.1 Types of Femoral Shaft Fractures:

- Type I Spiral or transverse (most common)
- Type II Comminuted
- Type III Open

5.2 Investigations:

X-ray shaft Femur with X-ray of Pelvis and Knee for associated injuries.

5.3 Treatment:

5.3.1 Conservative management-

Conservative management of fractures in children in Spica cast or with skeletal traction.

5.3.2 Surgical management-

- Kuntscher nail for Isthmic fractures
- Interlocking Nailing in comminuted fractures
- Plating for lower third fractures
- Plating of shaft Femur fracture in children

Blood transfusion if needed and correct hypovolemic shock.

6. Fracture of Tibia/Fibula

6.1 Introduction:

Mostly due to road traffic accident

- **6.2 Signs /Symptoms:**
- Pain, Swelling, Tenderness

6.3 Investigation:

• X-ray of Tibia along with X-ray of knee and ankle joint.

6.4 Treatment:

6.4.1 Conservative - In Undisplaced fracture, closed reduction and above knee cast for 12 weeks.

6.4.2 Surgical – Displaced fracture, Interlock nail or plating depending on fracture anatomy.

7. Fracture Calcaneus

7.1 Mode:

Fall from height.

7.2 Sign /Symptoms:

• Swelling, Oedema, Pain, Tenderness.

7.3 Investigation:

- X-ray of heel
- CT scan for intra articular fractures.

7.4 Treatment:

- Conservative (Boot Cast). Strict non-weight bearing till fracture heals.
- Intra-articular fractures of Calcaneus involving sub-talar joint should preferably treated with restoration of anatomical fracture geometry is must.

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12. DISLOCATIONS

Loss of Alignment of joint surfaces which should be treated as emergency.

1. Shoulder Dislocation

Most common dislocation.

1.1. Types

- i. Anterior
- ii. Posterior
- iii. Inferior (Luxatio erecta)



Fig. 12.1: X-RAY PICTURE OF SHOULDER DISLOCATION

1.2 Mechanism:

- Most commonly indirect / direct violence
- Posterior dislocation is common in Electric shock and Convulsions.

1.3 Clinical Features:

1.3.1 Anterior-

• Patient comes with injured shoulder in Abduction and External rotation.

1.3.2 Posterior-

• No striking deformity, Shoulder in Abduction and Internal rotation.

1.3.3 Luxatio erecta-

- Salute position Abduction forward elevation.
- Severe Pain, Neuro vascular deficit, more with latter two.

1.4 Investigation:

- X-ray
 - AP, Scapular view, Axillary view, CT scan

1.5 Treatment:

- Reduction Traction Counter Traction, Abduction, Extension, External rotation – Reduction.
- Stimson Sedation and prone position with 5 lbs. weight
- Surgery
- Irreducible dislocation
- Displaced fracture dislocation with ORIF

1.6 Complications:

- Recurrent dislocation
- Axillary Nerve and Artery injury
- Brachial plexus injury
- Stiffness

2. Elbow Dislocation

Most common- Posterior Dislocation of Elbow

2.1 Mechanism:

- Posterior Elbow Hyperextension, Valgus stress, Arm Abduction, Fore-arm Supination.
- Anterior-direct force over posterior fore-arm with Elbow in Flexed position.

2.2 Clinical Features:

- Gross instability
- Swelling
- Three-point bony relation altered
- Associated Injuries Radial head and Coronoid Fracture

2.3 Investigation:

- X-ray Elbow AP and Lateral
- Closed manual reduction under GA, followed with above Elbow posterior slab 90-degree Flexion.

2.4 Operative Indications:

- Re-dislocation
- Non-concentric Reduction
- Surgery: Open reduction and repair of soft tissues, Hinged external fixation, pinning
- Neurovascular Injury
- Femoral head fractures
- Heterotopic ossification
- Recurrent dislocation
- Thromboembolism

2.5 Complications:

- Loss of motion
- Neurologic compromise
- Vascular Injury
- Compartment syndrome
- Re-dislocation
- Myositis Ossificans

3. Hip dislocation

3.1. Types of dislocation:

3.1.1 Posterior-

Posterior dislocation is the most common type of dislocation. Occurs mainly due to dash board Injury.

3.1.2 Anterior-

Anterior dislocation occurs because of blow to the back in squatting position.

3.1.3 Central dislocation-

Occurs due to direct blow over the trochanter

3.2. Clinical Features:

3.2.1 Posterior-

- Limb in flexion, adduction, internal rotation, limb shortening
- Sciatic nerve Injury

3.2.2 Anterior-

- Limb in flexion, abduction, external rotation, limb lengthened
- Injury to femoral nerve.

3.3 Investigation:

- X-ray.
- Hip AP and lateral.
- CT, MRI.

3.2 Management:

- a) Resuscitate, CPR attempted with in-line traction with patient lying supine, under general anesthesia.
- b) Methods used are the classical Watson-jones, Bigelow and reverse Bigelow, Allis, Stimson gravity method.
- c) Maintain it with skeletal traction.
- d) If irreducible, nonconcentric, ipsilateral neck Fracture or Acetabular Fracture then open reduction is done.

3.5 Complications:

- Osteonecrosis
- Post-traumatic osteoarthritis of the joint

4. Knee dislocation

- Mode of Injury: high energy / low energy
- Hyperextension with or without Varus / valgus



Fig. 11.2: X-RAY PICTURE OF KNEE DISLOCATION

4.1 Clinical features:

- Gross knee distortion is present
- A neurovascular examination must be done

4.2 Investigations:

- First reduce the dislocation
- Then take X-rays AP and lateral, 45-degree oblique, patellar sunrise views
- MRI
- Arthroscopy
- Assess the ligament Injuries

4.3 Treatment:

- a) Immediate closed reduction, avoid direct pressure over the popliteal fossa after reduction, splint at 20 - 30 degrees of flexion.
- b) Operative indications:
 - o Unreduced, residual soft tissue interposition
 - Open Injuries, ORIF with ex-fix.
 - o Reconstruction of ligaments at later setting.

4.4 Complications:

- Limited range of movements
- Ligamentous laxity and instability
- Vascular compromise
- Nerve traction Injury

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13. LIGAMENTOUS INJURIES

1. Mode of Injury

Indirect, Twisting or Bending Forces on the Knee.

2. Clinical Features

• Pain, Swelling, Tenderness, Loss of range of motion and Positive Patellar Tap.

3. Investigations

• X-ray of associated joint and MRI

4. Treatment

- a) Initial management with Brace/Splint and NSAIDs
- b) Conservative management with Bracing and Physiotherapy

Bracing for 2 weeks followed by MRI

c) Operative: Arthroscopic ligament repair.

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14. SPINAL TRAUMA

1. Spinal Cord Injury (SCI)

It is an insult to the Spinal Cord resulting in a change, either Temporary or Permanent, in its Normal Motor, Sensory or Autonomic Function.



Fig. 14.1: HEAD TO TOE STABILIZATION IN CASE OF SPINAL INJURY ON SPINE BOARD

2. Optimal Diagnostic Criteria, Investigations, Treatment & Referral Criteria

2.1 Rural/ Sub District Hospital:

Optimal Standards of Treatment in Situations where technology and resources are limited.

2.1.1 Clinical Diagnosis-

After the ABC has been taken care of, the patient is gently log rolled and whole of the Spine is Palpated for Tenderness or a palpable step-off deformity. Neurogenic shock, Incontinence of Bowel, Bladder and Penile erection indicate severe spine Injury. A careful and detailed Neurological examination is then performed and meticulously documented.

2.1.2 Frankel's grades-

Spinal Cord Injury is most commonly graded using Frankel's grades (A to E).

- A: Complete Motor and Sensory loss
- B: Sensation only present below lesion
- C: Sensations present and Motor function is present but useless
- D: Motor useful but not normal
- E: No neurological deficit.

Table 1: ASSESSMENT OF MOTOR FUNCTION

C3-5
C4
C5
C6
C7
C8
T1-T12
L2
L3-4
L5-S1
S1-2

Table 2: ASSESSMENT OF SENSORYFUNCTION

Deltoid area	C5
Thumb	C6
Middle finger	C7
Little finger	C8
Nipple	T4
Xiphoid	Т8
Umbilicus	T10
Symphysis	T12
Anterior thigh	L2
Anterior knee	L3
Antero-lateral ankle	L4
Dorsum of great and 2 nd toe	L5
Lateral side of foot	S1
Posterior calf	S2
Perianal sensation	S2-5

After the motor and sensory examination, presence of sacral sparing may be noted by voluntary Rectal Sphincter tone and toe flexor contractions. **Presence** of Sacral sparing indicates a better neurological prognosis.

Although spinal shock is over by 24 hours, rarely it may be prolonged. A positive Bulbocavernous reflex or a positive anal wink indicates the end of spinal shock. If no motor or sensory function can be documented at this stage, a complete spinal cord injury is present.

2.1.3 Investigations-

All patients with suspected spinal Injury should have radiographic evaluation.

a) Initial screening can be done by conventional Antero-posterior and Lateral X-rays. The Cervical spine radiographs must include the C7-T1 junction to be considered adequate

- Additional Open-mouth views should be done to evaluate odontoid Injury.

- Whole spine should be evaluated with a patient of spinal Injury.

b) The patient should be referred for advanced diagnostic modalities only when the patient is stable:

i. CT

ii. MRI

2.1.4 Out Patient care-

A secondary hospital is expected to provide outpatient care to the Spinal cord injury patients who may be referred back from specialized centers after definitive treatment. This may be in form of a

- i. Physiotherapy for passive mobilization of all joints and active exercises for muscles.
- ii. Teaching of clean intermittent catheterization
- iii. Counselling of the patient and attendants
- iv. Care of bed sores

2.1.5 Day Care-

Day care might be needed for situations like Debridement of bed sores.

2.1.6 Referral criteria-

- i. The patient should be Hemodynamically stable and fully resuscitated at the time of referral.
- ii. All the patients who need surgery (indications discussed in the next section) need to be referred to a specialized tertiary care centre.
- iii. The decision of need for surgery can only be made by an experienced Spinal surgeon either Orthopedic or Neurosurgeon. In absence of these all patients with proven or suspected spine injury should be referred to a higher center.

2.2 At District Hospital

2.2.1 Clinical Diagnosis-

iv.

As described above (in situation 1)

2.2.2 Radiographic evaluation of patients with spinal Injury-

- i. Initial screening can be done by conventional Antero-posterior and Lateral X-rays.
- ii. Additional open-mouth views should be done to evaluate Odontoid Injury.
- iii. Special views like Swimmer's view and Oblique views can be done to see junctional areas.
- iv. CT scan of the whole spine should be done if in presence of clinical suspicion but fractures cannot be demonstrated on X-rays or if junctional areas are not visualized.
- v. MRI should be done to evaluate Ligamentous injury, Spinal cord injury.
- vi. In patients with pre-injury morbidities such as Ankylosing.
- vii. For Stiff spine, CT and MRI should be done to rule out occult instability even if X-rays are normal.
- viii. Whole spine should be evaluated with a patient of Spinal injury





Fig. 14.2: X-RAY VIEW OF CERVICAL SPINE TRAUMA Fig. 14.3: MRI OF SPINE TRAUMA

2.2.3Treatment: Standard Operating procedure (a) On arrival in Emergency room:

• Once the patient with a potential Spinal injury reaches the emergency, the patient should be transferred off the backboard onto a firm padded surface while maintaining spinal alignment. A baseline skin assessment can be performed at the time of shifting the patient from spine board to hospital bed. Adequate number of personnel should be employed for Logrolling during patient Repositioning, Turning and Transfers.



Fig. 14.4: LOG ROLLING WHILE EXAMINING & SHIFTING OF SPINAL INJURY PATIENT

No clinical evidence exists to definitively recommend the use of any Neuro protective pharmacologic agent, including Steroids, in the treatment of Acute Spinal Cord injury to improve functional recovery. However high dose Methyl-Prednisolone may be used as per NASCIS III recommendations (Methylprednisolone: Bolus dose of 30 mg/kg of body weight over 15 minutes, followed by a 45-minute pause, and then a 23-hour continuous infusion of 5.4 mg/kg/hr., (If patient presents between 3 and 8 hrs., give the above steroid infusion for total of 48 hrs.) if the patient presents within 8 hours of injury. The risk of complications as Infection, Sepsis, Respiratory complications and Gastrointestinal Hemorrhage should be kept in mind while administering steroids. It is basically a treatment option, not standard care.

Once initial Resuscitation is done, complete a comprehensive tertiary trauma survey in the patient with potential or confirmed Spinal Cord injury.

In the patient with acute Spinal Cord injury, particularly higher Cervical injury, assess frequently and early document any evidence of Traumatic Brain injury (TBI) in the form of Loss of consciousness and post traumatic Amnesia.

Screen for Thoracic and Intra-abdominal injury in all patients with spinal cord injury.

Perform early stabilization of extra spinal fractures. Perform this surgery as early as possible to facilitate early Rehabilitation and Concomitantly with any required Spinal Stabilization if the patient is medically stable.

(b) Out Patient

Outpatient care is needed for non-surgically treated patients on Ambulatory care and Surgically treated patients. This will include:

Prescription of appropriate Orthoses physiotherapy services, Counselling: Social, Physiotherapy, Vocational

(c) Day Care

Referral criteria:

Surgically treated patients may be referred back to secondary hospitals for Physiotherapy and care of Back, Bladder and Bowel.

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15. MANGLED EXTREMITIES (AMPUTATION)

1. Introduction

- Traffic is increasing day by day and so are road traffic accidents.
- Most of RTA involve trauma to extremities to varying level.

2. At PHC/Sub Center level

- Follow ATLS guidelines of A B C D E
- After stabilization of patient, examine for other associated injuries such as Pelvis and Spine which are common in poly trauma
- If such injuries are present, then refer the case to higher center without any delay.
- If absent, then grade the injury according to MESS (Mangled Extremity Severity Score) which is useful for decision making regarding definitive surgical treatment (Salvage or Amputation of the limb) and refer the patient to the higher centre.

3. Before Referring

- Wash the wound primarily with Normal Saline, Iodine solution and Hydrogen Peroxide till all visible contamination in removed.
- Stop any active bleed with help of Compression bandage or Tourniquet
- Avoid excessive handling of injured limb to avoid neurovascular complications
- Infuse IV fluids and Blood
- Inject first dose of higher Antibiotics
- Give Inj. TT. O.5 ml intra muscular

Things to be done Before Referral

- Inform the concern center about MESS score and expected time of arrival of patient
- Arrange for proper transport
- Make sure that patient will be Hemodynamically stable **during entire duration of transport.**

4. Standard treatment protocol for management of Mangled extremities at RH/SDH/DH

- No Delay in receiving the patient and starting treatment.
- Multi-systemic approach
- Patient should be examined according to ATLS protocol of ABCDE.
- Look for signs of poly trauma, involvement of Head injury, Spine or Abdominal and Pelvic injuries.

4.1 Informed consent:

- Two surgeons should certify that the limb needs amputation.
- Consent of the patient (if conscious and cooperative) should be taken.
- Consent of relative should be taken if patient is non responsive.
- Detailed informed consent in their local language should be taken.

4.2 Pre-operative care:

- Pain management
- Clinical assessment
- Decision making
- Discharge planning
- Record keeping

4.3 Peri operative care:

- The scheduling of operations
- Operation undertaken
- Antibiotic prophylaxis
- Thrombo prophylaxis

4.4 Post-operative care:

- Pain management
- Wound care
- Rehabilitation

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16. COMMON FRACTURES IN CHILDREN

1. Common fractures

Mode - Fall on out- Stretched hand

- Lower end Radius treated by closed reduction
- Greenstick fracture both bone Forearm requires Closed reduction under General anesthesia
- Supracondylar fracture Humerus Depending on classification either Closed reduction or closed reduction with cross k-wire or Open reduction
- Fracture Lateral Condyle Humerus requires Open reduction
- Fracture Clavicle treated by Figure of 8 bandage
- Fractures of lower limb

- Epiphyseal injury around Hip and Knee Closed reduction and Immobilization in splints
- Surgical treatment Open reduction and internal fixation with multiple cancellous screws or mini DHS plate
- Fractures around hip Surgical treatment
 Shaft femur
 - Undisplaced Conservative Displaced
 - Shaft tibia Undisplaced – Conservative Displaced – Surgical in the form of Closed or Open reduction and internal fixation with either elastic nails or plate and screws.

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17. BONE TUMORS

Primary Neoplasms of the skeleton are rare, amounting to only 0.2% of the overall human tumour burden. However, children are frequently affected and the etiology is largely unknown. Significant progress has been made in the histological and genetic typing of bone tumours. Furthermore, advances in combined surgical and chemotherapy have led to a significant increase in survival rates even for highly malignant neoplasms, including Osteosarcoma and Ewing sarcoma.

1. Primary bone tumors

1.1 Benign Tumors:

1.1.1 Osteoid Osteoma-

True benign tumor of bone

- **Age group**: 5 25yrs
- **Commonest site**: Diaphysis of long bone. e.g. tibia.
- Clinical features: Night pain, relieved by Salicylates.
- **Pathology:** Consists of a Nidus surrounded by dense Sclerotic bone
- X-ray Zone of sclerosis surrounding a nidus
- **Treatment**: Complete excision of Nidus with Sclerotic bone.
- Prognosis Good

1.1.2 Osteochondroma-

Commonest Benign tumor of bone, arises from adjoining Epiphysis to Metaphysis

- Age group: Adolescents
- **Clinical features**: Painless swelling around a Joint, Sessile or Pedunculated
- Multiple site involvement is called Diaphyseal Aclasia

- **Complications:** Bursitis, Neuropathy, limitations of movement, Malignant transformation (chondrosarcoma) occur rarely
- **X-ray:** Bony growth made up of mature cortical bone and marrow. Cartilaginous gap not visible.
- Treatment: Excision including Periosteum

1.1.3 Fibrous Dysplasia-

Normal bone is replaced by fibrous tissue. Erodes the cortices of bone from within.

- Thin layer of sub periosteal bone forms around the mass, so bone appears expanded. Site- upper end of Femur, Tibia, Ribs.
- Mono-ostotic one bone is affected. Poly-ostotic many bones are affected.
- **Clinical features**: Pain, Deformity, Pathologic fractures.
- **X-ray**: Ground glass appearance.
- Treatment: Curettage and bone Grafting.

1.1.4 Osteoclastoma (Giant Cell Tumor)-

Common bone tumor with variable growth potential

- Age group: 20 40 years
- Site: starts in Epiphysis and extends into Metaphysis
- Commonly lower end of Femur, upper end of Tibia
- Pathology
- Cell of origin is uncertain
- Tumor consists of undifferentiated spindle
- Cells with multinucleated giant cells
- Clinical feature: Swelling, Vague pain
- X-ray
 - Lytic expansile lesion
 - Eccentric location
 - Soap bubble appearance Pathognomonic.



Fig. 16.1: SOAP BUBBLE APPEARANCE OF GIANT CELL TUMOUR

- Excision with reconstruction
- Curettage with or without supplementary procedures like chemical Ablation, Cryotherapy, PMMA implantation
- Amputation: aggressive tumors, recurrence
- Radiation: tumor involving vertebrae

1.2 Primary malignant bone tumors:

1.2.1 Osteogenic Sarcoma-

- Usually in males aged 10 to 20
- Occurs most often in Femur, but also in Tibia and Humerus
- Occasionally in Fibula, Ileum, Vertebra or Mandible
- Tumor arises from bone-forming Osteoblast and bone-digesting Osteoclast
- Treatment
- a) Surgery
- b) Radical Tumor resection
- c) Mega Voltage Radiotherapy and / or Chemotherapy or combination of both.

1.2.2 Parosteal Osteogenic Sarcoma-

• Usually in females ages 30 to 40

- Occurs most often in distal Femur, may also be in the Humerus, Tibia, and Ulna
- Develops on surface of bone and progresses slowly

• Treatment

- a) Surgery
- b) Tumor Resection, possible Amputation
- c) Inter-Scapulothoracic surgery
- d) Hemipelvectomy
- e) Chemotherapy

1.2.3 Chondrosarcoma-

- Usually in males ages 30 to 50 years
- Occurs most often in Pelvis, proximal Femur, ribs and Shoulder girdle
- Develops from Cartilage and grows slowly
- Usually Painless, locally recurrent and invasive
- Hemipelvectomy
- Surgical resection (ribs)
- Radiation and / or Chemotherapy

1.2.4 Multiple Myeloma-

It is the most common primary malignancy of bone having short duration onset and aggressive in nature.
Age of occurrence – commonly around 60yrs. 2% less than forty.

Clinical features:

- Bone pain
- Weakness
- Weight loss
- Anemia
- Thrombocytopenia
- Peripheral neuropathy
- Hypocalcaemia
- Renal failure

It manifests in pathological fractures commonly in spine followed by Ribs and Pelvis.

Laboratory studies:

- Urine Bence Jones Protein present in 30% of cases
- Blood Very high ESR, A/G ratio reversal
- Serum electrophoresis Abnormal spike in Gamma Globulin region in 90% cases

Treatment:

- a) Chemotherapy Melphalan is the drug of choice
- b) Given in combination with Vincristine, Prednisolone and sometimes Cyclophosphamide. Cycles are repeated 3 - 4 weeks for 6 - 12 cycles
- c) Splintage of diseased part
- d) Radiotherapy- Useful in case of Neurological compression
- e) Surgical intervention in advanced tumors

2. Metastatic bone tumors:

- The most common bony malignancies are Metastatic Carcinomas
- Metastatic lesions represent the most common cause of pathology fractures due to a Neoplasm
- Usually are multiple but can be solitary
- The most common primaries are Breast, Prostate, Lung, Kidney and Thyroid, in that order
- Renal metastasis is quite vascular and have cold bone scans

Clinical features:

- Patients with known primary malignancy presents with symptoms suggestive of Secondaries in bone
- Bony pain, commonest site is spine
- Pathological fractures most common in spine

Investigation:

- X-ray Majority are Osteolytic, few are Osteoblastic. e.g., Male- Prostatic Secondaries, Female- Breast Secondaries
- Blood High ESR, elevated serum Calcium, elevated serum Acid Phosphatase in Prostatic Secondaries

Treatment:

- a) Symptomatic relief of Pain and prevention of pathologic fractures
- b) Chemotherapy
- c) Radiotherapy

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10. Anesthesiology

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1. INTRODUCTION

1. Definition

noxious stimuli with different inhalational and intravenous drugs.

Anesthesia is the reversible state of unconsciousness with amnesia, analgesia and unresponsiveness to

2. Types of Anesthesia



Fig. 1.1: ANAESTHESIA GENERAL PROTOCOL

3. Pre-Anaesthetic Checklist

- a) Check function of high vacuum suction machine.
- b) Check reserve oxygen supply.
- c) Check function of breathing system.
- d) Check vaporizer.
- e) Check absorber if in use.
- f) Inspect equipment for
 - Endotracheal intubation
 - Intravenous infusion
 - Resuscitation

- g) Check gas supplies, both cylinder, pipeline & circuit.
- h) Check oxygen supply failure alarm & oxygen flush.
- i) Check oxygen nitrous oxide ratio.
- j) Mechanical ventilator.
- k) Waste gas scavenging system.
- l) Check reserved gas supply.
- m) Apply and check monitoring system & cautery, circuit and tourniquet.
- n) Set appropriate alarm level.

4. Recommended minimum essentials in Operation theatre

4.1 Minimum Essentials Required:

- a) Continuous supply of oxygen via central pipeline or jumbo cylinders.
- b) Anesthesia machine with two oxygen and one nitrous oxide input connections.
- c) One small oxygen cylinder mounted on Boyles other than jumbo or central line and nitrous oxide cylinder along with cylinder valve opener should be there.
- d) Boyle's machine should have Hypoxic and alarm safety system.
- e) Working suction machine (Foot driven and Electric) with all connectors, tunings, and suction around the tip and exhaust fans.
- f) Multi-parameter (Pulse Oximeter, NIBP, ECG, ETCo2).
- g) Defibrillator (Adult & Paediatric).
- h) OT table with tilting facility.
- i) Adult (Bain) & Pediatric (JR) Circuit.
- j) Set of oral and nasal Airways sizes 00, 1, 2, 3, 4.
- k) Silicon bag with all sizes of masks & cuffed and plain Endotracheal Tubes. Working laryngoscope with 5 sizes of blades.

- Magill's forceps (Adult & Pediatric), Scissors, Ampoule cutter, Torch, Thermometer.
- m) IV sets, Micro Sets, Blood sets, three-way IV extension tube & central line.
- n) IV fluids crystalloids RL, DNS, D5 and Colloids plasma expanders.
- o) For Difficult Airways fiber optic boogie, LMA (Classic & Pro Seal), I-gel, Tracheostomy, tube, percutaneous tracheostomy set, Combitube and all size Stylet (flexible & Rigid).
- p) Disposable spinal needles 23, 24, 25, 26, 27, Epidural set 16, 18.
- q) Radiant warmer/blanket, heating mattress, fluid warmer.
- r) Postoperative recovery room with oxygen supply and monitors.
- s) BP apparatus & Stethoscope.
- t) Vaporizers-(Goldman) Halothane & Isoflurane
- u) Capnography To monitor End Tidal Carbon dioxide (ETCO₂) Where laparoscopic surgeries are done.

New Born resuscitation kit

- Weighing scale.
- Paediatric resuscitation kit with all masks & LMA & Stylet.
- Flexible smooth rubber cord for tourniquet to secure IV line.
- Central line (cava fix, double & triple Lumen).
- Glucometer, peak flow meter, height scale & refrigerator (for blood sample).

4.2 Emergency Medicine Kit:

Injections Atropine Glycopyrrolate Adrenaline Noradrenaline Dopamine Dobutamine Dobutamine Mephentermine Ephedrine hydrochloride Phenylephrine Vasopressin Prostaglandin Isoprenaline Digoxin Esmolol Propranolol 	 Metoprolol Hydrocortisone Dexamethasone Potassium chloride Calcium gluconate Soda bicarbonate Magnesium 25% & 50% Mannitol Tranexamic acid Dextrose 25% & 50% Insulin Deriphyllin Aminophylline Amiodarone. Xylocard 2% Furosemide (Lasix) 	 Nebulizer Hemolock Botroclot Naloxone ECG Lead Tablets Nifedipine Captopril Sorbitrate Atenolol
--	---	--

Chart No. 1.1: EMERGENCY MEDICINE KIT

4.3 Doses and Indication for Emergency Drugs:

Chart No. 1.2: EMERGENCY DRUGS & DOSES

Drug	Indication	Dose
Sodium bicarbonate	Acidosis	2-4 m eq/kg
Atropine	Bradycardia	0.03 mg/kg
Naloxone	Narcotic depression	5-10 mcg/kg
Calcium gluconate	Low perfusion	0.6 ml/kg
Epinephrine	Asystole	5 mcg/kg of 1:1000 solutions.

4.4 Various Types of Anesthesia Drugs:

Chart No. 1.3: ANAESTHESIA DRUGS

A. Premedication	D. Inhalational Anaesthetic Agent
 Anticholinergic a) Inj. Glycopyrrolate b) Inj. Atropine Antacid a) Inj. Ranitidine b) Inj. Pantoprazole 	a) Halothaneb) Isofluranec) Sevofluraned) Desflurane
3. Antiemetica) Inj. Metoclopramideb) Inj. Ondansetron	E. Reversal Agent a) Inj. Neostigmine
c) Inj. Granisetron4. Opioidsa) Inj. Pentazocin (Fortwin)	F. Obstetric Drugs a) Inj. Oxytocin b) Ini. Methyl ergometrine

b) Inj. Butorphanolc) Inj. Fentanyl	c) Inj. Prostaglandin F2 alphad) Inj. Magnesium Sulphate
 Benzodiazepines a) Inj. Diazepam 	F. Spinal Anaesthesia drugs
b) Inj. Midazolam	a) Inj. Lignocaine (heavy) 5 %b) Inj. Bupivacaine (heavy) 0.5%
	G. Local Anesthetics
	a) Inj. Lignocaine 2%
	b) Inj. Lignocaine 2% with Adrenaline
B. Induction Agents	c) Inj. Bupivacaine 0.5%
a) Inj. Thiopentone Sodium	d) Inj. Bupivacaine 0.25% preservative free
b) Inj. Propofol	e) Inj. Rocuronium 0.75%
c) Inj. Ketamine	H. Other Drugs
	a) Inj. Tramadol
C. Muscle Relaxants	b) Inj. Diclofenac
Depolarizing Muscles Relaxants.	c) Diclofenac Suppository
a) Ini Suxamethonium	a) Inj. Buprenorphine
Non Depolarizing Muscles Polevents	f) Inj. Clonidine
Non- Depolarizing Muscles Relaxants	g) Inj. Bupivacaine 0.5% heavy
a) Inj. Pancuronium	h) Lignocaine Jelly 2%
b) Inj. Vecuronium	
c) Inj. Rocuronium	
d) Inj. Atracurium	

4.5 Doses of Intravenous Anaesthesia Agents: Chart No. 1.4: INTRAVENOUS AGENTS IN ANAESTHESIA

DRUG	DOSE	REPEAT DOSE/ INFUSION RATE
Thiopentone	5-6 mg/kg	2 mg/kg
Propofol	1-3 mg/kg	0.2-1 mg/kg
Ketamine	1.5-2 mg/kg	40 mcg/kg/min
Fentanyl	0.025-2 mcg/kg	4-10 mcg/kg/hr (Infusion)
Alfentanil	5-10 mcg/kg	05-10 mcg/kg/min (I)
Sufentanil	0.1 mcg/kg	0.01 mcg/kg/min) (I)
Succinyl Choline	2 mg/kg	4-10 mg/min (I)
Pancuronium	0.08 mg/kg	0.02 mg/kg
Atracurium	0.5 mg/kg	4-12 mcg/kg/min (I)
Vecuronium	0.08-0.1 mg/kg	0.12-2 mcg/kg/min (I)
Rocuronium	0.6 mg/kg	9-12 mcg/kg/min (I)
Diazepam	0.1-0.3 mg/kg	

Midazolam	0.5-0.1mg/kg	
Flumazenil	0.1-0.5mg	
Neostigmine	0.05 mg/kg	
Adenosine	0.1 mg/kg	
Digoxin	15-20 mcg/kg	
Diltiazem	0.15-0.35 mg/kg	
Benadryl	1 mg/kg	
Furosemide	1-2 mg/kg	
Mannitol	0.25-1 mg/kg	
Esmolol		100-300 mcg/kg/min (I)
Dopamine		5-15 mcg/kg/min (I)
Dobutamine		7-15 mcg/kg/min (I)
NTG		1-1.5 mcg/kg/min (I)
		15 mg/kg (loading) (I)
Amiodarone		5 mg/kg/hr. (maintenance)
KCL		0.5 mEq/kg/hr. (I)
Sodium Nitroprusside		40-100 mcg/min (I)
Atropine	0.01 mg/kg	
Glycopyrrolate	4 mcg/kg	
Dexamethasone	0.1 mg/kg	
Paracetamol	15 mg/kg	
Diclofenac sodium	1.5 mg/kg	
Neostigmine	0.05 mg/kg	
Noradrenaline Bitartrate	4 mcg per mL	In titrated dose
Labetalol Hydrochloride	20 mg IV push over two minute 40-80 over 2 - 10 minute	In titrated dose

5. Adrenaline Doses

Adult:	Paediatric:	Neonatal:
1 mg	0.01 mg/kg (0.1 ml/kg) IV or	0.01 mg/kg to 0.03 mg/kg (0.1-0.3
Repeat Q 3-5 min	intraosseous	ml/kg)
Higher IV doses NOT recommended	Maximum dose 1 mg IV/IO or 10	IV, intraosseous, or umbilical
ET doso 2 to 2.5 mg	mg ET repeat Q 3-5 min	Repeat Q 3-5 min
	No Higher IV doses. ET dose 0.1	Higher IV doses NOT recommended
Drip 1-10 mcg/min	mg/kg (ten times IV dose)	
	Strong preference for IV/IO!	L1 does 0.1 mg/kg (ten times 1V dose)
	Drip 0.05-1 mcg/kg/min	,

Chart No. 1.5: ADRENALINE DOSES BY AGE

6. Important Aspects of Anaesthesia

Chart No. 1.6: TEN GOLDEN RULES OF ANAESTHESIA

1	Do an adequate preoperative assessment
2	Nil by mouth for six hours
3	Put patient on a tipping table
4	Check your machine and cylinders before you start
5	Keep a suction instantly ready
6	Keep patient's airway clear
7	Be ready to control patient's ventilation
8	Have a vein open
9	Check patient's pulse and blood pressure
10	Always have someone who can apply cricoid pressure in emergency

Chart No. 1.7: BREATHING CIRCUITS

Jackson Rees circuit	Use for neonate & children less than 25kg
Bain Circuit	Most commonly used circuit weight more than 25 kg

7. Airway

Chart No. 1.8: ORAL AIRWAYS

Formula for size of ETT		
Neonate	2.5 or 3	
Children < 6 years	age/3 + 3.5	

Children > 6 years	age/4 + 4.5
Adult female	ET Tube number 7 or 7.5
Adult male	ET Tube number 8.5 or 9

8. Grading of Patients & Fluid Requirements

8.1 Grading of Patient:

Chart No. 1.9: GRADING

ASA 1:	ASA 2:	ASA 3:
A Normal Healthy Patient with only surgical Condition	A patient with mild systemic Disease (like, HTN, DM etc. under control with treatment)	A patient with severe systemic disease that limits activity but is not incapacitating (Disease is not under control with treatment but not life threatening)

ASA – American Society of Anesthesiology

8.2 Fluid Requirement for Children:

Chart No. 1.10: FLUID REQUIREMENT FOR CHILDREN

Weight (kg.)	Hourly Fluid Requirement
<10	4 ml/kg
11-20	40ml + 2ml/kg > 10
>20	60ml + 1ml/kg > 20

9. Monitoring Standards

Chart No. 1.11: MONITORING STANDARDS

ESSENTIALS:	STRONGLY RECOMMENDED:	
 Continuous presence of qualified Anesthesiologist Oxygen supply failure alarm Observation of reservoir bag & chest expansion Ventilator disconnect alarm Oxygen analyzer of inspired gases Pulse oximetry Electrocardiogram Non-invasive arterial blood pressure 	 Temperature End – tidal Carbon dioxide Monitoring of neuromuscular blockade 	

10. Fasting Guidelines

Ingested	Minimum Fasting
material	Period (h)
Clear Liquids	2
Breast Milk	4
Infant formula	6
Non-human Milk	6
Light Meal	6

Chart No. 1.12: FASTING GUIDELINES

a) These recommendations apply to healthy patients of all age group who are undergoing elective procedure.

b) Clear liquids: water, fruits without pulp, carbonated, beverages, tea & black coffee.

11. Operation Theatre Cleaning Protocol

- Do not use Broom inside Operation Theatre
- Do Fogging after infected case and enter in register
- In between the two cases, clean O.T.
 - 1) Remove linen, instrument sets and used material.
 - 2) Clean surface, wipe with Bacillocid solution.
 - Mop the floor (ideally with Lysol 3% or Bacillocid)

12. Operation Theatre Fumigation Protocol (Power Jet and Auto Mist)

12.1 Fumigation agents

For O.T. fumigation 20% aqueous solution of Hydrogen Peroxide 11 % and Silver Nitrate solution 0.01% should be used. For 1000 cu. ft. Take 400 ml clean water and add 80 ml. of fumigant solution – Hydrogen Peroxide 11 % + Silver Nitrate 0.01 % solution in a fogging machine. Keep the room / OT closed for 60 min. Mop the flooring with clean floor soaked in the same solution.

12.2 Advantages:

Solution has broad spectrum of Antimicrobial activity, effective against HIV, HBV and other pathogenic bacteria, viruses, molds and spores. It is a non-toxic, eco-friendly. It liberates nascent oxygen having strong oxidizing effect.

12.3 Procedure:

- a) For 350 sq. ft. OT take 1000 ml (11t) water in tank and add solution in that.
- b) Keep fumigation machine on floor.
- c) Keep machine near to door so after fumigation you can easily switch off machine.
- d) Daily mopping of OT is compulsory with solution.
- e) Please cover all electric equipments, digital and water resistance equipment (C arm monitor microscope etc.) with cloth or plastic sheet.
- f) Empty the machine tank and use balance solution.
- g) Do not run (power jet) machine continuously more than 35 minutes and total one hour in a day; keep 4 to 6 hours of interval in between two running.
- h) Do not operate machine in presence of patient and do fumigation in vacant room only.

13. Biomedical Waste Management

Yellow Bag	Red Bag	White (Translucent)	Blue Bag	Black Bag
Human Anatomical Waste	Contaminated Waste	Waste sharps	Glass	General waste
Human tissues, Organs & body	Waste generates from	menuumg metals	Broken or	
parts	disposable items such	Needles, Syringes	discarded and	
Soiled waste	as tubing, bottles, IV tubes & sets, catheters,	with fixed needles, Scalpels, blades or	contaminated glass	
Items contaminated with blood,	urine bags, syringes	any other	Metallic Body	
dressings plaster casts cotton	(without needle).	object that may cause	Implants	
swabs & bags containing		puncture & cuts		
residual or discarded blood &		-		
blood components				
Microbiology, Biotechnology & other clinical laboratory waste				

Chart No. 1.13: SEGREGATE BIOMEDICAL WASTE

14. Universal Precaution for Health Care Workers in Operation Theatre

- a) Proper hand washing before and after contact with each patient and handling patient's specimens. Do not leave sharps on bed or beside of patient.
- b) Use of PPE i.e. gloves, mask, gowns and protective eye-wear.
- c) Prevent needles stick injuries with other sharp instrument.
- d) DO NOT BEND OR RECAP NEEDLES.
- e) In case of needles stick injuries or cuts, wash the area properly with water and soap and Contact for post exposure prophylaxis treatment.

- f) Dispose all sharps in puncture proof container.
- g) Avoid spills of blood & body fluids, If it occurs, cover it with absorbent material like gauze over which 1 % solution of sodium hypochlorite should be poured and left for 10-15 minutes (ideal 30min). Clean the area with a bleach mop. Wash and dry the mop.
- h) Proper segregation and disposal of Bio-Medical waste.
- i) Immunization (for Hepatitis B).

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Further reading



2. DIFFICULT AIRWAY ALGORITHM

Assess the likely hood and clinical impact of basic management problems: In case of difficult Intubation and difficult Ventilation awake intubation should be carried out. In case you cannot ventilate, cannot incubate-(CVCI) Emergency surgical airway (i.e. Cricothyrotomy) may be necessary if the patient is hypoxic.



Fig. 2.1: INTRODUCING LARYNGEAL MASK AIRWAY



Fig. 2.2: LARYNGEAL VIEW OF AIRTRAQ VIDEO LARYNGOSCOPE

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3. PRE-ANAESTHESIA CHECK UP PROTOCOL

1. Pre Anaesthesia Checkup (PAC)

Pre anesthetic checkup should be carried on prior day of surgery to avoid unnecessary postponement of patients.

1.1 Following history should be asked:

- a) High blood pressure, Diabetes, Asthma, Heart Disease, Stroke, Cough and Cold in the past 2 weeks.
- b) History of Shortness of breath, Difficulty in breathing on lying flat, Difficulty in breathing on climbing stairs, Chest pain, Palpitation, Chronic cough.
- c) History of Cardiac illness such as heart failure, heart valve problem, high cholesterol level, RHD, CHD or Cardiac arrest.
- d) History of Respiratory problems such as Chronic Bronchitis, Allergic rhinitis, Snoring, Tuberculosis, Lung cancer, Chest infection or Pneumonia.
- e) History of Neurological problems such as Epilepsy, Fainting, Coma, Paralysis, Chronic Headache, Dizziness, Parkinsonism, Brain Tumor.
- f) History of Muscle or Bone problems such as Osteoporosis, Neck pain or Neck stiffness, Difficulty to open mouth, Spine problem, congenital muscle disease.
- g) History of Kidney problem such as dialysis. Gastric problem such as Heartburn, Reflux, Jaundice.
- h) History of Blood or Endocrine problems such as Anemia, receipt of blood transfusion, prolonged bleeding, Thalassemia, Sickle cell anemia, Thyroid disease.
- i) History of Allergy.
- j) History of Medications such as Diabetes, Hypertension, Asthma, Psychotropic drugs, Steroids.
- k) History of Smoking, Alcohol, Addictive drugs, BOH.

- History of Dentures, braces, crown, loose teeth, implanted Pacemaker, implanted cardiac Defibrillator, Prosthetic Heart valve, Orthopedic implant, Organ recipient.
- m) History of STD, Drug resistance.
- n) History of Anesthesia with due Allergy history of Headache after anesthesia, Awareness during anesthesia, transfusion reaction, admission to ICU after surgery, any complication after anesthesia.

2. Following history should be asked

- a) Investigations as advised by Surgeon and Anesthetist.
- b) ASA: (American Society of Anesthesiologist) Assessment of patient in terms of I/II/III/IV/V/VI/E with additional risk factor.
- c) Do proper systemic examination of Cardiovascular system, Respiratory system, CNS.
- d) Evaluate the patient's airway Thyromental distance (If < 3finger anticipated difficult intubations), dentition, mouth opening.

3. Consent

Informed consent of patient should be taken before surgery which means consent given to a proposed specific intervention, without any force, undue influence, fraud, threat, mistake or misrepresentation, and obtained after disclosing to the person giving consent adequate information including risks and benefits of, and alternatives to, the proposed intervention in a language and manner understood by such person with no binding to consent after being informed. Consent should be attested by staff nurse of ward. There should be common consent for surgery and anesthesia unless it is essential to take consent for anesthesia separately.

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Further reading



4. CONDUCT OF ANAESTHESIA

1. Anesthesia can be conducted taking 5'P's into consideration

They are: -

- a) Pre-operative Assessment of Patients.
- b) Patients Preparation.
- c) Preparation of Operation Theatre.
- d) Premedication.
- e) Plan of Anesthesia.

2. Anesthesia to different groups of patients

2.1 Hypertensive Patients:

- a) Advise to take all anti-hypertensive drugs early in the morning of surgery.
- b) Avoid ACE inhibitors on the day of surgery to avoid intra operative fall in BP.
- c) Keep anti-hypertensive drugs like Nitroglycerine, Nifedipine and Metoprolol ready.
- d) Keep volume expanders like colloids ready.

2.2 Diabetic Patient:

- a) Advice to skip the morning doses of Insulin and Oral hypoglycemic.
- b) Check fasting blood sugar, urine ketones and serum electrolytes on the morning of surgery.
- c) Place the Diabetic patients first in the list of surgery.
- d) Specific care of the patients with autonomic disturbances.

e) Keep Glucometer and short acting regular insulin ready for intra-operative use.

f) Assess target organ function and monitor accordingly.

2.3 Obstetric patients:

- a) Prepare operation theatre ready for emergency LSCS.
- b) Follow Anti-Eclamptic regimens for patients with pregnancy induced hypertension.
- c) Verify the availability of blood.
- d) Keep the difficult intubation kit ready.
- e) Take care of prophylactic measures to treat aspiration (Mendelson Syndrome).
- f) Keep the Infant resuscitation bag, smaller endotracheal tubes, laryngoscope, suction apparatus, infant radiant warmer, oxygen hoods & emergency drugs for resuscitation.

2.4 Trauma Patients:

- a) Stabilize the patients taking into consideration ABC (Air, Breathing & Circulation) of resuscitation.
- b) Stabilize the cervical spine (with collar for suspected C-spine injuries).
- c) Place the intercostal drainage for chest injuries.
- d) Splint to the fractured site.
- e) Assess the GCS (Glasgow Coma Score).If<8, intubate for airway protection.
- f) Transport the patient to referral center with portable ventilator, oxygen cylinder, ambu bag on standby.

Inform the patient's details to referral center prior to or during transport.

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Further reading



5. COMPLICATIONS OF ANAESTHESIA



Fig. 5.1: COMPLICATIONS OF ANAESTHESIA

1. Management of common complications during General Anaesthesia

1.1 Aspiration:

- a) Follow appropriate fasting guidelines.
- b) Administer anti-aspiration prophylaxis like H₂ blocker, Proton pump inhibitors, Metoclopramide. Ondansetron, Granisetron.
- c) Intubate by rapid sequence technique in full stomach patients.

1.2 Hypercarbia:

a) Ventilate appropriately as Hypoventilation is the commonest cause.

1.3 Hypertension:

- a) Maintain good plane of anaesthesia.
- b) Use opioids for adequate pain relief.

1.4 Hypotension:

- a) Manage blood loss appropriately.
- b) Preload the patient adequately before spinal anaesthesia.

1.5 Post-Operative Apnoea:

- a) Commonly due to over dosage or sensitivity of Barbiturates, Opioids and inhalational agent and inadequate reversal.
- b) Titrate the doses of opioids. Use appropriate dose to avoid respiratory depression. Give adequate sedation to obese and geriatric patient.

 c) Keep Flumazenil (antidote for Benzodiazepine) & Naloxone (antidote for Opioids) ready to manage complications of over dosage.

1.6 Nerve Palsies:

- a) Position the patient appropriately to prevent pressure on the nerves.
- b) In prone position avoid pressure on eyeball and avoid over – abduction of arms to prevent Brachial Plexus injury.
- c) In lateral decubitus position, keep axillary roll beneath axilla to prevent Brachial Plexus injury.
- d) In lithotomy position, care must be taken to avoid injury to the lateral popliteal nerve.

1.7 Hypothermia and Shivering:

- a) Increase theatre temperature (21-degree C for adults & 28-degree C for children).
- b) Use warm intravenous fluids.
- c) Use warm blankets and forced air warming to combat hypothermia.
- d) Use IV Dexamethasone or IV Pentazocin or IV Tramadol to treat shivering.

1.8 Pulmonary Edema:



Fig. 5.2: PULMONARY EDEMA

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Further reading



6. REGIONAL ANAESTHESIA

1. Indication

For the production of local anesthesia.

2. Dosage and Administration

The dose is adjusted according to the response of the patient and site of administration.

3. Contraindications

Hypersensitivity is known to anesthetics of the Amide type. Solution containing Adrenaline is contraindicated for anesthesia of Fingers, Toes, tip of Nose, Ears and Penis. Bupivacaine is contraindicated for IV regional anaesthesia.

4. Precautions

Shock, heart block, known drug sensitivity, Liver disease, Kidney disease, Epilepsy, impaired Respiratory function.

5. Adverse Reaction

Over dosage may cause CNS reaction. Numbness light-headedness of tongue, Dizziness. and Blurred vision. Tremors. followed Drowsiness, Convulsions, by Unconsciousness and possibly respiratory arrest. Cardiovascular reactions of over dosage include Hypotension and Myocardial depression.

6. Treatment of over dosage

Treatment of patient with toxic manifestations consist of arresting convulsions assuring adequate ventilation with oxygen, if necessary by assisted or controlled ventilation (respiration) if convulsions occur they must be treated rapidly by intravenous injection of Thiopentone 100 to 200mg. Alternatively, Diazepam 5 to 10mg may be used and Intralipid (1.5 mg/kg body weight.).

Once convulsions have been controlled and adequate ventilation of the lungs ensured, no other treatment is generally required. If Hypotension is present. However, a vasopressor, preferably one with inotropic activity, e.g. Ephedrine 15 to 30 mg should be given intravenously in diluted from titrated dose.

Anxiety or slight twitching, a small intravenous dose of Inj. Diazepam may be given carefully and slowly; Oxygen should also be given. In severe circulatory depression barbiturate such as Thiopentone is contra-indicated when convulsion has developed. Cardiovascular collapse is treated with vasopressors & plasma infusion, Inj. Intralipid.

Concentration dependent adverse reactions for Lignocaine		
Plasma con. Mcg/ml	Adverse reactions	
4	Light-headedness, tinnitus, circumoral numbness	
6	Visual disturbance	
8	Muscle twitching	
10	Convulsions	
12	Unconsciousness	
20	Respiratory arrest	
24	Circulatory depression	

Chart No. 6.1: DRUG TOXICITY OF LIGNOCAINE

Methaemoglobinaemia may be treated by the intravenous administrations of 1% solution of Methylene blue in a dose of 1mg/kg.

7. Local Anesthetics:

- a) Inj. Lignocaine 2 % (3 to 4 mg/kg body weight).
- b) Inj. Lignocaine hydrochloride (2%) with

Adrenaline (5 to 7 mg/kg body weight).

c) Inj. Ropivacaine (0.75%) or Inj. Bupivacaine (0.5%).

8. Sedation

Inj. Midazolam 0.05 mg/kg and Inj. Ketamine 0.5 mg/kg and Inj. Propofol infusions in 25-75 mcg/kg/min range and deep sedation (50 to 100 mcg in drip for deep sedation).

9. Interscalene

Success rate in posterior Interscalene (Cervical PVBs) approach is more than anterior-lateral approach.

10.Supraclavicular/ Infraclavicular

Inferior-posterior quadrant of plexus for Supraclavicular Block and Infra Clavicular Block near posterior cord.

11. Sciatic (Infragluteal)

Infragluteal approach is more comfortable to

patients than Classic Labat approaches.

12. Explanatory Footnotes on Above Protocol

12.1 Local Anesthetics:

For consistency, 0.75% Ropivacaine is primarily used for blocks, with a dose of 20-40 ml depending on the type of block. Ropivacaine results in 12-24 hours block duration. 2% Lignocaine for 3-4 hours duration. Mixing a short acting and long acting local anaesthetic together appears to significantly decrease the duration of the block.

12.2 Adjuncts:

- a) **Clonidine:** 50-100 ug for an average of 3-6 hours increased duration.
- b) **Epinephrine and Clonidine**: Increase duration of regional block.
- c) **Inj. Dexamethasone:** 4 mg to 8 mg dose as it may result in very prolonged blocks.
- d) **Bicarbonate:** Dose is 1 mEq/10ml, in Bier Block.

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Further reading



7. CARDIO PULMONARY- RESUSCITATION (CPR)

CPR consists of a series of maneuvers by which oxygenated blood supply to brain and vital organs is maintained during cardiopulmonary arrest (CPA) i.e., cessation of respiration and circulation. In children, CPA is not sudden but end result of long period of hypoxemia secondary to inadequate ventilation, oxygenation or circulation. Therefore, prompt management of these is essential to prevent CPA, the outcome of which is poor.

1. Diagnosis of Cardiac arrest

- a) Absence of pulse in major arteries (carotid or femoral in older children and femoral or brachial in infants as carotid is difficult to palpate due to short neck).
- b) Absence of heart sounds on auscultation.
- c) A systole / ventricular fibrillation on ECG.

2. Respiratory arrest

Absence of respiration on looking (absent chest movements), listening (absent air flow on bringing ears in front of mouth) and feeling (absent air flow on keeping hands in front of mouth or nose).

3. Levels of CPR

There are two levels of CPR:

- a) BLS (Basic life support) the elements of CPR provided without additional equipment, skill and speed are more essential.
- b) ACLS (Advanced cardiac life support) Use of equipment and drugs for assisting ventilation or circulation.

3.1 Basic Life Support:

Call for help; position the victim supine on firm flat surface with head level with the heart. As per New AHA 2014 Guidelines ABC (Airway – Breathing – Circulation) changed to CAB (Circulation - Airway – Breathing) and chest compression and rescue breath ratio is 30:2.

3.1.1Circulation-

Determine the absence of pulse after 2 breaths (rescue breaths). External cardiac massage if Asystole and unresponsive to rescue breaths. In children, perform cardiac massage if HR<60/min with signs of poor perfusion.

3.1.2 Airway-

Clear airway cleaning like blood, secretions, foreign particles (suction if available).

Prevent posterior displacement of tongue due to muscle relaxation during CPA, by head tilt and chin or jaw thrust (may use an airway if available).

Head tilt: Put a hand at forehead and tilt head back to sniffing or neutral position in an infant and little more in older children and adults. (Caution: In a patient with suspected cervical spine injury head tilt should be avoided)

Chin lift: Put finger of other hand under bony part.

Jaw thrust: Place 2-3 fingers under each side of lower jaw at its angle and lift jaw upward with the elbow resting on the surface on which victim is lying.

3.1.3 Breathing-

Determine the absence of breathing. Give mouth to mouth / nose / mask / airway breath (may use bag and mask if available). Inhale and then make a seal around the mouth and nose together in infant and seal mouth only in older children and adults (nose pinched with the hand used for head tilt) to exhale smoothly. Rate of breaths should be 20/min for infants, 15/min in older child and 10-12/min in adults.

Rescuer should stand or kneel at the side of the patient so that his hips are on a level with the victim's chest.

In a newborn 2 thumbs are positioned side by side on sternum just below the nipple line, with fingers encircling chest and supporting the back and compress sternum by 0.6 - 1.2 cm (120/min).

In an infant put index finger at the intersection of inter mammary line and sternum. Use 2 - 3 fingers (index, middle and ring) to compress sternum by 1.5 - 2.5 cm (100/min) and do not lift the finger when compression is released. Two thumb – encircling hands technique can also be used.

In children (1-8 years) use heel of hand on lower half sternum with long axis of heel same as long axis of sternum and compress 2.5 - 3.5 (100/min) In adults the heel of one hand is placed on the lower sternum and the other hand placed on top of the first. The elbows should be locked in position with the arms

straight and the shoulders over the hands. Sternum should depress by 3.5 - 5.0 cm and the rate of compression should be 80 to 100/min. (CAUTION: Do not exert pressure on the ribs, costal cartilages or xiphoid).

3.1.4 Combination of ventilation and Cardiac massage -

If both cardiac and respiratory arrest – Compression: ventilation = 30:2 in adults and children > 8; Children and infants 1-8 years = 5:1; Neonates =3:1.

3.2 Advanced Cardiac Life Support (ACLS):

If ACLS facility is available, shift the patient to ACLS as soon as possible. If this is not available then continue cardiac massage till spontaneous HR is more than 60-80/min and continue Endotracheal intubation with IPPV till adequate respiratory efforts are present (good chest movement, no cyanosis or shock). For ALCS proceed in the following order;

3.2.1 Procedures-

ECG monitoring (if available)

- a) If ventricular fibrillation defibrillation.
- b) In adults, first shock at 200 Joules; if the first is unsuccessful then a second shock at 200-300 Joules. If both fail, additional shocks at 300-360 Joules are given.

In children, 2 Joules / kg and can be repeated a few time (if does not revert to normal rhythm). Continue cardiac, massage in the meantime.

- c) All patients require oxygen (100%) because even with best CPR, only a fraction of the cardiac output is provided and also there are other factors causing ventilation perfusion mismatch.
- d) Establish IV line as early as possible to give drugs and fluids and intubation of trachea should be done to continue artificial ventilation.

3.2.2 Drugs are used in the following order if indicated-

a) Inj. Adrenaline

Indication: Asystole symptomatic bradycardia unresponsive to ventilation.

In adults: 1 mg IV every 3 – 5 minutes.

In children: 0.1 ml/kg of 1: 10,000 solution (0.01 mg/kg) IV, intra-osseous or 0.1 ml/kg of 1:1000 solution by endotracheal tube followed by several positive pressure breaths. Can be repeated every 5 minutes by either route. IV route is preferred and

should be used as soon as IV access is achieved (Intracardiac route is not desirable).

b) Inj. Lignocaine

Indication: Ventricular tachycardia or fibrillation non responsive to recurs after defibrillation.

In adults: Initials bolus dose is 1.5 mg/kg. Additional bolus of 0.5-1.5 mg/kg can be given 5-10 minutes during CPR up to a total dose of 3 mg/kg.

In children: Inj. 1 mg/kg IV stat followed by infusion at 20-50 mcg/kg/min.

c) Inj. Amiodarone

Indication: Refractory shock with ventricular fibrillation (as an alternative to or after failure of Lignocaine).

In adults: Initially 300 mg rapid infusion in 20-30 ml saline followed by 150 ml over 10 minutes followed by 1 mg/min for up to 0.5 mg/kg/day.

d) Inj. Atropine

Indication: Vagally mediated bradycardia during intubation, HR<80 or asystole in an infant and symptomatic bradycardia with AV block in any child. **Dose and route**: 0.02 mg/kg bolus (not <0.1 mg or> 0.5 mg for a child and 1.0 mg for an adult) This dose may be repeated after 5 minutes for a maximum total dose of 1.0 mg for a child and 2.0 mg for an adult.

e) Inj. Naloxone

Indication: Narcotic overdose or poisoning and newborn resuscitation (if mother has been given Morphine or Pethidine during labour). **Dose and route**: 0.1 mg/kg IV.

f) Inj. Sodium Bicarbonate (NaHC03)

Not required routinely as it can cause Alkalosis later and worsen respiratory acidosis by releasing CO2 in inadequate ventilation.

Indication: Hyperkalemia, significant metabolic acidosis (pH<7.2) or prolonged CPR.

In adults and in children: Inj. Sodium bicarbonate 1 mEq/kg stat and 0.5 mEq/kg every 10 minutes in protracted resuscitation.

g) Inj. Calcium

Indication: Not used routinely now a day unless there is Hyperkalemia, Hypocalcaemia or Calcium channel blocker toxicity.

Dose and route: In children, 0.5 ml/kg of Calcium gluconate IV. In adults, 10 ml to be given as a slow infusion under ECG monitoring.

h) Inj. Glucose

Indication – Hypoglycemia

Dose and route: - 0.5-1 g/kg IV. Try to get ABG, serum electrolytes and blood sugar (dextrose stick /glucometer) – post resuscitation care.

- Maintain mechanical ventilation for several hours to ensure adequate oxygenation and ventilation.
- Look for and treat seizures.
- Inj. Mannitol 0.5 -1 g/kg IV if raised intracranial tension.
- Maintain temperature, fluid and electrolyte balance and ABG.
- Treat shock with fluids, Dopamine, Dobutamine and adrenaline infusion as required.

3.2.5 Summary of CPR-

• Treat the underlying pathology causing CPA.

3.2.3 Monitoring-

Pulse should be palpable and chest expansion should be seen during effective CPR, blood pressure, Sp02, ET CO2 (In intubated patient and if facility available), ABG should be monitored during and soon after CPR.

3.2.4 Termination of CPR-

If asystole persists for >10 minutes after CPR has been performed, ventricular fibrillation eliminated, and confirmed, adequate ventilation provided and appropriate medications given.

Chart No. 7.1: CPR GUIDELINES FOR HEALTH CARE PROVIDERS

	Adult	Child	Infant	Newborn
		(<8 yr)	(< 1 yr)	
Lone	If known asphyxia arrest, 5	5 cycles	of CPR	Ventilation priority,
Rescuer	cycles	unless sudden		compressions for HR<60,
Priority	CPR	witnessed collapse		
Compression		1-hand	2-thumbs	2-thumbs
method		Or	Or 2-fingers	or 2-fingers
	2-hands	2-Hand	If single	for access to
			Rescuer	umbilical vein
Compression Rate	100/min	100/min	100/min	200/min
Ratio of	30:2 until intubated.	30:2 until intubated.		30:1
compressions to	8-10 breaths/min	8-10 breaths/min		
ventilations	After ET.	After ET.		
Use of AED	Immediate AED	Immediate AED Consider		No
	Consider 5 cycles	5 cycles CPR if >4-5Min		Recommendation
	CPR if $> 4-5$ min	Unwitnessed Arrest.		(for infant &New Born)
	Unwitnessed arrest			

(Method, compression rate, and ventilation to compression ratio)

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Further reading



8. ANAESTHESIA OBSTETRIC PROTOCOL

1. Orientation

Labour room and the operation theatre should be connected with each other. All the anaesthetic equipment's and preparations should be ready 24/7 days apart from it. Laryngoscopes with stubby handle and McCoy blade should be available. NICU and prenatal ICU should be on the same floor. Emergency drugs and plasma expanders (e.g. Hetastarch & Voluven) should be present in the O.T. Blood and blood products should be available at nearest possible place. Anesthesiologists should check for anaesthesia machines, defibrillator, Oxygen, other equipment's and the required drugs.

2. Labour analgesia with Epidural technique

2.1 General Aspects:

- a) Proper pre-operative evaluation with counselling of the patient and the relatives.
- b) Explaining the procedure and its advantages and disadvantages.
- c) Secure broad gauge I.V. cannula with starting I.V. with crystalloid for preloading.
- d) Epidural catheter inserted with standard technique in lateral or sitting position under aseptic precautions at L3-L4 OR L4-L5 space. No CSE should be performed above L1 level.
- e) The catheter should not be more than 2 to 3 cm inside the epidural space. Check for any fluid coming out of the catheter.
- f) Can give the analgesic dose of local anaesthetic agent, preferably 0.125% of Bupivacaine about 8 ml with 2 mcg / ml of fentanyl can be given.
- g) The dose can be repeated as per the patient's demand. The patient should be hemodynamically monitored closely till the end of delivery.
- h) If intervention needed, then continue the epidural anaesthesia with higher conc. of Bupivacaine i.e. 0.5% around 8 to 10 ml. can be given for LSCS.

2.2 Complications:

- a) Epidural-Bloody Tap
- b) Patchy action or unilateral block
- c) Subdural Block

3. Antacid Prophylaxis

All patients for elective Caesarean Section must receive oral antacid premedication. If the patient posted for an emergency Caesarean Section then consider IV Metoclopramide 10 mg or Inj. Ondansetron 4 mg or IV Ranitidine 50 mg. Emptying the stomach with a large orogastric tube, keep it until patient anaesthetized and up to extubation.

4. Regional Anaesthesia for Caesarean Section

4.1. Advantages:

The avoidance of the hazards of aspiration of gastric contents and failed intubation during general anaesthesia. A reduction in blood loss / thromboembolism/absence of Neonatal depression. Postoperative analgesia may be provided using spinal or epidural opioids.

4.2. Disadvantages:

Hypotension can occur due to aorto-caval compression despite the use of left uterine displacement and seemingly adequate preload.

4.3. Nausea and vomiting:

The incidence of this is often related to hypotension and hence should be treated immediately. Nausea following a regional block is an indication of the B.P. fall. Consider giving 100% oxygen and legs up position before IV Ephedrine. Use Oxytocin for uterine contraction.

Diclofenac:

Do not administer Diclofenac to those allergic to NSAIDs. Do not give it to unstable asthmatics. Also avoid in those with renal dysfunction, peptic ulcer disease and severe pre-eclampsia especially if oliguria or thrombocytopenia.

5. General Anaesthesia for Caesarean Section

5.1 Pre-operative assessment:

Thorough pre-operative assessment.

- a) Antacid prophylaxis.
- b) NBM status.
- c) Informed consent from relatives.

- d) Make arrangement for blood and blood products.
- e) Patients should be transferred to theatre in left lateral position.
- f) Administer Sodium citrate prior to preoxygenation.
- g) IV access 18G or 20G cannula.
- h) Position of the mother supine on the table with 15 degree left lateral tilt with slight head up.
- i) Pre-oxygenate the mother using 100% oxygen for 3 minutes unless there is severe maternal and fetal emergency.
- j) ECG, NIBP and pulse Oximeter during preoxygenation and ETCO2 is switched on.
- k) Perform RSI using Thiopentone 5-6 mg/kg and Suxamethonium 1.5mg/kg. Intubate after adequate action of Suxamethonium.

Please remember - Patients die from prolonged attempts to intubate leading to hypoxia and from unrecognized oesophageal intubation and not from failure to intubate. "If in doubt, take it out" Ensure that the ETT is correctly placed by observing the Capnograph trace and by listening to breath sounds. Give Atracurium when Suxamethonium action wears off.

5.2 Intubation Hints:

Optimize the patient's position. A smaller ETT is often needed in obstetrics. An unexpected invisible larynx is often due to incorrectly applied cricoid pressure. And short stubbed laryngoscope or polio blade. An anterior larynx just out of reach of the ETT can usually be cannulated with a well lubricated gum-elastic bougie.

6. Anticoagulation and regional anaesthesia

- a) The incidence of epidural haematoma is less than 1: 100,000.
- b) NSAIDS are not a contraindication to CNB.
- c) Enoxaparin 40mg SC post-operatively.
- d) If the woman is on antenatal thromboprophylaxis. a) Enoxaparin should be stopped 12 hours prior to performing CNB. b) Epidural catheters should be removed 2 to 4 hours prior to administration of Enoxaparin.
- e) Heparin a) Heparin should be stopped 4 hours prior to attempting neuraxial blockade or removal of epidural catheters. b) Unfractionated Heparins should not be administered for 2 to 4 hours after catheter removal.

7. Management of a Dural tap

Don't panic! Place you thumb against the hub of the Touhy needle, relax and think. You have 2 options:

- a) Can give 0.5% Bupivacaine 2 2.5 c.c intrathecally and do LSCS under spinal anaesthesia.
- b) Insert epidural catheter intrathecally. Approximately 2cms should be threaded into the subarachnoid space.
- c) Remove the Touhy needle and insert at a different level. Don't perform a CSE. Intrathecal catheter top up only by the Anaesthetist. You can administer either: 1ml 0.25% Bupivacaine + 15-25µg of Fentanyl as regular top up. Or 0.5 2ml of standard LDM as regular top up. After delivery remove catheter as usual. If epidural in situ treat as normal Intrathecal epidural, but each top-up to be given by Anaesthetist under strict aseptic conditions.

8. Central Neural Block

8.1. Indications:

Labour analgesia. Anticipated difficult or operative delivery (e.g. multiple pregnancy, breech, premature foetus), Obstetric disease (e.g. pre-eclampsia), Maternal disease (e.g. specific cardiac, respiratory or neuromuscular disease), Surgery during pregnancy.

8.2. Contraindications:

This list contains both

- a) Absolute contraindications- Patient Objection, Local Sepsis, Coagulopathy.
- b) **Relative contraindications** Hemorrhage with hypovolemia, Raised intracranial pressure, Some forms of anticoagulant therapy, Disease of the nervous system, Gross spinal deformity, Systemic sepsis, Severe foetal distress.

8.3. Central Neural Block - Special Considerations

- a) **Low Platelet Count**: A platelet count of <50000 is an absolute C/I to a regional block.
- b) **Enoxaparin /L MWH:** Many patients at risk of venous thrombosis are on daily sub cutaneous Enoxaparin.
- c) **Unfractionated Heparin (UH):** If UH has been given, wait 4-6 hours before siting an epidural block. Remove the epidural catheter at least 4 hours after the last dose of UH.
- d) **Aspirin**: Patients on aspirin can have a regional block. A bleeding time is not required.
- e) **Pre-eclampsia (PIH):** Patients with mild to moderate PIH should have a recent (within 24 hrs.) platelet count before a regional block.
- f) Maternal Pyrexia: Administer antibiotics.

- g) **Haematological Disorders:** The most common type of patient presenting for regional blockade are Haemophilia Carriers and patients with Von Willebrand's Disease.
- h) HIV: HIV is not a Contraindications to a regional block nor indeed to a blood patch. Standard high risk precautions should be taken when instituting a block. Consider double glove protection / eye protection / do not resheath needles when performing a block.
- i) **Prolapsed Intervertebral Disc (PIVD)** Epidural or spinal analgesia / anaesthesia are not contraindicated.
- j) Harrington Rods / Spinal Instrumentation Many of these patients have severe scoliosis with consequent cardiorespiratory compromise. The surgical site should be avoided. The epidural needle should be advanced towards the convexity of the curve.
- k) Multiple Sclerosis (MS) MS is a CNS demyelinating disease with an incidence of 1:10,000. It is a disease of relapse and remission.

9. Placenta Previa

9.1. Anaesthetic management:

- a) Ensure adequate IV access (2x 14 or 16G cannulae).
- b) Two anesthetists preferable if Grade 4, previous Previa or previous LSCS; this is mandatory if proceeding under regional (see below).
- c) Manage major hemorrhage according to guidelines.
- d) Anaesthetic technique considerations.

9.2. Action:

- a) Ensure adequate airway / conscious level. Give high flow oxygen.
- b) At least 2 peripheral lines will be needed of not less than 14- 16G. If the decision is made to set up invasive monitoring, it must not interfere with resuscitation.
- c) Prevent Aorto-caval compression.
- d) Take at least 20ml of the patient's blood for:
 - i. Blood grouping / cross matching
 - ii. Full blood count
 - iii. Coagulation studies (including fibrinogen / FDP's if abruption or other cause of DIC suspected).
- e) Blood Transfusion
 - i. Order a minimum of 4 units of blood.
 - ii. All patients should be given blood of their

own group as soon as possible. However, for patients with severe haemorrhage, uncross matched group O, Rh negative can be lifesaving.

- iii. The principles of intelligent management are: While blood is gushing out it is useless and wasteful to give clotting factor or platelet replacements. Once surgical haemostasis has been more or less achieved, continued oozing may be due to blood clotting factor deficiencies. Further blood samples should be sent for coagulation screen and platelet count.
- iv. If there is massive blood loss the MASSIVE TRANSFUSION PROTOCOL should be instigated. This will ensure that FFP is available and platelets are ordered. While FFP can up to a point be issued "blind" as already mentioned, the possibility of low platelets or DIC may need different blood components for their correction. This can only be identified by laboratory testing.
- v. A pressure bag system is essential in order to infuse fluids rapidly.
- vi. Use a blood warmer as soon as possible.
- vii. Blood filters are NOT needed and will slow down transfusion.
- viii. Early use of CVP monitoring and direct arterial pressure monitoring.
- ix. Think ahead to order blood and blood products in plenty of time.
- x. Use the Hemocue (available in Theatre) to aid estimation of transfusion requirements.
- f) Measure the patient's temperature.
- g) Additional Calcium is rarely needed and only if there is evidence of a calcium deficiency. 10% calcium chloride is preferable to calcium gluconate.
- h) Treatment of the cause may involve delivery of baby and placenta, repair of lacerations and administration of Oxytocin (Ergometrine; 15methyl PGF2α [Carboprost - Hemabate]; PGE2 [Misoprostol].
- All patients with more than moderate continuing haemorrhage need proper monitoring of pulse rate, CVP, blood gases, and urinary output as well as dedicated care by the midwifery and medical staff. Serious consideration should be given to the potential advantages of transfer to an HDU.

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Further reading



9. SPINAL ANAESTHESIA

1. Definition

Spinal anesthesia is produced by introducing a Hyperbaric or hypobaric or Isobaric anesthetic into cerebrospinal fluid in the subarachnoid space result in to loss of sympathetic tone, sensation and motor function.

Sympathetic block is 2-3 segments higher than the sensory and the motor block 2-3 segment lower than the sensory block.

Position – 1) Sitting 2) lateral

2. Approach - Median and Par median

2.1 Saddle block given in sitting position by Hyperbaric anesthetic drug:

Spinal cord usually ends at the level of L2 in adult and L3 in children.

Important land mark is that line joining the top of the iliac crest corresponds to L4-5.

2.2 Practical implication:

While administering spinal anesthesia spinal needle pierce 1) Skin 2) Subcutaneous fat 3) Supraspinious & Interspinious ligament 4) Ligamentum flavum 5) Epidural space 6) Dura 7) sub arachnoid space.

Patient is adequately prepared, with the procedure fully explained, has reliable intravenous access, is in a comfortable position and resuscitation equipment is immediately available.

To be done under all aseptic precaution. Read the label.

Patient should be well hydrated and NVM at least 6 hrs.

Strict intraoperative vital parameters monitoring is mandatory. Height of analgesia is directly proportional to curvature of spinal column and volume of drug, specific gravity, force and rate of injection, barbotage technique.

Small bore needle makes smaller hole in the dura i.e. separate Cauda filum and are associated with a lower

incidence of headache than conventional cutting edge needle.

Preexisting neurological deficit has to be documented i.e. Diabetic neuropathy, Hanson's disease, Traumatic neuropathy

2.3 Drugs used for Spinal anesthesia:

- a) Inj. Lignocaine 5% solution
- b) Inj. Bupivacaine 0.5% solution
- c) Inj. Ropivacaine 0.75% solution with adjuvant-1) Inj. Clonidine 2) Inj. Buprigesic

2.4 Advantages:

- a) Economical.
- b) Minimum equipment required.
- c) Control hypotension and bradycardia reduces the bleeding at the site of surgery.
- d) Less respiratory infection.
- e) Less incidence of thromboembolic and pulmonary complication.
- f) Useful in sickle disease / trait and in Diabetic patient (Less stress related hyperglycemia).

2.5 Contraindications:

2.5.1 Absolute-

Allergy to local anesthetics, Local infection at needle insertion, Increased intracranial pressure (herniation of brain stem)

2.5.2 Relative-

Hypovolemia, Anticoagulation treatment, Systemic sepsis, Neurological diseases- Multiple sclerosis, Tabes dorsalis, Syringomyelia, Amyotrophic lateral sclerosis, Tumors of the spine, Airway compromise, Low back pain and peripheral neuropathies, Haemorrhagic CSF.

2.6 Complications:

2.6.1 Peri-operative-

Hypotension due to vasodilatation and bradycardia due to increased vagal tone as a result of sympathetic blockade, Respiratory insufficiency due to intercostal blockade, poor anaesthesia because of loculations of arachnoid.

2.6.2 Post- Operative-

Headache due lo leakage of CSF or aseptic inflammatory reactions, Urinary retention, Backache, Infection. - Meningism, Arachnoiditis, Transverse myelitis or the Cauda equine syndrome with varying patterns of neurological impairment and sphincter disturbances. Para-paresis and rarely paraplegia due to the "anterior spinal artery syndrome" i.e. prolonged vasospasm of the anterior spinal artery by the vasoconstrictor adjutants or due to prolonged uncorrected hypotension.

2.7 Treatment:

Hypotension – Nausea and vomiting may be the first sign of hypotension. Give adequate I.V. Fluid, Oxygen by mask, increasing the patient's circulating volume is by raising their legs, Vasopressors drug - Inj. Ephedrine 30 mg (6mg/cc) Inj. Phenyl epinephrine, Inj. Noradrenaline, Infusion. (titrated)

2.7.1 Total Spinal -

Needs to be quickly recognized and treat patient will be initially unable to talk louder than a whisper and will stop breathing. These patients have to be intubated and ventilated until the local anaesthetic effect wears off and also maintain the hemodynamic stability.

2.7.2 Headache post dural puncture headache (PDPH)-

Encouraged to drink water, give intravenous fluids to maintain adequate hydration, strict bed rest, Paracetamol, Aspirin or Codeine tea, coffee, epidural blood patch, Tab. Samaritan.

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11. Pathology

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1. INTRODUCTION

The Pathology department basically deals with diagnosis of disease by means of various types of tests performed on body fluids, blood and tissues.

It is divided into 4 sections:

1) Section I – Haematology Section:

In which the blood, urine and stool examination done to detect any abnormality like anemia, leukemia, renal infection and bowel infection and infestation

2) Section II – Serology Section:

It deals with the rapid terms to detect M.P., Typhoid, V.D., RC H.I.V. etc. along with blood culture to detect Typhoid etc.

3) Section III – Biochemistry Section:

It deals with different types of biochemical tests to detect functions of liver, kidneys, lungs, heart etc.

4) Section IV – Histopathology & Cytopathology:

In this section mainly tissue cell morphology is studied to detect infection / tumor in body.
2. HAEMATOLOGY SECTION

1. Specimen Collection:

Every specimen must be accompanied by a request slip, which should indicate date, patient's full name, age and sex, hospital identification number, name of referring doctor, provisional diagnosis, kind of specimen, laboratory services and equipment, exact time the specimen was obtained and the name of the person who collected the specimen with signature. It is necessary that the request slip be complete and clearly written. Incomplete information may lead to total loss of the report.

Specimens should be processed as early as possible. Temporary storage in refrigerators is advisable except for C.S.F. and specimens submitted for bacteriological cultures. Urine and other body fluid should be processed as early as possible (before 2 hrs.) as delay causes disintegration of cells and loss of morphology. Blood is one of the most common specimens studied in laboratories. Blood will clot within few minutes after it is removed from the body unless an anticoagulant is used.

1.1.1. Whole Blood: Blood with anticoagulant is called whole blood.

1.1.2. Plasma: Fluid portion of unclotted blood is called plasma and is obtained from anticoagulated blood.

1.1.3. Serum: Fluid portion of clotted blood and is obtained from clotted blood, which is collected without any added anticoagulant.

1.2. Anticoagulant:

The anticoagulant prevents the blood from clotting. Most of anticoagulants remove calcium which is one of factor required for coagulation.

1.1. Blood:

Sr. No.	Type of bulb	Content (Anticoagulant)	Used for
1	EDTA	Na and K salt of EDTA	Routine Haematological work
2	Trisodium Citrate	Sodium Citrate	ESR
3	Heparin	Heparin	PCV, Osmotic Fragility test
4	Double Oxalate mixture	NH4 and K Oxalate	CBC, Blood Group
5	Sodium Fluoride	Na Fluoride and K Oxalate	Sugar

Table 1: Various types of bulbs & its uses

1.3. Vacutainers:

These are plastic tubes with coloured cap with capacity of3-5ml blood used in laboratory now a day. These may be with or without vacuum (The small glass bottles can also be used for sample collections after it is properly prepared). The colour of the cap indicates the presence of anticoagulant present inside the tubes. The label is attached with the tube for patient's identification. Following are the types and uses.

1.3.1. RED Cap No chemical for serum separation (Biochemistry and Serological test)

1.3.2. GREY Cap: Sodium fluoride + Pot oxalate for glucose estimation

1.3.3. PURPLE Cap: Potassium EDTA for CBC, Hb, TLC, DLC, PBS (Peripheral Blood Smear), Reticulocyte count, ESR

1.3.4. LIGHT BLUE Cap: 3.2% Sodium Citrate for coagulation studies (PT, APTT, TT, FDP, D-Dimer, Factors – VIII & IX assay)

2. Hemoglobin Estimation 2.1. SAHLI'S (Acid Haematin) Method –

This method used in small laboratories at PHC level.

2.2. Principle –

When blood is added to N/10 HCL, Hb is converted to brown coloured acid Haematin. The resulting colour after dilution is compared with standard brown glass reference blocks of Sahli's haemoglobinometer.

Table 2: Haemoglobin estimation according to Sahli's method

Sr. No.	Test Name	Methods	Normal Value	Interpretation
1	Haemoglobin	a) Sahli's Method	Male 13-18gm/dl	Mild Anaemia up to 10gm%
		b) Hemocue Method	Female 12-16.5gm/dl	Mod. Anaemia up to 8 gm%
		c) Cell counter Method	Pregnancy 11-14gm/dl	Severe Anaemia below 7gm%
				Raised Hb- Polycythaemia

Table 3: WHO classification for Anaemia

Population	Non –Anaemia	Anaemia (Ha	emoglobin in gra	ms per litre)
		Mild	Moderate	Severe
Children 6 - 59 months of age	11 or higher	10 - 10.9	7 - 9.9	Lower than 7
Children 5 - 11 years of age	11.5 or higher	11 – 11.4	8 - 10.9	Lower than 8
Children 12 - 14 years of age-	12 or higher	11 – 11.9	8 - 10.9	Lower than 8
Non-pregnant women (15 years of age and above)	12 or higher	11 – 11.9	8 - 10.9	Lower than 8
Pregnant women	11 or higher	10 - 10.9	7 - 9.9	Lower than 7
Men (15 years of age and above)	13 or higher	11 – 12.9	8 - 10.9	Lower than 8

3. Leucocyte count:

3.1. Peripheral blood smear:

Preparation and staining of peripheral blood film is performed for doing total leucocyte count (TLC) and differential leucocyte count (DLC).

3.2. Automated Differential Leucocyte Counting:

This system is able to replace manual differential leucocyte count to a great extent. When any abnormal cell count or abnormal morphology of cells in seen, this system shows a flagging thereby suggesting a visual count mandatory in such cases.



Figure 3: Various types of White blood cells

Sr. No.	Test Name	Methods	Normal Value	Interpretation
2	Total Leucocyte	Manual by counting	4000-11000 cells / Cu mm	Raised TLC in Viral, Bacterial
	Count (TLC)	Cell counter method		Infections, & Leukemia
				Decreased TLC - Septicemia, Enteric fever, Aplastic Anemia.
3	Differential Leucocyte	Cell Counter	Polymorphs 40-65%	Raised in Acute Bacterial Infections.
	Count (DLC)	Manual by staining Slides	Lymphocytes 20-35%	Raised in chronic infection like tuberculosis, leprosy
			Eosinophils - 2-4%	Raised in worm infestation, allergic skin infection, Tropical
			Monocytes - 2-8%	Raised in viral infection
			Basophils - 0-1%	Raised in Leukemia

4. Estimation of erythrocyte sedimentation rate (ESR):

4.1. Principle:

When well mixed anticoagulated blood is allowed to stand undisturbed in a vertical tube the erythrocytes tend to sink to the bottom. Two layers are formed the upper plasma layer and lower one of red blood cells. The length of fall of the top of the column of erythrocytes in a given interval of time is the ESR (erythrocyte sedimentation rate).

Stages in the ESR – three stages in the ESR can be observed: -

- i. In the initial 10 minutes, there is rouleaux formation.
- ii. For about 40 minutes, sedimentation occurs at a constant rate.
- iii. Sedimentation slows in the final 10 minutes as cells pack at the bottom of the tube.

Sr. No.	Test Name	Methods	Normal Value	Interpretation
4	ESR	Wintrobe Tube Method Westergren Tube Method	Male 0-10 mm at 1 hr. Female 0-20mm at 1 hr.	Raised in chronic infection like Tuberculosis, Leprosy, Chronic
				anemia.

4.2. Interpretation or clinical significance of ESR:

ESR is markedly elevated in monoclonal blood protein disorders. such as Multiple Myeloma or Macroglobulinemia, Polyclonal Hyperglobulinemia, inflammatory due disease and to in Hyperfibrinogenemia. Moderate elevations are seen in active inflammation such as Rheumatoid Arthritis, Chronic infections such as Tuberculosis, Collagen disease and Neoplastic disease. Although ESR is a non- specific phenomenon, this indicates the presence of an active disease. If patient is improving ESR tends to fall. It provides an index of progress of diseases such as TB, Rheumatoid Arthritis. It is also a useful screening test in routine examination. An elevated ESR occurs as an early feature in Myocardial Infarction.

ESR is especially low in (0-1mm) Polycythaemia, Hypofibrinogenemia, CHF, and Sickle cell anaemia.

5. Bleeding time:

This test measures the time taken for blood vessel constriction and platelet plug formation to occur. No clot is allowed to form, so that the arrest of bleeding depends exclusively on blood vessel constriction and platelet action. This is one of the most important preliminary test for bleeding disorders and preoperative investigation in patients going for surgery.

The duration of bleeding from a standard puncture wound of the skin is a measure of the function of platelets as well as the integrity of the vessels wall.

Commonly used methods are: Duke's Method & Ivy's Method.

Bleeding time is prolonged in:

- i. Thrombocytopenia (Dengue Fever) DIC (Disseminated Intravascular Coagulation)
- ii. Acute Leukaemia Von Willebrand's disease
- iii. Aplastic Anaemia Aspirin intake

6. Clotting time:

The whole blood clotting time is one of the simplest, imprecise, methods to determine the efficiency of the clotting process. Many conditions can disrupt the pathways that produce a clot, including hereditary disorders, liver disease and drugs that interfere with the normal function of platelets and coagulation factors.

METHODS:

- i. Capillary tube method
- ii. Lee and white method

Normal Range for clotting time 5-10 minutes.

7. Prothrombin Time:

PT test is an indicator of the efficiency of common pathway and extrinsic coagulation pathway, in which preformed thromboplastin is added to the test plasma in the presence of calcium and the time taken for clotting of plasma is noted. The main factors involved in the pathway are V, VII, X, II and I. Deficiency of any one of them leads to prolongation of PT. The PT test is used to monitor patients taking certain medications (e.g. Control of anticoagulant therapy) as well as sensitive to the presence of Heparin in blood and to low fibrinogen levels.

Sr. No.	Test Name	Methods	Normal Value	Interpretation
5	Bleeding Time B.T.	Duke's Ivy's Method	1-5 min	Raised in Coagulation defect, vasculo-toxic snakebite, rat poisoning, Hemophilia ITP.
6	Clotting Time C.T.	Capillary method	4-9 min	
7	Prothrombin Time P.T.	Quick's method or	12-16 sec	
		Semi-automated coagulometer	12-16 sec	Raised P.T. (factor no. II V, VII, & X)

8. Tests for Sickle Cell Anaemia

Sickle cell test is done for all ANC patients, for all population in tribal area & also done for all patients of severe anaemia.

Initially the patients are screened by Solubility test. If Solubility test comes positive, then it is confirmed by Hb Electrophoresis.

Sr. No.	Test Name	Methods	Normal Value	Interpretation
8	Sickle cell Anemia	Solubility test or Electrophoresis test or HPLC method	Sickle Hemoglobin (HbS) is normally absent	 (HbS) Present & less than 40% is sickle cell trait or carrier HbS more than 40% is sickle cell disease

Laboratory Tests

1. Routine screening tests (in any haematology laboratory)

2. Definitive or special tests (in reference laboratory)

Laboratory testing

- 3. Screening tests
- 4. Sickling test
- 5. Solubility test

8.1. Sickling test

Na – Metabisulphite is used

Cells form sickle shape under low Oxygen tension Homozygotes – 1 hour at 37 °C Heterozygotes – longer time



Figure 2 Sickling Test showing sickle cell

8.2. Solubility Test

- Reagents: Phosphate Buffer (pH 7.1)
- Stock solution: KH₂PO₄ 125 g
 - K_2 HPO₄ 217 g Saponin – 2.5 g DW– 1 litre

- Working solution: 10 ml of stock solution + 0.1g Na – dithionate just before use
- Procedure: 20 µl RBCs (washed) + 2ml working solution
- Read after 10 minutes





8.3. Confirmatory Test by Electrophoresis

Confirmatory test for HbS after Hb electrophoresis

Reason: Sensitivity is 93% compared to HPLC

- Cannot differentiate between HbAS & HbSS
- Preliminary test in remote area but confirmation must be done by HPLC or Hb electrophoresis. (3)

9. URINE EXAMINATION

The urine examination is referred to as a liquid tissue biopsy of the urinary system.

It is useful in -

- i. Diagnosis and management of renal or urinary diseases.
- ii. The detection of metabolic or systemic disease not directly related to the kidneys.
- Microscopic examination of the sediment may indicate the kind of lesion present or the state of activity of a lesion.

9.1. Collection of Urine: -

For routine work 15 ml or more is preferred and it should be collected in a clean container, after properly cleaning the external genital area to avoid vaginal secretions and cellular debris of the urethral meatus.

9.2. Specimen Evaluation:

This includes proper labelling (name, date and time of collection), proper specimen, proper receptacle, storage condition, preservative (Thymol, formaldehyde if added).

9.3. Physical Examination:

Following parameters are looked for:

- i. Quantity
- ii. Colour
- iii. Appearance
- iv. Character
- v. Odour
- vi. Specific gravity

9.4. Chemical examination

- i. Test for Proteins
- ii. Reducing Substances in Urine (Urine for Sugar)
- iii. Ketone Bodies
- iv. Bile Pigments
- v. Bile Salts

9.4.1 Benzidine Test (for occult blood)

Results: Faint Green-Trace

Green+

Greenish Blue++

Blue+++

Deep Blue++++

9.5. Microscopic Examination:

Examination of urinary sediment – the specimen should be examined while fresh since casts begin to lyse within 1 to 3 hrs. Mix the specimen well. Take 10 ml of urine in centrifuge tube, centrifuge at 2000 rotation/min for 5 minutes. Remove the supernatant.

Place a drop of sediment of slide and put a cover slip. Examine under both low power & high power.

Cells - RBC, WBC, Epithelial cells.

Cast - Hyaline, granular, epithelial, WBC cast, RBC cast, Fatty, waxy.

Crystal and amorphous chemical deposits.

Miscellaneous - Mucus, spermatozoa, bacteria, yeast, parasite.

Compare the urine strip results with the colour on bottle.

Table 1: Urine Examination

	Finding	Interpretation
Α	ROUTINE EXAMINATION	
1	Physical examination	
1.1	Colour- Yellow/Reddish	Yellow in Hyperbilirubinemia, Red in Hematuria
1.2	Appearance - clear/turbid	Turbid in UTI, Nephrotic syndrome, Preeclampsia, Eclampsia.
2	Chemical Examination	
2.1	Albumin - +/-	UTI, Nephrotic syndrome, Preeclampsia, Eclampsia
2.2	Sugar - +/-	Diabetes, Hyperglycemia
2.3	Ketone bodies - +/-	Present in Starvation, Diabetic Ketoacidosis.
2.4	Bile salts & Bile pigment - +/-	Present in Hyperbilirubinemia,
В	MICROSCOPIC EXAMINATION	
1	No of RBCS / HPF	Hematuria, renal calculi, normal menses

2	No. of Pus cells/ HPF	UTI
3	No. of Epithelial cells /HPF	Normally present
4	Casts - Present /Absent	Nephropathy
5	Crystals - Present / Absent	Present in Calciuria (Oxalate, Phosphate, Urate crystals)
6	Trichomonas - Present / Absent	It is motile parasite
7	Spermatozoa - present /absent	Present in contamination with semen
8	Bacilli - present / absent	UTI

10. Detection of Malaria Parasite by Malarial Antigen test (RDK)

Malaria is serious, sometimes fatal, parasitic disease characterized by fever, chills and anemia caused by a parasite that is transmitted from one human to another by the bite of infected female Anopheles mosquitoes. There are four kind of malaria that can infect humans: Plasmodium Falciparum, Plasmodium Vivax, Plasmodium Ovale and Plasmodium Malaria.

10.1. Principle:

Malaria antigen kit contain a membrane strip which is pre-coated with one monoclonal antibody and one polyclonal antibody as two separate lines across attached strip. One monoclonal antibody (test line PF) are specific to the HRP-II (Histidine Rich Protein- II, Specific to Plasmodium Falciparum) and other polyclonal antibody (Test Line PAN) are PAN specific to the Lactate dehydrogenase of plasmodium species (P. Falciparum, P. Vivax, P. Malariae and P.Ovale).

10.2. Interpretation:

In case of positive test, band for respective kind of malaria is visible.

In case of negative test, band is absent.

10.3. Peripheral blood smear for Malaria Parasite

Specimen collection by using Lancet-

Pierce tip of finger with sterile lancet & take one drop of blood on clean glass slide & spread it with the help of spreader to make thin blood smear

Fixation- Fix the blood smear by alcohol spray or spirit

Staining of Smear-Staining of blood smear by field A & B stain.

Microscopic Examination of peripheral smear: - After applying oil on smear with the help of oil emulsion lens screen the morphology of RBC's & any of the following form of malaria parasite.

Commonly present Gametocyte form – in	Malaria Detection	Thick & thin smear under oil emulsion	Ring form Trophozoite / schizont form, Gametocyte form	Ring form - multiple small rings - Falciparum, single large ring - vivax Trophozoite / schizont form Commonly present Gametocyte form – in
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Fig. 4: Different forms of Malaria Parasite

11. Rapid test for Leptospirosis

11.1. Principle: Qualitative and differential definition of IgG and /or IgM antibody to Leptospira in human serum or plasma. It is only a screening test.

11.2. Method: Rapid test is solid phase immunochromatographic assay.

11.3. Sample: Serum or plasma.

11.4. Procedure: Remove test device from sealed pouch and place horizontally. Add 5 micro liter of plasma or serum in sample well(s), odd 4 drops of diluents in another well provided. Read interpretation in 20 minutes.

11.5. Interpretation:

- 1) Negative- Only one color line at control © in result window.
- 2) Positive-
- IgM positive- Two color lines at control (c) and at Ig M (M)
- IgG Positive- Two color lines at control (c) & at IgG.
- IgM and Ig G Positive -Three color lines at control (c), IgM (M) &IgG.
- 3) Invalid Test- No control © line in result window.
- No color line in (c) & also in Ig M (M) and / or G. result window

12. Stool Examination:

12.1 Collection of Sample: -

Take approximately 5-10gm of stool passed by the patient at the end of defecation, it is usually semi solid with the half of wooden spatula (Ice cream stick) into a plastic container with cap (Urine collection cup). Morning sample is preferred and is transported to laboratory immediately.

12.2 Preservation of Faecal Sample: -

This is done when sample cannot be examined immediately or it has to be transported. The sample of stool is made into thin paste with tap water. To this add equal part of steaming 10% formalin and left for a few hours. The supernatant is discarded. It should be noted that formalin preserved ova become in a ratio of 30:1 to stool sample. Ova can be examined for about one month.

12.3 Examination of Stool for Ova and cyst:

Methods

- i. Direct Smear
- ii. Concentration by floatation method
- iii. Direct Centrifugal floatation.

	Finding	Interpretation
А	Routine Examination	
1	Physical Examination	
1.1	Colour - Black, White, Red	1. Black in Occult blood in upper G.I. bleed. 2. Reddish in lower G.I. bleed, 3. White in biliary obstruction
1.2	Consistency - soft, loose, hard, semisolid	Loose in Diarrhea & Dysentery, Hard in chronic constipation
1.3	Worms	Adult worm – Ascaris Lumbricoides, Pin worm (small round worm)
2	Chemical Test	
2.1	Reducing substance	For Lactose intolerance in infants
2.2	Occult blood	Upper G.I. bleed
В	Microscopic exam	
1	PUS cells +/- / HPF	Diarrhea & Dysentery

2	RBCS +/- /HPF	Lower G.I. bleed
3	OVA & Cyst	Cyst of 1. Entamoeba histolytica, Giardia Lamblia 2. Enterobius vermicularis (Pinworm) 3. Ancylostoma duodenale (Hook worm) 4. Taenia solium (Tape Worm)
4	Mucous +/-	Amoebic Dysentery

13. Sputum for AFB 13.1 Introduction:

Sputum should be obtained from the lower respiratory tract (Bronchi of the lung). Saliva produced by the salivary gland in the mouth is not sputum, but is frequently and mistakenly sent to the laboratory instead of sputum.

Note: -Purity of the specimen should be checked. True sputum should contain fewer than 10 squamous epithelial cells and more than 25 leucocytes/ low power field.

13.2 Specimen Collection:

The patient should be instructed properly as to the type of the specimen required. Specimen is collected in the early morning. It must be coughed up from deep down in the chest. Apply the aerosol technique if the patient is unable to produce the required specimen.

13.3 Direct Examination of Sputum

- Work safely with the sputum as it is infectious.
- If possible bleach the specimen with 5% sodium hypochlorite or bleach (1:1)

13.4 Acid fast staining (Ziehl-Nelson method)

Result:

- All acid fast bacilli are not pathogenic. So the result should be reported as-
 - AF organism present or positive.
 - AF organism absent or negative.
- Technician should examine 10 microscopic fields under oil immersion and then report.

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Further reading



3. SEROLOGY SECTION

Serological tests usually done in laboratory are broadly classified into following two groups.

- Agglutination Tests: This group includes tests like Widal test, VDRL test, RA factor test, Blood Group test, ASO test, CRP test, HBsAg test etc.
- Rapid Tests (Also known as Spot/ Line Tests): This group includes tests like, HBsAg test, HIV test, Dengue test, Malaria test, Hepatitis C test etc.

Serological Tests:

Various serological tests are done for diagnosis of diseases. Here we are discussing common serological tests like VDRL, HBsAg, HIV, Widal, RA factor, dengue antibody test.

1. RPR Test for Syphilis (VDRL)

The RPR carbon reagent is a stabilized suspension of cholesterol crystals coated with cardiolipin. Lecithin is added to adjust the sensitivity and charcoal particles to improve the reading of the reaction. The reagent act as antigen against antibodies present in person suffering from syphilis caused by Treponema pallidum. Reagents are a group of antibodies against some components of the damaged tissue from patient infected by Treponema pallidum.

This test is rapid method for diagnosis of syphilis, macroscopic agglutination indicates positive result.

1.1. Storage:

Kit should be stored at 2-8°C and protected from light.

1.2. Patient Sample:

Fresh serum (Free of Haemolysis)

1.3. Interpretation of Result:

Medium & Large aggregates against white back ground – Reactive

Finely dispersed aggregates against white back ground – Weak Reactive

No aggregates, even grey back ground - Non Reactive.

False positive results can be seen in diseases like leprosy, SLE, infectious mononucleosis, malaria, viral

pneumonia. Any positive result must be confirmed by other serological assay (e.g. TPHA).

2. HBsAg Test

2.1. Principle:

The HBsAg one-step Hepatitis B surface antigen test device (serum/plasma) is a qualitative, solid phase, two-site sandwich immunoassay for the detection of HBsAg in serum or plasma.

2.2. Interpretation:

Double line- Positive

Single line with control - Negative

3. Rheumatoid factor test

Principle

The RF reagent is a suspension of polystyrene latex particle sensitized with specially prepared Human IgG. When serum containing RF is mixed with the latex reagent visible agglutination occurs. When serum containing greater than 0.8mg/dl RF is mixed with the latex reagent, visible agglutination occurs.

Result: In positive case agglutination occurs.

4. ASO titer

Principle

It is an immunological reaction between streptococcal exoenzyme bound to biologically inert latex particles and streptococcal antibodies in the test specimen. When serum containing greater than 200 IU/ml of ASO titre is mixed with the latex reagent, visible agglutination occurs.

Result: In positive cases agglutination occurs.

5. Widal test

Principle

Widal kit contains killed bacterial suspension of Salmonella which carries specific 'O' and 'H' antigen. This reacts with immunospecific antibodies present in the sample resulting in agglutination.

6. HIV – Tridot spot test

Confirmation with ELISA test

7. Dengue –

Spot test ELISA test

8. Urine Pregnancy Test (UPT)

8.1. Principle: -

Qualitative detection of human chorionic gonadotropin (HCG) in urine in early detection of pregnancy.

8.2. Method:

Pregnancy test device (Urine) is rapid chromatography immunoassay for qualitative detection of HCG.

8.3. Sample:

Early morning sample for early detection of pregnancy.

8.4. Interpretation

Negative: -

One coloured line appears in control line region and no any coloured line appears in test region (+)

Positive: -

One coloured line in control and other coloured line in test.

Invalid test: -

No coloured line in control (c).

9. Semen Analysis/Examination.

9.1. Indication:

(a) To rule out male infertility

(b) Post vasectomy

9.2. To perform routine examination of semen

9.2.1. Requirement: -

Glass slide covers tip, pipettes, Neubauer counting chamber, pH paper, semen diluting fluid, wide mouth container for semen collection.

9.3. Specimen-

Freshly Collected Semen

9.4. Physical Examination:

Colour, volume, viscosity of semen.

9.5. Chemical Examination-

pH, Detection of fructose in semen.

9.6. Microscopic Examination:

Under low and high power

- Study of sperm motility & its percentage
- Morphology's Abnormal forms & its percentage
- Pus cells, RBC or epithelial cells / HPF
- Determination of sperm count with help of Neubauer chamber & semen diluting fluid.

Note: It is necessary to note while reporting for routine semen examination

- Abstinence days
- Method of sample collection
- Time & place of Sample collection.

10. Swine Flu Diagnosis / Sample collection

10.1. Principle - Detection of H1N1 (Swine Flu) Virus by polymerase chain reaction (PCR) method.

10.2. Sample- Throat & nasal swab

10.3. Reagent- Sterile swab, Viral transport media (VTM)

10.4. Procedure- One throat swab & nasal swab by sterile swab should be taken & place in one VTM with universal precaution.

VTM is kept at 2 to 8°C & transported to virology lab by maintaining temperature with icepack in thermocol container or vaccine carrier.

Detail history sheet should be provided with sample.

11. Common Rapid Test:

1	Leptospirosis	RDK card method	Positive/Negative detected for Leptospirosis	IgM indicates recent infection of Leptospirosis; IgG indicates old infection of Leptospirosis
2	Dengue	RDK card method	Dengue NS1, IgG & IgM can be detected for Dengue	Dengue NS1 present in 1 st 5 days of infection, IgM indicates recent infection of Dengue, IgG indicates old infection of Dengue
3	HIV	RDK card method	HIV I & II can be detected	HIV I or II can be detected.
4	HBsAg	RDK card method	Positive/ Negative detected for Hepatitis B	Hepatitis B viral infection
5	Malaria	RDK card method	Pl Vivax & PL Falciparum can be detected	Pl. Vivax & Pl. Falciparum is detected
6	UPT	RDK card method	Positive / negative detected for pregnancy	Pregnancy can be detected
7	Widal Test	Rapid Agglutination Method	Positive /Negative to detect Enteric /Typhoid Fever	Enteric /Typhoid Fever can be detected
8	CRP Test	Rapid Agglutination Method	Positive/Negative to detect infection & septicemia	Detected in infection & septicemia
9	RA Test	Rapid Agglutination Method	Positive / Negative to detect Rheumatoid Arthritis	Detected in Rheumatoid Arthritis
10	VDRL	Rapid Agglutination Method	Positive/ Negative to detect Syphilis infection	Detected in Syphilis infection

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Further reading



4. BIOCHEMISTRY SECTION

Biochemistry tests usually done in laboratory include following tests. These tests are done with available standard biochemical kits on semi-automatic / fully automated auto-analyzer. Procedure, method and interpretation varies as per available kit.

- 1) Blood Sugar
- 2) Blood Urea/ Blood Urea Nitrogen
- 3) Serum Creatinine
- 4) Serum Bilirubin
- 5) SGOT

- 6) SGPT
- 7) Alkaline Phosphatase
- 8) Serum Total Proteins
- 9) Serum Albumin
- 10) Serum Na/K/Cl
- 11) Serum LDH
- 12) Serum Cholesterol
- 13) Serum HDL Cholesterol
- 14) Serum Triglycerides

Further reading

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5. HISTOPATHOLOGY

1. Definition:

It is a branch of pathology, which deals with the study of disease in a tissue section.

The tissue undergoes a series of steps before it reaches the examiners desk to be thoroughly examined microscopically to arrive at a particular diagnosis.

To achieve this, it is important that the tissue must be prepared in such a manner that it is sufficiently thick or thin to be examined microscopically and all the structures in a tissue may be differentiated.

The objective of the subsequent discussions will be to acquaint the staff with their responsibility; the basic details of tissue handling, processing and staining.

The term **histochemistry** means study of chemical nature of the tissue components by histological methods.

The cell is the single structural unit of all tissues. The study of cell is called cytology.

A tissue is a group of cells specialized and differentiated to perform a specialized function. Collection of different type of cells forms an organ.

2. Type of material obtained in laboratory:

The human tissue comes from the surgery and the autopsy room.

From surgery two types of tissue are obtained.

- i. As biopsy- A small piece of lesions or tumor which in sent for diagnosis before final removal of the lesion or the tumor (Incisional biopsy).
- ii. If the whole of the tumor or lesion is sent for examination and diagnosis by the surgeon, it is called excisional biopsy. Tissues from the autopsy are sent for the study of disease and its course, for the advancement of medicine.

3. Fixation 3.1. Definition:

It is a complex series of chemical events, which brings about changes in the various chemical constituents of cell like hardening, however the cell morphology and structural detail is preserved.

Unless a tissue is fixed soon after the removal from the body, it will undergo degenerative changes due to autolysis and putrefaction so that the morphology of the individual cell will be lost.

3.2. Principle of fixation:

The fixative brings about cross linking of proteins which produces denaturation or coagulation of proteins so that the semifluid state is converted into semisolid state; so that it maintains everything in vivo in relation to each other. Thus semisolid state facilitates easy manipulation of tissue.

3.3. Tissue Processing

The tissue processing is the heart of any tissue section, which will be cut adequately only if the tissue is properly preserved and processed. The study of this topic is to understand the coarse and fine details of tissue processing so that excellent sections are obtained.

4. Staining

The sections, as they are prepared, are colourless and different components cannot be appreciated. Staining them by different coloured dyes, having affinities of specific components of tissues makes identification and study of their morphology possible.

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Further reading



6. CYTOLOGY STUDY

Cytology is divided into four groups -

1) Fine needle aspiration cytology (FNAC)-

It is done for diagnosis of any lump/mass in body. With the help of 20 bore needle fine aspiration from any lump or mass in the body is done and smear made from aspirated material is fixed with bio fixative and stained with Hematoxylin and Eosin staining (H & E).

Results

Nucleus - blue Cytoplasm and background – pink

2) Exfoliative cytology –

It is done in the female patients to diagnose gynecological pathology. The cervical smears

are fixed by bio fixatives and are stained with PAP stain.

Results

Nucleus - blue Cytoplasm and background – pink

3) Body fluid cytology-

It is done on CSF / Pleural fluid / Ascitic fluid / Synovial fluid etc. All the body fluids are examined for physical / chemical examination. Cell count is done on Neubauer chamber. The smear of the deposited material is stained with Leishman stain, Gram stain and ZN stain.

4) Bronchial lavage cytology-

Smears are stained with H&E, Gram stain, and ZN stain.

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Further reading



7. BLOOD BANK

Blood Grouping (ABO & Rh system)

There are more than 300 blood group systems but ABO and Rhesus (Rh) systems are of importance from clinical point of view. These are inherited characters which give rise to antigen-antibody reactions.

1. ABO system:

It was discovered by Landsteiner in 1900. The red cells contain different types of antigens (Agglutinogen), while plasma contains antibodies (Agglutinins). In order to determine the blood group of a subject, the red cells are allowed to react with a sera containing known antibody (Agglutinin).

1.1. Practical aspects of ABO grouping:

- Routine ABO grouping must include both cell and serum grouping as each test serves as a check on the other.
- ABO grouping test should be done at room temperature. Testing at 37 °C weakens the reaction.
- Tubes, slides, reagents and microplates should be labelled properly.
- Serum should be added before adding cells and examine each tube after serum has been added.
- Tubes or slides should be clean and dry and disposable plastic tubes may be used.
- Microscope should be used to examine reactions that appear negative by naked eye.
- Results should be recorded immediately after observation.

1.2. Blood sample

- Clearly labelled samples of blood in plain tubes. No sign of haemolysis should be there.
- If serum has not completely separated or become clear, centrifuge the blood sample at 1000-3000 rpm for 3 minutes.
- Take one to two millilitres of serum in pipette and place into a relabelled tube for serum grouping and other tests.

1.3. Methods for ABO Grouping:

The two main methods of ABO grouping for a routine blood bank are:

- i. Slide method: The slide test is not recommended as it is not reliable. It is less sensitive than the tube test. The slide is prone to drying effect that may be wrongly interpreted as agglutination.
- ii. Tube method: The tube method is recommended because:
 - It allows longer incubation of antigen-antibody mixture without drying.
 - Tubes can be centrifuged which enhance antigen-antibody reactions.
 - Both cell and serum grouping can be performed.
 - Weaker antigens and antibodies can be detected.
 - Less reagents are required.
 - Results can be read comfortably as there is no drying.

2. Rh blood group system:

Rh blood group system was first reported Landsteiner and Weiner in 1940.

In contrast to ABO system, Rh antigens are present on red blood cells only and Rh antibodies develop only in response to a known stimulus. Rh factor is present in 85-95% of human beings.

2.1. Methods of Rh Grouping:

The two main methods of Rh grouping for a routine blood bank are:

- i. Slide method: The slide test is not recommended as it is not reliable. It is less sensitive than the tube test. The slide is prone to drying effect that may be wrongly interpreted as agglutination.
- ii. Tube method: The tube method is recommended because:
 - It allows longer incubation of antigen-antibody mixture without drying.
 - Tubes can be centrifuged which enhance antigen-antibody reactions.
 - Both cell and serum grouping can be performed.

- Weaker antigens and antibodies can be detected.
- Less reagents are required
- Results can be read comfortably as there is no drying.

2.2. Interpretation:

- i. Agglutination in the tube 'test' and in the tube 'positive control' and smooth suspension of cells in the tube 'negative control': This is interpreted as test cells Rh (D) positive.
- ii. A smooth suspension of red cells in tubes of 'test' and 'negative controls' and agglutination in the tube labelled as 'positive control': This is interpreted as test cells Rh (D) negative.
- iii. Blood Donor red cells found negative should be further tested by AHG test for weak D.
- iv. If in any test 'negative control' gives agglutination or 'positive control' does not give agglutination, the results are invalid.

3. Cross Match

- Major crossmatch: Recipient's serum is cross matched with donor red cells for IgM and IgG antibodies compatibility.
- Minor crossmatch: Donor' serum is cross matched with recipient's red cells for IgM and IgG antibodies compatibility. If donor's serum is tested negative for unexpected or irregular antibodies, minor crossmatch can be avoided.
- If incompatibility is not detected in cross matching, then it is likely that the donor blood transfused into patient will survive normally.
- Finding of incompatibility indicates that transfusion of such blood is potentially dangerous and further steps should be taken to identify the antibody.

Note: It is mandatory to test the blood for the following transfusion transmissible infections before it is taken on stock and issued to the patient.

- i. HIV antibody screening by ELISA
- ii. Hepatitis B surface antigen (HBsAg) by ELISA
- iii. Hepatitis C antibody by ELISA
- iv. Syphilis by RPR test
- v. Malaria by thick and thin blood smear or malaria antigen.

After the testing is complete, the blood bag is labelled and then taken on stock and used for crossmatch and issue to the patients.

4. Procedure of blood

TRANSFUSSION:

Important aspects:

The blood bag received in the ward for transfusion to the patient is to be maintained at room temperature for one hour, so that it will come to the body temperature.

Pre-transfusion medication to patient is done half-hour before blood transfusion.

The blood bag should not be warmed in hot water otherwise hemolysis may take place.

Do not transfuse the blood bag immediately after receiving because at that time the blood bag is at 4 to 6 degree centigrade. Transfusion of cold blood may get transfusion reaction.

Rate of blood transfusion should be such that it will take 4 hours to transfuse the bag. In emergency the clinician will take decision.

During transfusion the patient is monitored for body temperature/ pulse/blood pressure/respiratory rate and watch for transfusion reaction.

4.1. Blood transfusion reaction:

- 1) Fever
- 2) Pain
- 3) Jaundice
- 4) Allergic
- 5) Other

In case of blood transfusion reaction, BTO should be informed immediately after stopping blood transfusion and blood bag along with blood transfusion reaction card duly filled should also be returned to blood bank.

4.2. Complication of blood transfusion reactions-

- i. Haemolytic transfusion reactions which could be immediate or delayed.
- ii. Allergic reactions like 1) Fever 2) Anaphylactic reaction 3) Allergic reactions 4) Graft versus-host diseases
- iii. Circulatory overload leading to pulmonary congestion and acute Heart failure, thrombocytopenia & dilution of coagulation factors.
- iv. Transmission of infections e.g. Hepatitis B & C, malaria, HIV Infection, syphilis, infectious mononucleosis, toxoplasmosis.

In case of blood transfusion reaction, for carrying out investigations pre and post-blood transfusion patient's blood and urine sample is needed. Blood bag along with blood transfusion reaction card duly filled should also be returned to blood bank by the medical officer.

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Further reading

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8. NORMAL LAB VALUES

Hematology			
Hemoglobin	ँ: 13.5-17.5 g/dl		
	♀: 12.0-16.0 g/dl		
Hematocrit	<u>ङ</u> : 39-49%		
	♀ : 35-45%		
Red Blood Corpuscles			
	♀: 3.8-5.1 ×10 ⁸ /µl		
Platelet	$150-450 \times 10^{3}$ / µl		
White Blood Cells (differential below)	$4.5-11.0 \times 10^{3}/\mu$ l		
Neutrophils	57-67%		
Lymphocytes	22-33%		
Monocytes	3-7%		
Eosinophils	1-3%		
Basophils	0-1%		
Erythrocyte Sedimentation Rate (ESR)	♂": <15mm/hr		
	♀ : <20mm/hr		
Ferritin	ङ : 20-250 ng/ml		
	♀: 10-120 ng/ml		
Fibrinogen	150-350 mg/dl		
Haptoglobin	26-185 mg/dl		
Hb A1c	5.0-7.5%		
Mean Corpuscular Hemoglobin	26-34 pg		
Mean Corpuscular Hemoglobin Concentration	33-37%		
Mean Corpuscular Volume	80-100fl		
Prothrombin Time	10-14 sec		
Activated Partial Prothrombin Time	20-40 sec		
Reticulocytes	0.5-1.5%		
Total Iron Binding Capacity	250-400 μg/dl		
Transferrin	200-400 mg/dl		
Chemis	stry		
Sodium	135-145 mEq/l		
Potassium	3.5-5.3 mEq/l		

Chloride	95-105 mEq/l
Bicarbonate	22-29 mEq/l
Blood Urea Nitrogen	10-26 mg/dl
Creatinine	0.6-1.3 mg/dl
Glucose	70-115 mg/dl
Anion Gap	7-16 mEq/l
Osmolality	275-300 mOsm/kg
Calcium Total	8.5-10.5 mg/dl
Calcium Ionized	4.65-5.28 mg/dl
Magnesium	1.3-2.4 mEq/l
Phosphate	2.5-4.5 mg/dl
Alfa Fetoprotein	<10 ng/mol
Albumin	3.5-5.5 g/dl
IgA	70-312 mg/dl
IgG	640-1350 mg/dl
IgM	56-350 mg/dl
Lactate	0.5-1.3 mEq/l
Protein (Total)	6.0-8.0 g/dl
Uric Acid	ँ: 3.0-7.4 mg/dl
	♀: 2.1-6.3 mg/dl
Zinc	55-135 μg/dl
Liver/Par	icreas
SGOT/ALT	0-40 IU/1
Alkaline Phosphatase	ST: 38-126 U/I
	♀: 70-230 U/l
Ammonia	10-50 μmol/l
AST/SGPT	7-40 IU/I
Bilirubin (Total)	0.2-1.0 mg/dl
Bilirubin (Conjugated)	0-0.2 mg/dl
Lecithin dehydrogenase	90-190 U/I
Amylase	25-125 U/I
C Peptide	0.7-1.89 ng/ml
Lipase	10-140
	>60 years: 10-180

Lipids				
Total Cholesterol	<200 mg/dl			
Low density Lipoproteins	<130 mg/dl			
High density Lipoproteins	∽ ": >29 mg/dl			
	? :>35 mg/dl			
Triglycerides	♂: 40-160 mg/dl			
	? : 35-135 mg/dl			
Othe	er			
Phosphokinase	☞: 38-174 U/I			
	? : 26-140 U/l			
Phosphokinase MB	<5%			
Acid Phosphatase	<0.8 IU/ml			
B12	100-700 pg/ml			
CA-125	<35 U/ml			
Copper (Cu)				
coppor (ou)	$\frac{9}{2}$. 20 155 up (4)			
	+ : 80-155 μg /dl			
Folate	3-15 ng/ml			
Lead (Pb) $<10 \ \mu g \ /dl$				
Zine (Janized)				
Zinc (Ionized)	/0-150 μg/dl			
	Artorial	Vanous		
	7.35-7.45	1.32-1.42		
	35-45	41-51		
	80-100	25-40		
HCO3	21-27 24-28			
Oxygen Saturation	95-99%			
Urin				
Ph	5-9			
Mınımal Volume	0.5-1.0 mg/kg/hr			
Specific Gravity	1.015-1.030			
Osmolarity	600-1400 mOsm/kg			

Creatinine	∽": 14-26 mg/kg/day		
	? : 11-20 mg/kg/day		
Creatinine Clearance	♂": 100-150 ml/min		
	? : 90-140 ml/min		
	* Rough estimate only varies with BMI		
Urea Nitrogen	12-20 g/day		
Calcium (Ionized)	100-300 mg/day		
Potassium (Ionized)	25-125 mEq/day		
Sodium (Ionized)	40-220 mEq/day	40-220 mEq/day	
Phosphate	0.4-1.3 g/day		
Uric Acid	250-750 mg/day		
Albumin	10-100 mg/day		
Amylase	1-17 U/hr		
Glucose	<0.5 g/day		
Protein	10-100 mg/day		
CSF	7		
Pressure	60-180 mmH2O		
White Blood Cell	0-5 /ul		
Protein	15-45 mg/dl		
Glucose	40-80 mg/dl		
Synovial Fluid			
White Blood Cell	<200 /ul		
	Trauma, OA, SLE	<3,000/ul	
	Gout. RA	>4,000/ul	
	Septic	>60,000/ul	
Protein	<3.0 g/dl		
Glucose	>50 mg/dl		
Uric Acid	<8.0 mg/dl		
Endocr	rine		
Aldosterone	Supine: 3-10 ng/dl		
	Upright: 5-30	pright: 5-30	
Cortisol	0800h: 6-23 µg/dl		
	1600h: 3-15 μg/dl		
	2200h: ≤50% of 0800h value		
Gastrin	<100 pg/ml		

Growth Hormone (<60yr)	∽ : <2 ng/ml			
	♀ : <10 ng/ml			
Growth Hormone (>60yr)	∽ ": <10 ng/ml			
	♀ : <14 ng/ml			
Estrogen	Follicular: 60-200 pg/ml			
	Luteal: 160-400 pg/ml			
	Menopause: ≤130 pg/ml			
Follicle stimulating Hormone	Follicular: 1-9 mU/ml			
	Ovulation: 6-26 mU/ml			
	Luteal: 1-9 mU/ml			
	Menopause: 30-118 mU/ml			
Luteinizing Hormone	Follicular: 1-12 mU/ml			
	Mid-Cycle: 16-104 mU/ml			
	Luteal: 1-12 mU/ml			
	Menopause: 16-66 mU/ml			
Progesterone	Follicular: 0.15-0.7 ng/ml			
	Luteal: 2.0-25 ng/ml			
Prolactin	<20 ng/ml			
Para Thyroid Hormone	10-65 pg/ml			
Sperm	20-200 million/ml			
Testosterone	Free	52-280 pg/ml		
	Total	300-1000 ng/dl		
Т3	Uptake	0.82-1.18		
	Total	100-200 ng/dl		
T4	Total	5.0-12.0 µg/dl		
	Free	0.3-2.3 ng/dl		
Thyroid stimulating Hormone	<10 µU/ml			
	>60 years 🗢 : 2-7.3 µU/ml			
	>60 years ? : 2-16.8 µU/ml			
Toxic Levels				
Acetaminophen	>200 µg/ml			
Ethyl Alcohol (in a non-alcoholic patient)	Intoxicated	>100 mg/ml		
	Lethargic	>200 mg/ml		
	Coma	>300 mg/ml		

	Resp. Distress Death	>500 mg/ml
Ethylene Glycol	>20 mg/dl	
Lead	>100 µg/dl	
Salicylate	>300 µg/ml	

Normal of Cerebral Spinal Fluid					
ColorOpening Pressure (mmH2O)Glucose (mg/100ml)Cells (#mm3)					Protein (mg/100ml)
Adult	clear	70-180	45-80	0-5 WBCs	15-45
Newborn	clear	70-180	2/3 of serum	40-60 WBCs	20-120

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9. LABORATORY TESTS PERFORMED

Clinical Services Related to:	РНС	Rural / Sub-district hospital	District Hospitals and all 100 bedded hospitals
Viral infection	Blood smear	Blood smear	Blood smear, serology test
Bacterial Infection	Urine exam, blood smear, sputum examination, Gram stain, ZN stain	Urine exam, blood smear, Widal test, CSF exam, Gram stain, ZN stain	Urine exam, blood smear, Widal test, CSF exam, serology, Gram stain, ZN stain
Protozoal	Urine exam, Stool, Malarial parasite, Filarial Blood smear	Urine exam, Stool, Malarial parasite, Rapid malaria test, Filaria, Blood smear,	Urine exam, Stool, Filaria, Blood smear
STD	Urine exam, Blood smear	Urine exam, Blood smear, HIV& VDRL tests	Urine exam, Blood smear, HIV& VDRL tests, serology
Poisoning cases	Urine exam, Blood smear	Urine exam, Blood smear,	Plasma Cholinesterase, Sr. Creatinine in aluminum phosphide poisoning
Animal Bites	Urine exam, Blood smear	Urine exam, Blood smear	Urine exam, Blood smear
Environmental disorders	Urine exam	Urine exam, Blood smear, Sr Creatinine & urea	Urine exam, Blood smear, Sr Creatinine & urea
Gastro-intestinal Tract Disorder	Stool exam, Blood smear	Stool exam, Blood smear, Total Eosinophilic count	Stool exam, Blood smear Total Eosinophilic count
Respiratory disorder	Sputum Gram & AFB stain exam, Blood smear	Sputum Gram & AFB stain exam, Blood smear	Sputum Gram & AFB stain exam, Blood smear
Cardio-vascular Disorder	Urine exam, Blood smear	Urine exam, Blood smear, SGOT	Urine exam, Blood smear, Sr CMB, Troponin
Hematological Disorder	Hb %, Blood smear	Hb %, Blood smear, sickling tests, BT & CT	Hb %, Blood smear, sickling tests, RBC indices, BT, PT, APTT

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12. Dentistry

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1. DENTAL CARIES

Dental caries is a microbial disease of the calcified tissues of the teeth characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth.

1. Signs and Symptoms:

Grayish black discolouration of tooth in pit and fissure areas.

- Cavity formation and food lodgment.
- Sensitivity if caries reaches to the dentin.
- Pain and swelling in vicinity area if pulp is involved due to caries.
- Periapical abscess & draining sinus in advanced stages.



Figure 1.1. Dental Caries

2. Diagnosis:

2.1 Visual Examination:

For visual examination – teeth should be cleaned, dried with compressed air and illuminated under adequate light source and observe for cavitation, surface roughness, opacity of marginal ridge or occlusal area, discolouration.



Figure 1.2 IOPA X-Ray Showing Radiolucent Carious lesion

2.2Tactile Examination:

Includes determining roughness or softness of tooth surface/discontinuity of enamel with a sharp explorer. Discolouration of pits and fissures may be a universal finding in normal healthy adult teeth-may be mistaken for presence of caries.

2.3 Radiographic investigation:

Radiograph shows dental caries and its extent as radiolucent lesion due to demineralization of tooth structure.



Figure 1.3: Stages of Dental Caries

3. Treatment:

Avoid extreme hot and cold beverages.

Restoration of cavitated lesions if limited to enamel and dentin.

Endodontic therapy for pulp involvement due to caries.

Abscess drainage followed by endodontic therapy.

Tooth extraction may be performed in advanced cases when conservation of tooth structure is critical.

4. Prevention of Dental Caries

4.1 Diet modification / Dietary counseling

4.2 Maintenance of oral hygiene

- Daily removal of plaque by toothbrushing/flossing/rinsing is the best measure for preventing caries and periodontal disease.
- Proper brushing technique.
- Flossing should be done in the interdental areas to maintain the proximal surface clean.



Figure 1.4: Proper brushing technique

4.3 Antimicrobial Agents:

- a) Antimicrobial agents like Chlorhexidine, Fluoride and antibiotics available to help prevent caries.
- b) 0.12 0.2% Chlorhexidine mouth rinse prescribed for home use at bedtime as a 30-second rinse.

4.4 Fluorides:

To reduce caries risk primarily achieved by systemic and topical fluoridation.

Use of Fluoride:

- a) Systemic use: Systemic fluoridation can be achieved by community water fluoridation (1ppm) in the areas of low fluoride content (below 1ppm).
- b) Topical use:

Self-application: Daily use of fluoridated toothpastes and mouthwashes.

Professional application: application on tooth surfaces.

- i) Use of APF gel 1.23%.
- ii) Use of 2% Sodium Fluoride.

5. Pit & fissure sealants:

- Sealants mechanically fill pits and fissures with an acid resistant resin.
- Renders pits and fissures easier to clean by tooth brushing and mastication.

KEY MESSAGE:

- Avoid sticky and food with rich sugar content.
- Brush the teeth twice a day with proper brushing technique.
- Visit your doctor every six months for regular dental check up and get the treatment done at early stages.

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2. DISEASES OF DENTAL PULP AND PERIAPICAL TISSUES

Diseases of the dental pulp initiate dental pain of pulpal origin, which can be due to pulpitis, apical periodontitis and periapical abscess.

1. Pulpitis is an inflammation of pulp which may be acute or chronic.

1.1. Acute Reversible Pulpitis: -

Mild to moderate inflammatory conditions of pulp caused by noxious stimuli in which pulp is capable of returning to the uninflamed state following removal of the stimuli.

1.1.1 Clinical features:

- Sharp momentary pain on exposure to sweet or thermal stimuli (cold).
- It does not occur spontaneously and does not continue when the cause has been removed.
- Reacts normally to percussion, palpation and mobility and the periapical tissue is normal on radiograph.

1.1.2 Treatment:

- Removal of etiology and restoration of the tooth.
- Cap. Amoxicillin 250/500 mg 3 times a day for 3 to 5 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

1.2. Acute Irreversible Pulpitis: -

A persistent inflammatory condition of pulp symptomatic or asymptomatic caused by a noxious stimulus.

1.2.1 Clinical features:

- Sharp pain usually caused by thermal stimuli and can occur spontaneously.
- The pain persists for several minutes to hours, lingering after removal of the thermal stimulus.
- In early stages the pain is sharp shooting, piercing while in later stages pain is more severe and is generally described as boring, gnawing or throbbing and may be more intense.

• Inspection generally discloses a deep cavity extending to the pulp or decay under filling. The pulp may be already exposed.

1.2.2 Investigation:

- Radiograph may disclose an interproximal cavity, or may suggest involvement or exposure of pulp, caries under filling.
- Vitality Test.

1.2.3 Treatment:

- Removal of inflamed pulp tissues with endodontic therapy.
- Cap. Amoxicillin 250/500mg 3 times a day for 3 to 5 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

1.3. Chronic Pulpitis:

1.3.1 Clinical features:

- It occurs as the chronic type of pulpal disease from its onset.
- Dull pain is present which is more often intermittent and there is a delayed response in vitality testing.

1.3.2 Treatment:

• Endodontic treatment.

1.4. Chronic Hyperplastic Pulpitis (Pulp Polyp):

It is a productive pulp inflammation due to an extensive carious exposure of a young pulp.



Figure 2.1: Chronic hyperplastic pulpitis



Figure 2.2: Chronic hyperplastic pulpitis

1.4.1 Clinical features:

- Occurs generally in children and young adults.
- Involved teeth with large, open carious lesions.

1.4.2 Signs & Symptoms:

- Chronic hyperplastic pulpitis is symptomless, except during mastication, when pressure of the food bolus may cause discomfort.
- Pulp appears as a pinkish red globule of tissue protruding, from the pulp chamber.

1.4.3 Investigation:

Radiograph shows large open cavity with direct access to the pulp chamber.

1.4.4 Treatment:

- Elimination of the polypoid tissue followed by RCT, provided the tooth can be restored otherwise extraction is needed.
- Cap. Amoxicillin 250/500 mg 3 times a day for 3 to 5 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

2. Diseases of Periapical tissues:

2.1 Apical Periodontitis:

Apical Periodontitis is a painful inflammation of the periodontium as a result of trauma, irritation or infection around the root apex regardless of whether the pulp is vital or non- vital.

2.1.1 Clinical features:

There is pain on mastication and tooth is tender on percussion.

2.1.2 Investigation:

Radiographic examination may show a thickened periodontal ligament or a small area of rarefaction if a pulpless tooth is involved and it may show normal periradicular structures if a vital pulp is present.

2.1.3 Treatment:

If the inflammation is caused by occlusal trauma it should be relieved by selective grinding. If it is due to spread of pulpal infection RCT is indicated.

Cap. Amoxicillin 250/500 mg 3 times a day for 3 to 5 days.

Tab. Metronidazole 200/400 mg 1BD for 3 days. Tab. Diclofenac 50mg 1BD for 3 days.

2.2 Acute Alveolar Abscess:

Acute alveolar abscess is a localized collection of pus in the alveolar bone at the apex of a tooth following death of pulp with extension of the infection through the apical foramen into periradicular tissue.

2.2.1 Clinical features:

- The patient has severe throbbing pain with swelling of the overlying soft tissue.
- Pus may exude through tiny openings.

2.2.2 Treatment:

- Establishing drainage and completion of RCT after subsidence of symptom is done.
- Cap. Amoxicillin 250/500 mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400 mg 1BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

2.3 Chronic Apical Periodontitis: (Periapical granuloma):

Localized mass of chronic granulation tissue formed in response to infection.

2.3.1 Clinical features:

The involved tooth is non- vital and slightly tender to percussion. Patient may complaint about mild pain on biting. The patient feels tooth is slightly elongated in its socket.

2.3.2 Radiographic examination:

Periapical granuloma appears as a radiolucent area of variable size attached to root apex.

2.3.3 Treatment:

- Root canal treatment with or without subsequent apicoectomy.
- Cap. Amoxicillin 250/500mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400mg 1BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

2.4 Radicular Cyst:

It is slowly growing epithelial cyst at the apex of the tooth. Radicular cyst presupposes physical, chemical or bacterial injury resulting in death of pulp.

2.4.1 Clinical features:

The majority of cases are asymptomatic. The associated tooth is non-vital or shows deep carious lesion or a restoration which is seldom painful or even sensitive to percussion.

2.4.2 Investigation:

Roentgenographic features: - Radiolucent area surrounded by radio-opaque border attached to root apex.

2.4.3 Treatment: -

Involved tooth is extracted and periapical tissue is carefully curetted.

2.5 Osteomyelitis:

Inflammation of bone and its marrow contents.

2.5.1 Acute Suppurative Osteomyelitis:

It is a serious sequel of periapical infection that often results in a diffuse spread of infection throughout medullary space, with subsequent necrosis of variable amount of bone.

2.5.1.1. Clinical features:

- Severe pain, trismus and paraesthesia of lips in case of mandibular involvement and manifests an elevation of temperature with regional lymphadenopathy.
- The teeth in the area of involvement are loose.
- Pus exudes from gingival margin.
- In the maxilla the disease is well localized to the area of initial infection and in mandible bone involvement is more diffuse & widespread.
- Radiographically: Little evidence of its presence until the disease has developed for at least one to two weeks. At this time diffuse lytic changes in the bone, radiolucent areas begin to appear, ill-defined margins and have moth-eaten appearance.

2.5.1.2. Treatment:

General principles of management include

- Debridement
- Drainage
- Cap. Amoxicillin 250/500 mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400 mg 1BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

If sequestrum is small, it gradually exfoliates through the mucosa. If large, surgical removal may be necessary. Acute suppurative osteomyelitis may proceed to development of periostitis, soft tissue abscess or cellulitis.
2.5.2 Chronic Suppurative Osteomyelitis:

It may develop in inadequately treated acute osteomyelitis or may arise from dental infection without preceding acute stage.



Figure 2.3: Chronic Suppurative Osteomyelitis

2.5.2.1. Clinical features:

Similar to acute suppurative osteomyelitis except all signs and symptoms are milder. The suppuration may perforate the bone and overlying skin or mucosa to form fistulous tract and empty on surface.

2.5.3 Chronic Focal Sclerosing Osteomyelitis: A reaction to mild bacterial infection entering the

bone through a carious tooth in persons who have a high degree of tissue resistance and tissue reactivity. **2.5.3.1. Clinical features:**

- Most common in children and young adults.
- Commonly involve mandibular first molar.
- Mild pain associated with an infected pulp.

2.5.3.2. Investigation:

• Radiographic features show well circumscribed radiopaque mass of sclerotic bone surrounding and extending below the apex of carious tooth involving one or both roots. Intact lamina dura. Periodontal ligament space is widened.

2.5.3.2. Treatment:

- Extraction of carious tooth. The sclerotic bone constituting the osteomyelitis is not attached to the tooth and remains after the tooth is removed. Surgical removal of sclerotic lesion is not indicated unless symptomatic.
- Cap. Amoxicillin 250/500mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400mg 1BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

2.5.4 Chronic Diffuse Sclerosing Osteomyelitis:

It is a proliferative reaction of the bone to low- grade infection. Portal of entry for the infection is through diffuse periodontal disease.

2.5.4.1 Clinical features:

- Occur at any age but is most common in older person especially in edentulous areas.
- Vague pain, unpleasant taste and mild suppuration with the spontaneous formation of fistula when acute exacerbation of dormant chronic infections.

2.5.4.2 Investigation:

• Radiographic features: - Diffuse patchy, sclerosis of bone often described as cotton wool appearance. Radiopaque lesion may be extensive and is sometimes bilateral. The border between sclerosis and normal bone is indistinct.

2.5.4.3 Treatment:

- Conservative treatment of acute episodes by antibiotic administration. If the tooth is present in one of these sclerotic areas, then it must be extracted.
- Cap. Amoxicillin 250/500mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400mg 1BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

2.5.5 Chronic Osteomyelitis with Proliferative Periostitis:

There is focal gross thickening of periosteum with peripheral reactive bone formation resulting from mild irritation or infection.

2.5.5.1 Clinical features:

- Occurs exclusively in the mandible, in children and young adults and most cases occur in the bicuspid and molar region.
- The patient complains of toothache or pain in the jaw and a bony hard swelling on outer surface of jaw.

2.5.5.2 Radiographic examination:

- Intra oral periapical X- ray will reveal a carious tooth.
- Occlusal roentgenogram shows a focal overgrowth of bone on the outer surface of the cortex which may be described as duplication of cortical layer of bone. This mass of bone is smooth and well calcified and may itself show a thin but definite cortical layer.

2.5.5.3Treatment:

- Extraction of carious tooth.
- Cap. Amoxicillin 250/500mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400mg 1BD for 3days. Tab. Diclofenac 50mg 1BD for 3 days.

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3. PERIODONTAL DISEASES

Periodontium is the anatomical structure surrounding tooth which involves gingival periodontal ligament, cementum, alveolar bone.



Figure 3.1: Periodontal Diseases

1. Gingivitis

Inflammation of gingiva is called gingivitis.



Figure 3.2: Gingivitis

1.1 Clinical features:

1.1.1 Acute Gingivitis:

- Diffuse puffiness and softening of gingiva.
- Redness of gingiva, presence of exudates.
- Plaque usually present and calculus is often seen.
- Sloughing with grayish debris adhering to eroded surface.
- Vesicle formation.

1.1.2 Chronic Gingivitis:

- Soggy puffiness that pits on pressure, marked softness and friability with fragmentation on exploration with probe.
- Firm, leathery consistency.

1.2 Treatment:

Scaling and polishing



Figure 3.3: ANUG

2. Acute Gingival Infections:

2.1 Necrotizing Ulcerative Gingivitis:

It is microbial disease of gingiva characterized by death and sloughing of gingival tissue.

2.1.1 Clinical Features:

•

- Sudden onset sometime with respiratory tract infection.
- Lesions are punched out crater like depressions on crest of interdental papilla.
- Ulcers are covered with grey pseudomembranous slough.
- Spontaneous gingival hemorrhage and also bleeding on slightest provocation.
- Constant radiating, gnawing pain of gingiva.
- Metallic foul taste.
- Excessive amount of pasty salivary secretions.
- Local lymphadenopathy.
- In severe and acute cases high grade fever, loss of appetite, generalized lassitude, insomnia, constipation, headache, mental depression may be seen.
- Necrotizing ulcerative gingivitis is caused by fusiform bacilli and spirochetes.

2.1.2 Treatment of Acute Necrotizing Ulcerative Gingivitis (ANUG)

With moist cotton and lignocaine jelly gently remove pseudo membrane.

- Antibiotic and Analgesic Cap. Amoxicillin 500mg 8 hourly 10 days for patient sensitive to Amoxicillin.
- Cap. Erythromycin 500mg x 8 hourly.
- Tab. Diclofenac 50mg 1BD for five days
- Rinsing with 3% H202 + equal quantity of water 2-3 hourly and/or 0.12% Chlorohexidine mouth wash BD.
- After 2 days if pseudo membrane is absent and erythematous gingiva are present, evaluate for scaling if sensitivity permits.
- After 5 days discontinue Hydrogen Peroxide gargles but continue with Chlorhexidine gargles.
- Vitamin B complex 1 OD for ten days.

2.2 Pericoronitis:

Inflammation of the gingiva in relation to the crown of an incompletely erupted tooth is called Pericoronitis.

2.2.1 Clinical features:

- Partially erupted or impacted mandibular third molar is common site of Pericoronitis.
- Pericoronitis is exacerbated by trauma, or foreign body trapped under tissue flap.
- Trismus may or may not be present.
- Pericoronitis presents as a red swollen sometimes suppurating lesion with tenderness, and pain radiating towards ear, nose or floor of mouth.
- Fever and malaise may be present.
- Difficulty in swallowing may be present.
- Many a times infection may spread to different tissue spaces leading to tonsillar abscess formation, cellulitis or Ludwig's angina.

2.2.2 Treatment:

- Area should be anesthetized.
- Drainage is established by gently lifting the soft tissue operculum.
- Area should be irrigated by normal saline to remove debris.
- Cap. Amoxycillin 250/500 mg 3 times a day for 3 to 5 days.
- Tab. Diclofenac 50mg 1BD for 3 days.
- Advice warm saline gargles every 2 hours.
- Once swelling subsides, partially erupted tooth should be treated by excision of pericoronal flap or by extractions.

3. Gingival Enlargement:

Increase in size of gingiva is a common feature called as gingival enlargement or gingival hyperplasia or gingival hypertrophy.

3.1 Inflammatory Enlargements:

3.1.1 Acute Inflammatory enlargements:

3.1.1.1 Clinical Features:

- Gingival abscess, localized, painful, sudden onset, rapidly expanding.
- Usually on marginal gingival or interdental papilla.
- Appears as a red swelling with a smooth shiny surface.
- Purulent exudates may be present.
- Tooth sensitive to percussion.

3.1.2 Chronic inflammatory enlargement

3.1.2.1 Clinical Features

- Ballooning of marginal gingiva and interdental papilla or both.
- Progresses slowly and painlessly.
- Appears like pedunculated mass on gingival.
- Painful ulcerations may occur.
- Exposed gingival surface appears red edematous and shiny in mouth breathers.

3.1.3 Treatment

- Scaling and root planning.
- If the shrinkage of fibrotic tissue doesn't occur, then surgical therapy is the treatment of choice.



Figure 3.4: Drug Induced Gingival Enlargement

3.2 Drug Induced Gingival Enlargement

3.2.1 Clinical features

- Painless bead like enlargement.
- May enlarge to cover crown portions.
- Enlargement is generalized.
- Enlargement more in anterior teeth.
- Recurs after surgical excision.
- Patient gives history of taking medicines like anticonvulsants, immunosuppressant, and calcium channel blockers.

3.2.2 Treatment

- Possibility of discontinuing the drug or changing the drug should be assessed by consulting the physician.
- Plaque control helps to limit the enlargement and subsequent periodontal diseases.
- If required by assessing the periodontal status go for periodontal surgical procedures like gingivectomy or flap as indicated.

3.3 Gingival Enlargement Associated with Systemic Diseases:

3.3.1 Pregnancy Gingivitis

- Gingival enlargement may be marginal or generalized, single or multiple tumor like masses.
- It occurs due to increased progesterone and estrogen levels in pregnancy especially in third trimester.

3.3.1.1 Treatment of Pregnancy Gingivitis:

Removal of local irritants in early stages of pregnancy and emphasis should be given on preventing gingival diseases.

3.3.2 Gingival Enlargement in Vitamin C Deficiency (SCURVY):

- Acute vitamin C deficiency itself does not cause inflammation. But it does cause hemorrhage, collagen degeneration, oedema of gingival tissue.
- These changes are in response to plaque.
- Regular intake of vitamin C and maintenance of oral hygiene prevents scurvy.

4. Pyogenic Granuloma:

4.1 Clinical features:

- It is a tumor like gingival swelling that is considered as exaggerated conditioned response to minor trauma.
- It presents as a pedunculated spherical tumor like mass.
- It is red or purple.
- It can be firm or friable. Sometimes surface may be ulcerated and purulent.

4.2 Treatment:

Excision of mass and removal of local irritating factors.

5. Periodontal Abscess:

5.1 Clinical features:

- Mild to severe discomfort.
- Localized red, ovoid swelling.
- Periodontal pocket, exudation.
- Mobility.
- Tooth elevation in socket.
- Tenderness to percussion or biting
- Elevated temperature, regional lymphadenopathy.

• Radiograph shows periodontal angular bone loss and furcation radiolucency.

5.2 Treatment:

- Drainage through pocket retraction or incision.
- Scaling and root planning.
- Required periodontal surgery.
- Systemic antibiotics -
- Cap. Amoxicillin 250/500 mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400 mg 1BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.
- Tooth removal if required.

6. Periodontitis:

6.1 Chronic Periodontitis:

An infectious disease resulting in inflammation within the supporting tissues of the teeth, leading to progressive attachment loss and bone loss.

6.1.1 Clinical Features:

- Supragingival and subgingival accumulation with calculus formation.
- Gingival inflammation and pocket formation.
- Loss of periodontal attachment and alveolar bone.
- Occasional pus discharge and poor oral hygiene.
- Moderate swelling of gingiva with pale red to magenta colors.
- Blunt or rolled gingival margins.
- Bleeding on probing may be present.
- Sometimes tooth may be mobile.
- It may be localized or generalized.

6.2 Necrotizing Ulcerative Periodontitis:

Necrotizing ulcerative periodontitis is an extension of necrotizing ulcerative gingivitis. Historically it is described as Vincent's infection or trench mouth.

6.2.1 Clinical features

- Areas of ulceration and necrosis of interdental papilla with yellowish white soft layer of pseudo membrane is present.
- Lesions are painful.
- Bleed without provocation.
- Punched out lesions present.

- Associated with malodor, localized lymphadenopathy, fever and malaise.
- Destructive progression of disease includes destruction of periodontal attachment and bone loss.
- Deep interdental osseous craters are very typical with necrotizing ulcerative periodontitis.
- Tooth mobility with untimely loss of tooth is a common feature.

6.3Aggressive Periodontitis

6.3.1 Localized Aggressive Periodontitis: (Juvenile Periodontitis):

- Usually first molar or incisor is affected with interproximal bone loss.
- There is lack of inflammation despite loss of deep periodontal pocket.
- Rate of bone loss is four fold as compared to chronic periodontitis.
- Mobility of teeth increases fast.
- Sensitivity to root surfaces.
- Deep dull, radiating pain during mastication.
- Usually disease starts around beginning of puberty.
- Radiological vertical bone loss is seen.



Figure 3.5: Generalized Aggressive Periodontitis

6.3.2 Generalized Aggressive Periodontitis

- Affects individuals below the age of 30.
- Gives very poor response to antibiotics.
- Characterized by generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molar and incisor.
- Periods of advanced destruction is followed by stages of quiescence of variable length (weeks to months to years).

- Quantity of amount of plaque seems inconsistent with the amount of periodontal destruction.
- Deep pockets and suppuration.
- Systemic manifestation may be seen such as weight loss, mental depression and general malaise.

6.3.3 Treatment:

- Conventional periodontal therapies for aggressive periodontitis consist of patient education, oral hygiene improvement, scaling and root planing and recall maintenance.
- Resective periodontal surgery can be effective to reduce pocket depth.
- Anti-microbial therapy consists of Cap Tetracycline (500 mg 1BD for 14 days every 8 weeks).

7. Oral Malodor (HALITOSIS)

Breath odor is defined as subjective perception after smelling someone's breath. If unpleasant, the term breath mal odor, halitosis or bad breath can be applied.

7.1 Etiological factors:

- Deep caries with infection, candidial infections, Pericoronitis, periodontitis, oral ulcerations, xerostomia, tongue and tongue coating especially in fissured tongue and hairy tongue.
- Infections like pharyngitis, sinusitis, and chronic purulent tonsillitis, chronic bronchitis, bronchiectasis, bronchial carcinoma, gastric hernia, regurgitation, esophagitis, and intestinal gas production, chronic glomerular nephritis, diabetes.
- Trimethylaminuria hereditary metabolic disorder causes typical fishy odor.

7.2 Treatment:

The treatment of malodor is cause related.

• Mechanical reduction of plaque and microorganisms by cleaning dorsum of the tongue by tongue scrapper and interdental spaces by interdental brush and proper cleaning of oral cavity. If required professional periodontal treatment is to be done

- Reduction of oral microbial load by mouth rinsing with Chlorhexidine 0.12% mouthwash twice a day for 15 days.
- Masking the malodor with rinses and mouth sprays.

KEY MESSAGE

- Maintenance of proper oral hygiene by using correct teeth brushing technique and rinsing the mouth to avoid food lodgement.
- Local etiological factors like accumulation of dental plaque, calculus must be eliminated in the dental clinic.
- Rule out and manage systemic disorders leading to periodontal diseases.

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4. SPREAD OF ORAL INFECTION

Many of the fascial spaces of head and neck communicate either directly or indirectly with each other, thus infection in the oral cavity spread from one region to another through these spaces.

- The spaces that get first infected are called primary spaces.
- Spaces, which get infected later, are called as secondary spaces.

The fascial spaces in head and neck are the potential spaces between the various layers of the fascia normally filled with loose connective tissue and bounded by anatomical barrier usually bone, muscle and fascial layer.

Important space infections Canine Space: -

- Swelling of cheek and upper lip.
- Obliteration of nasolabial fold.
- Drooping of angle of the mouth.
- Edema of lower eyelid it indicates pointing of abscess below medial corner.

1.2 Buccal Space Abscess: -

- Space infection is dome shaped and periorbital edema develops due to impaired venous and lymphatic drainage.
- Swelling begins at the lower border of the mandible and extends upwards to the level of the zygomatic arch.
- Trismus is not usually present.

1.3 Submasseteric Space: -

1.3.1 Clinical features:

- Swelling over the masseter muscle i.e. the swelling is seen extending from lower border of the mandible to zygomatic arch and anteriorly to anterior border of the masseter and posteriorly to posterior border of the mandible.
- Pain over the region.
- Severe trismus is present.
- Tenderness over the angle of the mandible
- Pyrexia and malaise.

1.4 Pterygomandibular Space:

1.4.1 Clinical features:

- Severe trismus.
- Extreme radiating pain.
- No evident extraoral swelling.
- Dysphagia is present.
- Difficulty in breathing
- Midline of palate is displaced to unaffected side and uvula is swollen.
- Medial displacement of the lateral wall of the pharynx.
- Redness and edema of the area around the third molar.

1.4.2 Differential diagnosis:

The Pterygomandibular space abscess must be distinguished from peritonsillar abscess. In peritonsillar abscess there is no dental involvement and less trismus.

1.5 Submandibular Space:

1.5.1 Clinical features:

- Firm swelling in submandibular region, below the inferior border of mandible.
- Redness of overlying skin, dysphagia, moderate trismus.

1.6 Submental Space

1.6.1 Clinical features:

- Distinct, firm swelling in midline beneath the chin.
- Dysphagia.

1.7 Sub lingual Space

1.7.1 Clinical features:

- Swelling in the floor of the mouth.
- Dyspnea.
- Dysphagia.

1.8 Treatment:

All space infections need antibiotics & analgesics, incision & drainage depending upon severity.

- Cap. amoxicillin 250/500mg 3 times a day for 3 to 5 days.
- Tab. Metronidazole 200/400mg BD for 3 days.
- Tab. Diclofenac 50mg 1BD for 3 days.

2. Cellulitis

Cellulitis is a diffuse inflammation of soft tissues, tends to spread through tissue spaces and along fascial planes.

Cellulitis of face and neck most commonly results from dental infection either as sequel of apical abscess or osteomyelitis or following periodontal infection or pericoronitis.

2.1 Clinical features:

- Painful swelling of the soft tissues involved that are firm and brawny.
- Patient is moderately ill and has raised temperature and leukocytosis.
- If the facial cellulitis persists the infection frequently tends to become localized and a facial abscess may form. When this happens the suppurative material present seeks to point or discharge upon a free surface. If early treatment is instituted a resolution usually occurs without drainage through a break in the skin.

2.2 Treatment:

Administration of antibiotics and removal of the cause of infection.

- Cap. Amoxicillin + Clavulanic acid 375/625 mg 1BD for 5 days
- Tab. Diclofenac 50mg 1BD for 5 days.

To avoid the further spread of infection or solidification of abscess the patient should be advised not to massage the affected area with any medication.

3. Ludwig's Angina:

Massive, firm, brawny cellulitis, beginning usually in submaxillary space and secondarily involving the sublingual and submental space bilaterally.

3.1 Clinical feature:

• A rapidly developing board like swelling of the floor of mouth and subsequent elevation of the tongue.

- Swelling is firm painful and diffuse.
- Trismus, dysphagia.
- Difficulty in breathing.
- High fever, rapid pulse and fast respiration
- Tongue may be raised against palate.
- As the disease continues, the swelling involves the neck, and edema of the glottis may occur. This carries the serious risk of death by acute respiratory failure or septicemia.



Figure 4.1: Ludwigs Angina

3.2 Antibiotic therapy:

Usually IV antibiotics with proper dosage and frequency are necessary according to age and severity.

- Injection Ampicillin 250/500mg 6 hourly for3 days Injection Gentamycin 40/80mg BD for 3 days
- Injection Metronidazole 50 mg (100ml bottle IV) TID for 3 days
- Injection Diclofenac sodium 3CC for 3 days
- Injection Ranitidine 2ml BD for 3 days
- Magnesium Sulphate dressing till the swelling subsides
- I.V fluids to maintain the electrolyte balance.
- Refer to higher center for further management immediately.

KEY MESSAGE:

- Avoid hot fomentation or application of any counter irritants locally.
- Perform incision and drainage of abscess at

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5. ORAL MUCOSAL LESIONS

1. Chronic Cheek or Lip Biting

Superficial lesions produced by frequent and repeated rubbing, sucking or chewing movements that abrade the surface of a wide area of lip, buccal mucosa, lateral border of tongue without producing discrete ulceration.

1.1 Clinical feature:

- It can occur at any age, common sites are buccal mucosa at the level of occlusion, lip and lateral border of tongue.
- Clinically it appears as a linear or diffuse lesion irregular opaque white. In some cases, lacerated and reddened area usually with patch of partly detached surface epithelium.
- It is rough on palpation as area becomes thickened and scaled.

1.2 Treatment:

Recommendation to stop the habit, smoothening of sharp edges of teeth. Acrylic shields can be fabricated to cover the facial surface of teeth and there by restricting access to buccal and labial mucosa.



Traumatic Lesion involving buccal mucosa and lateral border of tongue Figure 5.1 Figure 5.2

2. Oral Candidiasis:

Candidiasis is the disease caused by infection with yeast like fungus Candida albicans. It can occur either solely confined to the oral mucosa or as a part of any of the several mucocutaneous candidiasis syndromes.

2.1 Oral Thrushes or

Pseudomembranous Oral

Candidiasis

Candidiasis is the disease caused by infection with yeast like fungus Candida albicans.

2.1.1 Symptoms:

Burning sensation and rapid onset of bad taste.

2.1.2. Clinical features-

Common sites are roof of the mouth, retromolar area and mucobuccal fold.

Clinically pearly white or bluish white plaques are present on oral mucosa. Patches are loosely adherent to oral mucosa. Mucosa adjacent to it appears red and moderately swollen. White patches are easily wiped out with wet gauze leaving a bright red raw bleeding surface. In some cases, it may present as a brightly erythematous mucosa with only scattered white patches.

2.1.3 Treatment:

2.1.3.1 Topical treatment

- Clotrimazole: One oral Troche (10 mg tablet) dissolves in the mouth 5 times daily.
- Nystatin oral pastille, 1 or 2 pastilles dissolves slowly in the mouth 5 times a day.
- Clotrimazole 1 % mouth paint topically 1 to 2 times/day till lesion subsides.
- Nystatin suspension 100,000 units (1 ml of suspension held in the mouth before swallowing).

2.1.3.2 Systemic treatment

- Nystatin -250 mg TID for 2 weeks.
- Supplemental therapy by Vit B12. Tab Vit B Complex 10D for 10 days.

2.2 Denture Stomatitis: -

Denture stomatitis is a common form of oral candidiasis that manifests as a diffuse inflammation of the denture bearing areas and often associated with angular cheilitis.

2.2.1 Treatment:

Troches containing Clotrimazole and Nystatin 4-5 times should be applied on the denture after meals. Antifungal therapy and cessation of denture wearing till elimination of mucosal inflammation. Rinsing the denture and applying antifungal cream before inserting the denture.

2.3 Angular Cheilitis:

Angular cheilitis is the term used for an infection involving the lip commissures. The majority of cases is candida associated and respond promptly to antifungal therapy. Other possible etiologic cofactors include reduced vertical dimension, nutritional deficiency (Iron deficiency anemia and Vitamin-B or Folic acid deficiency) and rarely Diabetes, neutropenia and AIDS. Treatment includes correction of primary cause.

3. Ulcerative lesions of oral cavity

Ulcerative lesions are a group of common oral mucosal lesions. The most common causes of these lesions are mechanical and reactive factors, infectious diseases and neoplasms as well as autoimmune and hematological disorders. The main clinical feature in all these conditions is an ulcer. Ulcer consists of margins, edges, floor and base. Site, size, shape of the ulcer should be examined.

3.1. Recurrent Aphthous Stomatitis (RAS)

RAS is a disorder characterized by recurring ulcers confined to the oral mucosa in patients with no other signs of disease. Precipitating factors are trauma, endocrine conditions, stress, allergy, immunological abnormalities and nutritional deficiency.



Figure-5.3: Aphthous Ulcer

3.1.1 Clinical features:

Sites: Occurs most commonly on buccal & labial mucosa, buccal & lingual sulci, tongue and soft palate. It begins with prodromal stage of tingling, burning or itching for 1 to 2 days before the ulcer occur. Lesions are round to oval in shape, symmetric, single or multiple. Superficial ulcers covered by grey membrane. It is surrounded by localized area of erythema.

a) Minor aphthae: Minor aphthae are less than 1 cm in diameter and heal completely in 7 to 10 days

b) Major aphthae: Size of the lesion is larger than 1 cm and may reach up to 5 cm in diameter. They interfere with speech and eating. The lesions heal slowly and leave scars.

c) Herpetiform ulcers: Multiple small ulcers often upto 100 in number. It is found on any intra oral mucosal surface. It begins as small pinhead size erosion that gradually enlarges and coalesces. Lesions are more painful. Patient gets relief immediately with 2% Tetracycline mouth wash.

3.1.2 Treatment: -

Medication prescribe treatment should relate to the severity of the disease.

- i. Anaesthetic cream topical protective emollient base in mild cases with two or three small lesions. Pain relief can be obtained with use of topical anaesthetic agent.
- ii. Topical Corticosteroid Triamcinolone Acetonide3 to 4 times daily. Nutritional supplements withTab. B Complex 1 BD for 10 days.
- Maintenance of oral hygiene: Chlorhexidine
 0.12 % mouth wash twice a day for 10 days to maintain oral hygiene.



Herpetic Gingivo stomatitis Figure 5.4 Figure 5.5

4. Viral Infection 4.1 Herpetic Gingivo-stomatitis:

Herpes simplex, an acute viral infectious disease caused by Herpes simplex virus.

4.1.1 Clinical features:

The disease occurring in children is frequently primary attack and is characterized by development of fever, irritability, headache, pain upon swallowing, regional lymphadenopathy. Within few days mouth becomes painful and the gingiva become intensely inflamed which appear erythematous and edematous. The lips, tongue, buccal mucosa, palate may be involved. Shortly yellow fluid filled vesicles develops. These rupture and form shallow, ragged extremely painful ulcers covered by an erythematous halo. They heal spontaneously with 7 to 14 days and leave no scar.

4.2 Recurrent or Secondary Herpetic Labialis and Stomatitis:

It is usually seen in adult patients and manifests itself clinically as an attenuated form of primary disease. The recurrent form of disease is often associated with trauma, fatigue, menstruation, pregnancy, upper respiratory tract infection, emotional upset etc.

4.2.1 Clinical feature:

Recurrent herpes simplex infection may occur at widely varying intervals, from nearly every month in some patients to only once a year or even less in others. Lesion may either at the site of primary inoculation or in adjacent area supplied by the involved ganglion. In may develop on lips (recurrent herpes labialis) or intra-orally. The lesions are preceded by burning or tingling sensation and a feeling of tautness, swelling or slight soreness at the location in which vesicle are small 1mm or less in diameter, tend to occur in cluster and coalesce to form large ulcer. These gray or gray-white vesicles rupture quickly leaving a small red erythematous halo.

4.2.2 Treatment:

- Acyclovir: Acyclovir cream for lips to be applied 4 hourly for 5 days. Tab Acyclovir 200 mg 5times /day for 3 to 5 days.
- ii. Chlorhexidine 0.12% mouthwash twice a day for 10 days
- iii. Avoid stress.

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6. PREMALIGNANT AND MALIGNANT LESIONS

1. Oral Leukoplakia: -

It is a whitish patch or plaque that cannot be characterized clinically or pathologically as any other diseases and which is not associated with any other physical or chemical causative agent except the use of tobacco.

1.1 Homogenous Leukoplakia:

It is also called as leukoplakia simplex; uniform raised white plaque formation, varying in size, with regular edges.

1.2 Nodular or speckled leukoplakia:

Also called as leukoplakia erosiva. It is a mixed red white lesion in which small keratotic nodules are scattered over an atrophic patch of oral mucosa. Nodules may be pinhead sized or even larger. It has got a high malignant potential.



Figure 6.1: Oral leukoplakia

1.3 Ulcerated Leukoplakia:

It is characterized by red area, which at times exhibit yellowish area of fibrin giving the appearance of ulceration white patches are present at the periphery of the lesions.

1.4 Verrucous Leukoplakia:

It is also called as 'leukoplakia verrucosa'. It is characterized by verrucous proliferation above the mucosal surface. These lesions demonstrate sharp and blunt projection.

1.5 Erythroleukoplakia:

In some lesions of leukoplakia red component is present. This intermixed lesion is called as erythroleukoplakia.

1.6 Treatment:

- Patient should be asked to cease tobacco related habits immediately.
- Removal of chronic irritant like sharp, broken teeth dissimilar metal restorations and other predisposing factors like syphilis, alcohol, etc should be controlled and eliminated.

1.6.1 Conservative treatment: -

- Vitamin A: It is given orally, parentally or topically. Therapeutic dose -75000-300000. IU for 3months.
- Antioxidant therapy: -β-Carotene supplementation can be beneficial for treatment of leukoplakia.
- Vitamin B: complex: It is given as supplement in cases of commissural and lingual lesions.

1.6.2 Surgical management: -

Conventional surgery, Cryo surgery, Fulguration (electro cautery and electro surgery), Laser treatment. Biopsy may be carried out if there is no change in leukoplakia after cessation of stimulus and may require conservative or surgical treatment.

2. Erythroplakia:

Erythroplakia is bright red velvety plaque or patch which cannot be characterized clinically or pathologically as being due to any other condition. "Erythroplakia" is premalignant lesion with frequency less than leukoplakia but more malignant potential.

2.1 Clinical features:

Erythroplakia occurs predominantly in older men, in the sixth and seventh decades of life. Erythroplakia is more commonly seen on the floor of the mouth, the ventral tongue, soft palate and tonsillar fauces. Although the etiology is uncertain, most cases are associated with heavy smoking. Erythroplakia is asymptomatic.

2.2 Diagnosis:

Red velvety patch with no sign of infection and inflammation.

2.3 Treatment:

Removal of cause: elimination of suspected irritant and observation for one to two weeks. Prompt biopsy if lesions persist. If biopsy shows dysplasia or carcinoma total excision is indicated.

3. Oral Submucous Fibrosis (OSMF):

Etiological factors are chilly and spicy food, betel nut, tobacco & lime, nutritional deficiency etc.

3.1 Clinical features:

- It affects both sexes, majority of patients are between 20 to 40 years of age.
- The most frequent location of OSMF is buccal mucosa and retromolar area. It also commonly involves palate, palatal fauces, uvula, tongue and labial mucosa.



Figure 6.3: Oral submucous fibrosis

- Sometimes floor of mouth and gingival.
- The onset of condition is insidious and is often of 2 to 5 years of duration.
- Initial symptom commonly seen as burning sensation of oral mucosa aggravated by spicy food followed by either hyper salivation or dryness of mouth. Vesiculation, ulceration, recurrent stomatitis.
- Late symptoms are trismus, difficulty in tongue protrusion, swallowing.
- The most common and earliest sign is blanching of mucosa.
- As disease progresses the mucosa becomes stiff and vertical fibrous band appears. These bands can be palpable easily.
- The mobility of soft palate is restricted. Uvula when involved is shrunken.
- Tongue becomes smooth and its mobility is limited.

3.2 Treatment:

The treatment of patients with OSMF depends on the degree of clinical involvement.

3.2.1 Restriction of habit/behavioural therapy -

The preventive measure should be in the form of stoppage of habit, through health education. Affected patients should be explained about the disease and its malignant potential. Improvement in clinical features like- gradual increase in interincisal opening has been observed in most of patients who discontinue the habit.

3.2.2 Medicinal therapy

Administration of Vitamin B complex and antioxidants as supportive therapy.

- Vitamin rich diet along with iron preparation is helpful to some extent.
- B-Complex preparation Intramuscular injection starts with small doses and continuing with larger doses (2ml ampule daily). The course of 5 injections is repeated after 7days.
- Steroids –

Local – Injection Hydrocortisone 1.5 ml (25 mg/ml) locally in the area of fibrosis twice a week for four weeks or more as per conditions Systemic – A therapy with Hydrocortisone 25mg tablet in doses of 100mg per day for 7 days is useful in relieving burning sensation without untoward effects.

Placental extract -2 ml biweekly or weekly for three to four weeks locally injected around fibrous bands.

3.2.3 Surgical treatment like Conventional surgery, Laser, Cryosurgery

3.2.4 Oral Physiotherapy –

This includes mouth opening and ballooning of mouth.

3.2.5 Diathermy 4. Oral Lichen Planus:

It is a chronic immunologic inflammatory mucocutaneous disorder that varies in appearance from keratotic (reticular or plaque like) to erythematous and ulcerative lichen planus.



Figure 6.4 Figure 6.5 Oral Lichen Planus

4.1 Clinical features:

It may occur in adulthood with age range for male 35- 44 years and for female 45-54 years. It has more predilections for females.

4.1.1 Sites: Common are buccal mucosa and to lesser extent tongue, lips, gingiva, floor of mouth and palate.

4.1.2 Symptoms: Patients may report with burning sensation of oral mucosa.

4.1.3 Clinical features: Oral lesion is characterized by radiating white and gray velvety thread like papules in a linear, annular or retiform arrangement forming typical lacy, reticular patterns, rings and streaks over the buccal mucosa to a lesser extent on lip, tongue and palate. Tiny white elevated dots are present at the intersection of white lines called as Wickham's straie. Types are reticular, papular, plaque, atrophic, bullous,

hypertrophic, annular and erosive.

4.2 Treatment:

- Small and moderately sized either erosive or ulcerative painful lesions can be treated with Triamcinolone Acetonide cream base. Systemic steroid Tab. Prednisolone (5 mg tab.) in tapering doses of 30 mg per day for the first of the three weeks, 15 mg per day for 2nd week and 5 mg per day for 3rd and final week.
- Symptomatic treatment with topical analgesic, topical anesthetic and antihistaminic rinse.
- Psychotherapy

5. Cyst of Jaws:

It may be epithelial or non-epithelial. Epithelial cyst may be odontogenic or non-odontogenic. Clinically presenting as smooth, hard, painless prominence. Dentigerous cysts are mainly associated with impacted molars and canines. Odontogenic keratocyst are seen in the lower third molar area extending into ramus. Radiographically cyst appears as well defined round or oval areas of radiolucency circumscribed by a sharp radiopaque margin.

5.1 Treatment:

- 1. Marsupialization.
- 2. Enucleation of cyst.

6. Tumors:

Presenting as intra oral swelling may be odontogenic or non-odontogenic variety, may be central or peripheral, epithelial or connective tissue origin. The most common tumor occurring in oral cavity is squamous cell carcinoma. Tobacco, alcohol, smoking are the predisposing factors. The patient may present with a swelling, ulcero-proliferative growth with involvement of lymph nodes. Pain may accompany if it involves the adjacent structures or superadded with infections. Radiographically it may show bone erosion.

6.1 Clinical features: -

6.1.1 Benign Tumors: -

- Encapsulated tumor
- Base of tumor is of two types sessile and pedunculated.
- Not involving the adjacent tissue.
- Painless and slowly growing.

6.1.2 Malignant Tumors: -

- Ulcero-proliferative lesion
- Invading into adjacent structures
- Pain, paraesthesia
- Trismus
- Bony erosion
- Mobility of teeth
- Not encapsulated
- Palpable Lymph nodes
- Pathological Fracture of the bone

6.1.3 Investigation:

Lateral oblique X-ray, OPG.

6.2 Common Cancers

6.2.1 Squamous Cell Carcinoma

A malignant epithelial neoplasm exhibiting squamous differentiation as characterized by formation of keratin and/or presence of intercellular bridges. The most important task is to establish an early diagnosis at the first stages of the disease. Risk factors include use of tobacco in its various forms, alcohol, ultraviolet light, chronic irritation to oral mucosa, human papilloma virus infection.



Figure-6.6: Carcinoma of buccal mucosa

6.2.1 Clinical features: -

6.2.1.1 Symptoms: -

- Ulcerative lesion in the oral cavity that does not heal.
- A persistent red or white patch
- Loosening of the teeth or pain around the teeth or jaw.
- Voice changes.

- **6.2.1.2 Site:** Tongue, floor of mouth, lower alveolus, buccal mucosa, upper alveolar / hard palate, retromolar area, lip, floor of mouth.
- **6.2.1.3 Signs:** Clinically almost all oral cancers, except those in earliest stages have two characterized features in the form of ulceration and indurated margin.

6.2.2 Diagnosis:

History and detail clinical examination, lymph node examination.

It is important for early detection to decrease mortality rate.

6.2.3 Prevention: -

- Oral cancers can be prevented by /sensible attitude towards maintenance of oral care and regular checkups.
- Proper education regarding the adverse effect of the dreaded habits.
- Successful persuasion to quit the bad habits (smoking, alcohol consumption, gutkha).
- The presence of persistent grayish white patches developing in people who smoke or chew tobacco needs to be investigated.
- Maintenance of good oral hygiene.
- Any faulty restoration or a sharp tooth needs to be corrected.

6.2.5 Treatment:

Treatment includes surgical intervention, radiation therapy and chemotherapy.

7. Tobacco Cessation Centers (TCC):

Setting up TCC facilities at district hospital level. At the district hospital Tobacco cessation centers are combined with dental units.

KEY MESSAGE:

Any discolouration or grayish white patch in the oral cavity must be properly evaluated and managed at early stage considering possibility of oral malignancies. Cessation of tobacco consumption and

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7. OROFACIAL PAIN

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage associated with oral lesions.

1. Trigeminal Neuralgia (TIC DOULOUREUX)

1.1 Clinical features:

- Age Middle and old age group
- Episodes of intense shooting stabbing, pain that lasts for a few seconds and then completely disappears.
- Quality of pain Electric shock like, unilateral along the course of the affected nerve.
- Maxillary branch is most commonly affected followed by the mandibular branch and (rarely) the ophthalmic branch.
- Involvement of more than one branch occurs in some cases.
- Trigger zones precipitated by light touch on a "trigger zone" present on the skin or mucosa within distribution of the involved nerve branch. Common sites for trigger zones include nasolabial fold and the corner of the lip
- Pain aggravated by: Shaving, showering, eating, speaking or even exposure to wind.
- Number of attacks varies from 1-2 per day to several per minute

1.2 Treatment:

Drug therapy

Carbamazepine: - Effective therapy for greater than 85% of newly diagnosed cases. Tab. Carbamazepine as an initial dose, 100 mg is given twice daily until relief is established.

Baclofen: - Patient who do not respond to Carbamazepine alone may obtain relief from Baclofen (5 to 10 mg 3 times a day) or by combining Carbamazepine with Baclofen.

Refer to higher centre for further management

2. Glossopharyngeal Neuralgia

(Ninth cranial nerve neuralgia)

2.1 Clinical features:

- Characterized by severe paroxysmal pain in the tonsils and ear.
- Less intense than trigeminal neuralgia.
- Similar clinical features as trigeminal neuralgia.
- Precipitating factors Yawning, talking, chewing and swallowing.
- Trigger zone: pain sensation following the distribution of glossopharyngeal nerve namely, the pharynx, posterior tongue, ear and intraarticular retromandibular area.

2.2 Treatment:

- Similar to trigeminal neuralgia.
- Good response to Carbamazepine and Baclofen.

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8. TRAUMATIC INJURIES TO TEETH

1. Classification:

Traumatic injuries are classified as

Class	Simple fracture of the crown, involving
Ι	little or no dentin.

- Class Extensive fracture of the crown, II involving considerable dentin but not dental pulp.
- Class Extensive fracture of the crown, III involving considerable dentin and exposing the dental pulp.
- Class Traumatized tooth that become non-IV vital, with or without a loss of crown structure.
- Class Teeth lost as a result of trauma. V
- Class Fracture of the root, with or without loss VI of the crown structure.
- Class Displacement of a tooth, without fracture VII of the crown or root.
- Class Fracture of crown en mass and its VIII replacement.
- Class Traumatic injuries to primary teeth IX



Figure-8.1: Crown fracture without pulp exposure

2. The various effect of trauma to teeth are

2.1 Crown Fracture:

If it involves only enamel and dentin (Uncomplicated fracture) aesthetic restoration of the teeth is sufficient.

2.1.1 Enamel dentin fracture:

Radiograph is mandatory to determine the full extent of the injury.

2.1.2 Fracture Crown without pulp exposure: Objective in treating a fractured crown without pulp exposure is three fold.

- Elimination of discomfort.
- Preservation of vital pulp.
- Restoration of fracture crown.

The tooth should be tested with electric pulp tester or with ice or ethyl chloride spray. If the pulp test is vital and tooth is comfortable it should be checked again after a week, 3 weeks, 3 months, 6 months and 1 year.

2.1.3 Fracture with pulp involvement:



Figure 8.2: Crown fracture with pulp exposure

If there is involvement of pulp (Complicated Fracture) vital pulp therapy can be done for which optimal time is first 24 hours.

- In traumatic exposure after 72 hours in immature teeth, full pulpotomy can be considered for apexogenesis. In mature teeth, pulpectomy should be done.
- In non-vital teeth with the open apex, apexification is indicated to create an apical stop.

2.1.3 Treatment

Crown with pulp exposure: Following treatments are possible:

- Pulpotomy (Pulp is vital).
- Endodontic treatment.

2.2 Root fracture:

In this coronal segment is reduced quickly and functional splint is placed for 2 to 4 weeks. When attended in time, pulp necrosis is remarkably low. It will occur only 25% of the time and in majority of cases, necrosis will occur only in the coronal segment and only that is root canal treated.

- If in the middle 1/3 of root: If tooth is restorable stabilize, RCT, Resect fracture portion, restoration.
- If tooth is not restorable or unable to stabilize, extraction is needed.

2.3 Displacement injuries:

Lateral Luxation and avulsion: Lateral Luxation, extrusion, and avulsion need emergency splinting & later RCT if required.



Figure 8.3: Splinting of traumatically displaced upper anterior teeth

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9. DISCOLOURATION OF TEETH

1. Introduction:

Discoloration of teeth is a cosmetic problem that is often the patient's primary concern. Although restorative procedures are available, discoloration can often be corrected totally or partially by a more conservative approach.

2. Etiology of Tooth Discolouration:

Extrinsic stains	Intrinsic stains	
 Diet related Bacterial strains Medications Tobacco related Habits Poor oral hygiene 	 Dental Fluorosis Tetracycline staining Amelogenesis imperfecta Erythroblastosis foetalis Porphyria Jaundice Pulpal necrosis Intrapulpal haemorrhage due to trauma Dentin hyper-calcification 	

3. Treatment:

Discolouration can be minimized by oral prophylaxis, bleaching techniques, composite resin, laminates.

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10. MAXILLO-FACIAL INJURIES

Any injury to either a soft tissue or hard tissue of face caused by an assault, road traffic accidents, sharp instruments, fall, sports injuries or violence can cause loss of soft tissue and hard tissue which has to be handled by an oral or dental surgeon meticulously at the time of casualty.

1. Soft tissue injuries:

Abrasion, contusion, ecchymosis, haematoma, avulsion or degloving injury

2. Hard tissue injuries:

Fracture: External force beyond the modulus of elasticity of the bone.

2.1. Simple fracture:

Closed linear fractures of bone.

2.2. Compound fracture:

Fractures which communicate to the exterior through skin or mucous membrane.

2.3. Greenstick fracture:

It occurs in immature bone where one surface is compressed and opposing surface is stretched leads to fracture.

2.4. Dentoalveolar fracture:

- Fracture of dentate alveolus may occur as a separate clinical entity or in conjugation with other
- Facial bone fractures are usually associated with injury to teeth like fracture, subluxation or avulsion.
- Fractures of maxillary tuberosity and antral floor are relatively common complications which occur during exodontia.

3. Le Fort fracture:



Figure 10.1. Le Fort fracture

4. Mandibular fractures

4.1 Classification:

Fractures of mandible are more common than the fracture of middle third.

- **4.1.1** Mandibular fractures are classified by the anatomic areas involved. These areas are as follows: symphysis, body, angle, ramus, condylar process, coronoid process and alveolar process.
- **4.1.2** Mandibular fractures are also classified into simple, compound, and comminuted.



Figure 10.2: Mandibular Fracture

4.1.3 An important classification of mandibular angle and body fractures relates to the direction of the fracture line and the effect of muscle action on the fracture fragments.

Angle fractures may be classified as

- i. Vertically favorable or unfavorable and
- ii. Horizontally favorable or unfavorable.

4.2. Diagnosis of mandibular fractures:

The patient's health history may reveal pre-existing systemic bone disease,

The type and direction of traumatic force can be extremely helpful in diagnosis. Fractures sustained in vehicular accidents are usually far different from those sustained in personal alterations.

4.3. Clinical Examination

The signs and symptoms of mandibular fractures are as follows.

- Change in occlusion
- Any change in occlusion is highly suggestive of mandibular fracture.
- Anesthesia, paresthesia, or dysesthesia of the lower Lip.
- Abnormal mandibular movements, limited opening and trismus, deviation on opening toward the side of a mandibular condylar fracture, Inability to close the jaw.
- Change in facial contour and mandibular arch form
- Lacerations, hematoma, and ecchymosis.
- The diagnostic sign of ecchymosis in the floor of the mouth indicates mandibular body or symphyseal fracture
- Loose teeth and crepitation on palpation.
- Dolor, Tumor, Rubor, and Color.
- Tenderness on palpation.

4.4 Radiology

- Panoramic radiograph
- Lateral oblique radiograph
- Posteroanterior radiograph
- Occlusal view
- Periapical view
- Reverse Towne's view
- Temporomandibular joint, including tomograms
- Computed tomography (CT) scan, CBCT



Figure 10.3: Orthopantomogram (OPG)



Figure 10.4: 3D Computed tomography (CBCT) scan

4.5. Management of mandibular fractures

4.5.1 Closed reduction basic methods

- Direct interdental wiring
- Indirect interdental wiring
- Continuous or multiple loop wiring
- Arch bars
- Gunning type splints

4.5.2 Open reduction with direct skeletal fixation:

Allows the bones to be directly manipulated through an incision so that the fractured ends meet, then they can be secured together either rigidly (with screws or plates and screws) or non-rigidly (with trans osseous wires).



Figure 10.5 Close Reduction

Figure 10.6 Open Reduction

4.6 Post-operative care:

- Soft diet and liquids
- Control of infection
- Oral hygiene maintenance by using mouth rinses.

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11. TEMPOROMANDIBULAR JOINT DISORDERS

1. Trismus:

Restriction of normal oral opening or inability to open the mouth fully. Trismus is also defined as a condition in which muscle spasm on contracture prevents opening of the mouth (due to infection or other condition which alter muscle).

1.1. Causes of trismus

1.1.1 Articular: -

- i. Ankylosis: Fibrous, Bony
- ii. Arthritis: Stills disease (children), Rheumatoid (adults)
- iii. Pyogenic arthritis
- iv. Osteoarthritis
- v. Psoriatic
- vi. Reiter Syndrome
- vii. Gout
- viii. Marie Strumpell disease
- ix. Congenital syphilis
- x. Fracture Condyle

1.1.2 Extra Articular

- i. Myositis Ossificans: following trauma to the masticatory muscles especially masseter.
- ii. Infection:-Orofacial infection around the TMJ area can bring about trismus or limitation of oral opening. Odontogenic infection like pericoronitis, Ludwig's angina, Submasseteric space Infection, Mumps, Tuberculosis osteomyelitis, Parotid infection
- iii. Oral Submucous fibrosis
- iv. Systemic causes: -Tetanus, Tetany.
- v. Trauma: -Fractures involving zygomatic arch, fracture of mandible also causes trismus, pain and tenderness or muscle spasm.
- vi. Malignancies of oral cavity: Either due to infiltration or due to pain
- vii. Osteomyelitis
- viii. Coronoid hyperplasia
- ix. Scarring of the temporalis
- x. Fibrosis of Pterygomandibular raphae due to cleft palate surgery.

1.2. Clinical examination:

Detailed clinical history. Inspection, Palpation, Auscultation in an around the temporomandibular joint and muscles of mastication.

1.3. Investigation:

OPG, CT scan, MRI

1.4. Management:

- i. Before starting treatment reassure the patient.
- ii. Anti-inflammatory drugs and analgesics.
- iii. Antibiotics in case of infection.
- iv. Space infection: Antibiotics, incision and drainage.
- v. Muscle relaxants and mouth gags followed by physiotherapy.
- vi. Tetanus- IM Immunoglobulin followed by antibiotics.
- vii. Tetany- IV Calcium Gluconate 10mg.
- viii. Splints (soft as well as anterior bite planes).
- ix. Fracture: Reduction and fixation either closed or open under LA or GA.
- x. Surgical intervention.

2. Dislocation, Subluxation, Hypermobility of Temporomandibular Joint (TMJ):

2.1 The dislocation can be unilateral or bilateral

Anterior mandibular dislocation

- i. Acute
- ii. Chronic recurrent (habitual) subluxation
- iii. Long standing

The term luxation is used for complete dislocation. Subluxation or hypermobility is a partial or incomplete dislocation.

2.2. Causes:

Extrinsic or Iatrogenic causes: -

- i. Blow on the chin while mouth is open
- ii. Injudicious use of mouth gags during general anaesthesia.
- iii. Excessive pressure on mandible during dental extraction.
- iv. Post traumatic

Intrinsic or Self -induced causes: -

- i. Excessive yawning,
- ii. Vomiting,
- iii. Opening mouth too wide for eating
- iv. Hysterical fits.

2.3 Clinical feature:

2.3.1 Unilateral acute dislocation.

- Difficulty in mastication and swallowing.
- Difficulty in speaking.
- Profuse drooling of saliva.
- Deviation of the chin toward contralateral side.
- Deviation produces a lateral cross and open bite on the contralateral side.
- The mouth is partly open and the affected condyle cannot be palpable.

2.3.2 Bilateral acute dislocation: -

- Pain, inability to close the mouth, tense masticatory muscles.
- Difficulty in speech.
- Profuse drooling of saliva
- Protruding chin
- Mandible is protruded and movements are restricted.
- Patients complain of pain in temporal region rather than the joint.
- Distinct hollowness can be felt in both preauricular regions.

4. Treatment:

- Reassuring the patient.
- Tranquilizer or sedative drugs.
- Pressure and massage of the area.
- Manipulation with or without anesthesia.



Figure 11.1:

Press his premolar teeth downwards at the same time press the underneath of his chin upwards and backwards

2.4.1 Manipulation:

Remains the same irrespective of type of anesthesia.

First of all, patient should be given assurance about procedure and asked to relax completely.

Operator has to stand in front of the patient; he has to grasp the mandible with both the hand one on each side.

The thumb of operator should be covered with gauze to prevent injury during manipulation. The thumbs are placed on occlusal surfaces of lower molars and finger tips are placed below the chin. Operator has to exert full body pressure on posterior teeth to depress the jaw and at same time the finger tips are placed below the chin to elevate it by giving upward pressure. The downward pressure overcome spasm of muscle, plus it brings the locked condylar head below the level of articular eminence and then backward pressure is given to push the entire mandible posteriorly.

After this reduction procedure, the mouth is closed and patient is asked to keep the mouth opening restricted.

Immobilization can be carried out by giving barrel bandage for 10 to 14 days' period. Patient is kept on semisolid diet. Anti-inflammatory analgesics prescribed for 3to 5 days.

Long standing acute dislocation which do not respond to above procedure can be reduced by administering General Anesthesia. If manual reduction fails than open surgical procedure as last resort.

3. Temporomandibular disorder (TMD):

Temporomandibular joint syndrome or temporomandibular disorder (TMD) is the most common cause of facial pain after toothache. TMD can be classified broadly as TMD secondary to myofascial pain and dysfunction (MPD) and TMD secondary to true articular disease.

MPD form is associated with pain without apparent destructive changes of the TMJ on x-ray. It is frequently associated with bruxism and daytime jaw clenching in a stressed and anxious person.

3.1 Etiology:

The etiology of TMD is multifactorial and

includes malocclusion, jaw clenching, bruxism, personality disorders, increased pain sensitivity and stress and anxiety.

3.2 Clinical features:

- i. Pain
- ii. Muscle tenderness
- iii. A clicking or popping noise in the temporomandibular joint.
- iv. Limitation of jaw motion unilaterally or bilaterally.

3.3 Differential Diagnosis:

Cluster headache, Migraine headache, Post herpetic Neuralgia, Temporal or Giant Cell Arteritis, Ttrigeminal Neuralgia.

3.4 Treatment:

Counselling regarding self-care and reversal of parafunctional habits.

• Heat application to increase local circulation which acts as a sedatives and lowers muscle tension to be given for 15 to 20 minutes 4 times a day.

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- Cryotherapy with Ice packs application to the painful area 4 times a day for 20 minutes for relief of pain followed by an acute injury superimposed over a chronic TMD.
- Mild analgesic Tab. Diclofenac 50 mg 1 BD.
- Anti-anxiety agents for short duration in acute pain like. Tab Diazepam 2 to 5 mg a bed time for 10 days.
- Physiotherapy and active stretch exercise.
- Intraoral appliances: Use of Splints, orthopedic appliances, bite guards, night guards or bruxism guards.
- Stress management

12. DENTAL IMPACTION

1. Introduction:

Impaction is cessation of eruption of tooth caused by physical barrier or ectopic positioning of tooth.

An impacted tooth is one that is erupted, partially erupted or unerupted and will not eventually assume normal arch relationship with other teeth and tissue commonly seen with third molar and sometimes maxillary canine teeth.



Figure 12.1: Pericoronitis



Impacted Wisdom Teeth

Figure 12.2: Impacted Wisdom Teeth

2. Types of Impaction:

- Soft tissue impaction.
- Bony impaction.

3. Third molar impaction:

This can be classified by angulation in relation to long axis of second molar

- Mesioangular
- Distoangular
- Horizontal

- Vertical
- Buccoangular
- Linguoangular

4. Clinical Features:

- Difficulty in opening the mouth and deglutition.
- Localized tenderness and swelling.
- Radiating pain towards ear and sometimes headache.

• Purulent discharge may be present from the site of infection.

5. Investigations:

IOPA X-Ray, lateral oblique x-ray, OPG, Complete haemogram

6. Treatment:

- Advice antibiotics and analgesics. Cap. Amoxicillin 500mg 1 BD for 5 days. Tab. Diclofenac 50mg 1 BD for 5 days.
- Surgical removal of impacted tooth.

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