KALAM BOOKS

Volume 3

# APPENDIX
## *for* PGMEE

FIRST EDITION



Medicine ❖ Surgery ❖ Obstetrics and Gynaecology
Pediatrics ❖ Orthopedics

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**Vaibhav Bharat | Aditi Bharat | Ishad Aggarwal**

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# APPENDIX
## for PGMEE

Volume 3

**First Edition**

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# Preface

First of all it is our pleasure and duty to thank all our readers, who have time and again shown faith in our endeavours. It is always encouraging if your work is appreciated and we are grateful to all our readers. We started our Journey in 2011 with DNB CET Review which was an instant success and is our legendary creation till date. The collections of tables in the form of APPENDIX, at the end of the book were much appreciated and is in high demand even today. Hence we decided to recreate the magic of APPENDIX again, this time on a juggernautic scale and precision.

With changing pattern of PGMEE we have included colour pictures in our APPENDIX and made it a totally coloured book in three easy to carry volumes. We have done our level best to come up with up-to-date material, but to err is human, and we are humans too. However we constantly keep in touch with our readers through our website www.medeasyindia.com, and our Facebook fan page **https://www.facebook.com/MedEasyindia/** to keep them updated with any correction, change or improvement in our book.

We heartily invite any suggestions, corrections or discussions of PG Medical entrance material and MCQs on our mail id **info@medeasyindia.com**

Thanks
Authors/ Editors
APPENDIX FOR PGMEE
By Team MedE@sy

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## Appendix Medicine

## Appendix Surgery

## Appendix Obstetrics and Gynecology

**Appendix Pediatrics**

**Appendix Orthopedics**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Sickle-β thalassemia | Sβ | FS | FS | 0 | >3.5 | 2 to 15 | 80 to 92 | 0 |
| Sickle-β⁺thalassemia | Sβ⁺ | FSA or FS | FSA | 5 to 30 | >3.5 | 2 to 10 | 65 to 90 | 0 |
| HbSC disease | SC | FSC | FSC | 0 | <3.5 | 1 to 5; may be higher in rare cases | 45 to 50 | 45 to 50 |

HbA: adult hemoglobin; HbF: fetal hemoglobin; δβ: delta-beta; HPLC: high-performance liquid chromatography

**APPENDIX 50: HUMAN PORPHYRIAS: MAJOR CLINICAL AND LABORATORY FEATURES**

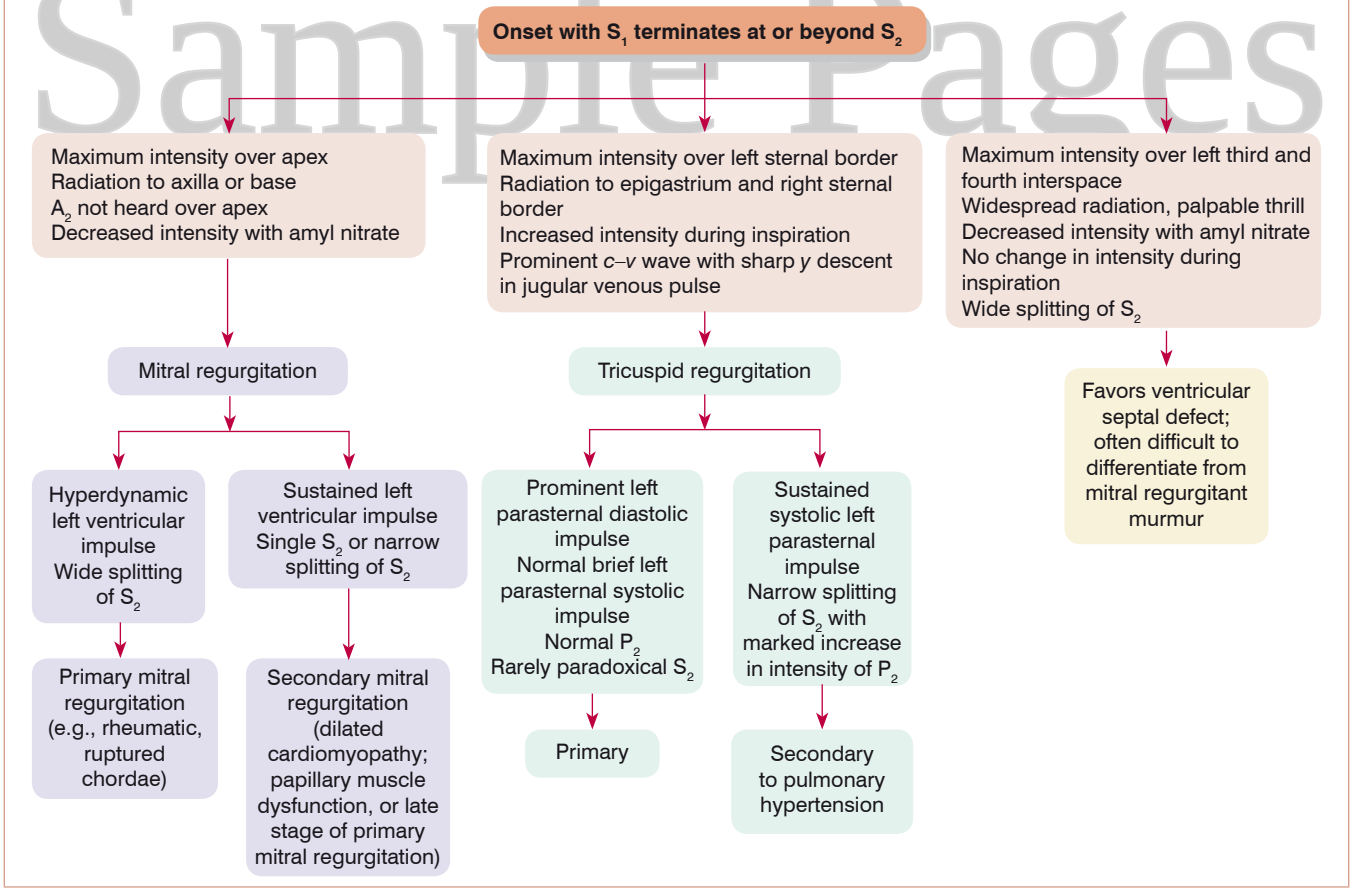
| | | | Principal Symptoms | | Increased Porphyrin Precursors and/or Porphyrins | | |
|---|---|---|---|---|---|---|---|
| **Porphyria** | **Deficient Enzyme** | **Inheritance** | **NV or CP+** | **Enzyme Activity % of Normal** | **Erythrocytes** | **Urine** | **Stool** |
| **Hepatic Porphyria** | | | | | | | |
| 5-ALA dehydratase-deficient Porphyria (ADP) | ALA-dehydratase | AR | NV | 5 | Zn-Protoporphyrin | ALA, Coproporphyrin III | — |
| Acute intermittent Porphyria (AIP) | HMB-synthase | AD | NV | 50 | — | ALAa, PBG, Uroporphyrin | — |
| Porphyria cutanea tarda (PCT) | URO-decarboxylase | AD | CP | 20 | — | Uroporphyrin, 7-carboxylate porphyrin | Isocoproporphyrin |
| Hereditary coproporphyria (HCP) | COPRO-oxidase | AD | NV & CP | 50 | — | ALA, PBG, Coproporphyrin III | Coproporphyrin III |
| Variegate Porphyria(VP) | PROTO-oxidase | AD | NV & CP | 50 | — | ALA, PBG, Coproporphyrin III | Coproporphyrin III Protoporphyrin |
| **Erythropoietic Porphyria** | | | | | | | |
| Congenital erythropoietic Porphyria (CEP) | URO-synthase | AR | CP | 1–5 | Uroporphyrin I Coproporphyrin I | Uroporphyrin Ib Coproporphyrin I | Coproporphyrin I |
| Erythropoietic protoporphyria (EPP) | Ferrochelatase | AR[a] | CP | 20–30 | Protoporphyrin | — | Protoporphyrin |
| X-linked protoporphyria (XLP) | ALA-synthase 2 | XL | CP | >100 | Protoporphyrin | — | Protoporphyrin |

HMB synthase; also known as PBG deaminase
**Abbreviations:** AD, autosomal dominant; ALA, 5-aminolevulinic acid; AR, autosomal recessive; COPRO I, coproporphyrin I; COPRO III, coproporphyirn III; CP, cutaneous photosensitivity; ISOCOPRO, isocoproporphyrin; + Nv, neurovisceral; PBG, porphobilinogen; PROTO, protoporphyrin IX; URO I, uroporphyrin I; URO III, uroporphyrin III; XL, X-linked.

## APPENDIX 60: HOLOSYSTOLIC (PANSYSTOLIC) MURMUR

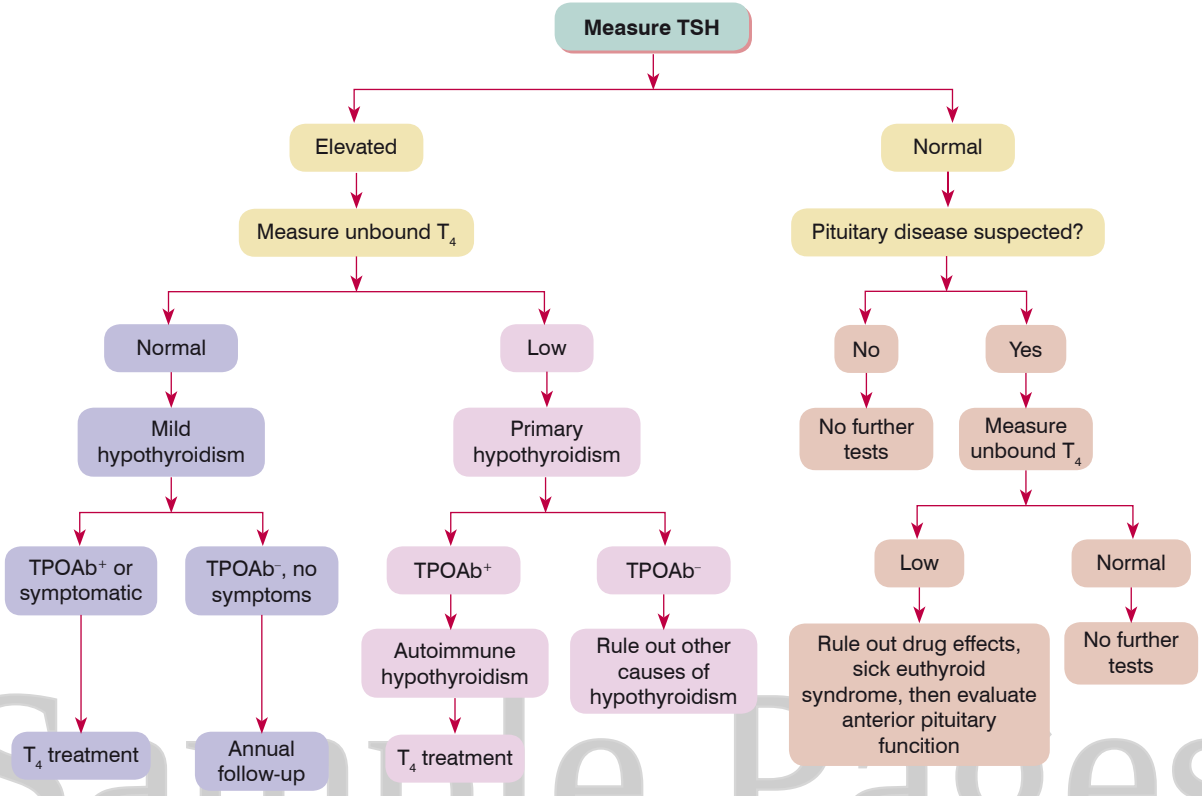
| Condition | Description |
|---|---|
| Tricuspid insufficiency | Can be best heard over the 4th left sternal border. The intensity can be accentuated following inspiration (**Carvallo's sign**) due to increased regurgitant flow in right ventricular volume. TR is most often secondary to pulmonary hypertension. Primary tricuspid regurgitation is less common and can be due to bacterial endocarditis following IV drug use, Ebstein's anomaly, carcinoid disease, or prior right ventricular infarction. |
| Mitral regurgitation | The $S_1$ is generally absent, soft, or buried in the **holosystolic murmur** of chronic MR. In patients with severe MR, the aortic valve may close prematurely, resulting in wide but physiologic **splitting of $S_2$.** A low-pitched $S_3$ occurring 0.12–0.17 s after the aortic valve closure sound. It may be followed by a short, rumbling, mid-diastolic murmur, even in the absence of MS. S4 is often audible in patients with *acute* severe MR who are in sinus rhythm. A presystolic murmur is not ordinarily heard with isolated MR. A systolic murmur of at least grade III/VI intensity is the most characteristic auscultatory finding in chronic severe MR. It is usually holosystolic, but as previously noted it is decrescendo and ceases in mid to late systole in patients with acute severe MR. The systolic murmur of chronic MR not due to MVP is intensified by isometric exercise (handgrip) but is reduced during the strain phase of the Valsalva maneuver. |
| Ventricular septal defect | No intensification upon inspiration. Flow during systole occurs from the L to R ventricle, producing the holosystolic murmur. It can be best heard over the left 3rd & 4th intercostal spaces and along the sternal border. It is associated with normal pulmonary artery pressure and thus S2 is normal. This fact can be used to distinguish from pulmonary stenosis, which has a wide splitting S2. When the shunt becomes reversed ("Eisenmenger syndrome"), the murmur may be absent and S2 can become markedly accentuated and single. |

### HOLOSYSTOLIC MURMUR DIFFERENTIAL DIAGOSIS



## APPENDIX 83: HYPOTHYROIDISM

### EVALUATION OF HYPOTHYROIDISM






**Signs and Symptoms of Hypothyroidism (Descending Order of Frequency)**

| Symptoms | Signs |
|---|---|
| Tiredness, weakness | Dry coarse skin; cool peripheral extremities |
| Dry skin | Puffy face, hands, and feet (myxedema) |
| Feeling cold | Diffuse alopecia |
| Hair loss | Bradycardia |
| Difficulty concentrating and poor memory | Peripheral edema |
| Constipation | Delayed tendon reflex relaxation |
| Weight gain with poor appetite | Carpal tunnel syndrome |
| Dyspnea | Serous cavity effusions |
| Hoarse voice | |
| Menorrhagia (later oligomenorrhea or amenorrhea) | |
| Paresthesia | |
| Impaired hearing | |

## APPENDIX 84: CRITERIA FOR THE DIAGNOSIS OF DIABETES MELLITUS

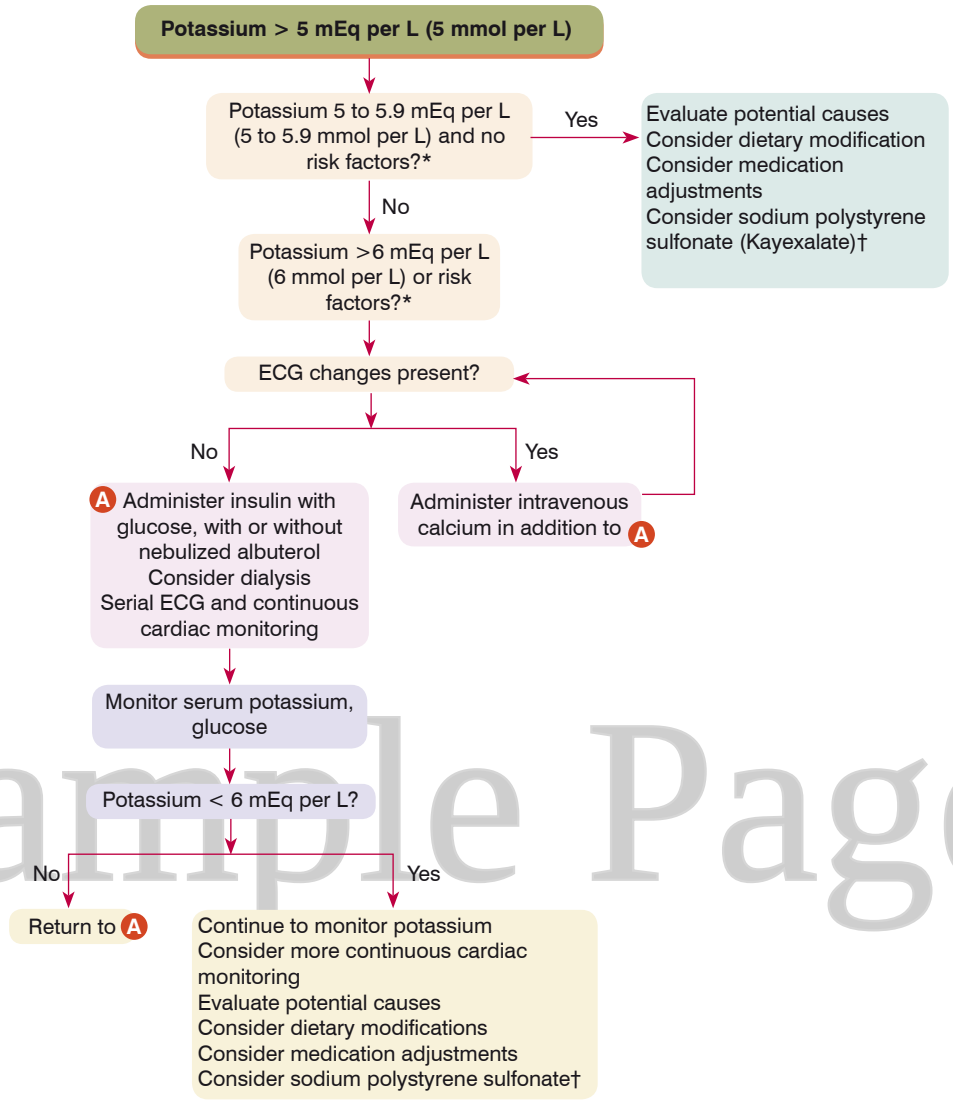
- Symptoms of diabetes plus random blood glucose concentration ≥11.1 mmol/L (200 mg/dL)[a] or
- Fasting plasma glucose ≥7.0 mmol/L (126 mg/dL)[b] or
- Hemoglobin A1c ≥ 6.5%[c] or
- 2-h plasma glucose ≥11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[d]

| APPENDIX 95: MEN SYNDROME | | | |
|---|---|---|---|
| **Feature** | **MEN 1** | **MEN 2A** | **MEN 2B** |
| Eponym | Wermer syndrome | Sipple syndrome | Williams-Pollock syndrome, Gorlin-Vickers syndrome, Wagenmann–Froboese syndrome, MEN 3 |
| Gene | MEN 1 gene encodes for **Menin**, found on the long arm of chromosome **11q13**, mutation in 70-90% | RET, Receptor tyrosine kinase, 10q11.2 | RET, Receptor tyrosine kinase, 10q11.2 |
| Entero-Pancreatic tumors | Total 40-80% Gastrinoma (25%), Insulinoma (20%), VIPoma, Glucagonoma, PPoma | - | - |
| Pituitary adenoma | 30-60% | - | - |
| Parathyroid hyperplasia | 80-90% | 10-25% | - |
| Medullary thyroid carcinoma | - | 90% | > 90% |
| Pheochromocytoma | - | >50% | > 50% |
| **Associated abnormalities** Mucosal neuromas Marfanoid habitus Medullated corneal nerve fibers Megacolon | - | - | 40-50% |
| Gene(s) | MEN1 | RET | RET |
| Approx. prevalence | 1 in 35,000 (1 in 20,000 to 1 in 40,000) | 1 in 40,000 | 1 in 40,000 |

**Footnote:**
- Primary hyperparathyroidism is the most common manifestation of MEN1
- Hyperparathyroidism is the earliest manifestation of the syndrome in most MEN1 patients.
- Enteropancreatic tumors are the second most common manifestation of MEN1
- Gastrinomas are the most common enteropancreatic tumors observed in MEN1 patients
- Insulinomas are the second most common enteropancreatic tumors in patients who suffer from MEN1. Unlike gastrinomas, most insulinomas originate in the pancreas bed, becoming the most common pancreatic tumor in MEN1.

| APPENDIX 96: SIGNS AND SYMPTOMS OF ADRENAL HORMONE DEFICIENCIES | | |
|---|---|---|
| **Glucocorticoid Deficiency** | **Mineralocorticoid Deficiency** | **Adrenal Androgen Deficiency** |
| - Fatigue, lack of energy<br>- Weight loss, Anorexia<br>- Myalgia, Joint pain<br>- Fever<br>- Normochromic anemia, lymphocytosis, eoisnophilia<br>- Slightly increased TSH<br>- Hypoglycemia<br>- Low blood presure, postural hypotension<br>- Hyponatremia (due to loss of feedback inhibition of AVP release | - Abdominal pain, nausea, vomiting<br>- Dizziness, postural hypotension<br>- Salt craving<br>- Low blood pressure, postural hypotension<br>- Increased serum creatinine<br>- Hyponatremia<br>- Hyperkalemia | - Lack of energy<br>- Dry and itchy skin (in women)<br>- Loss of libido<br>- Loss of axillary and pubic hair (in women) |

**Management of Hypokalemia**




*—Symptoms of hyperkalemia, rapid-onset hyperkalemia, or underlying heart disease, cirrhosis, or kidney disease.
†—Avoid in patients with or at risk of developing abnormal bowel function.

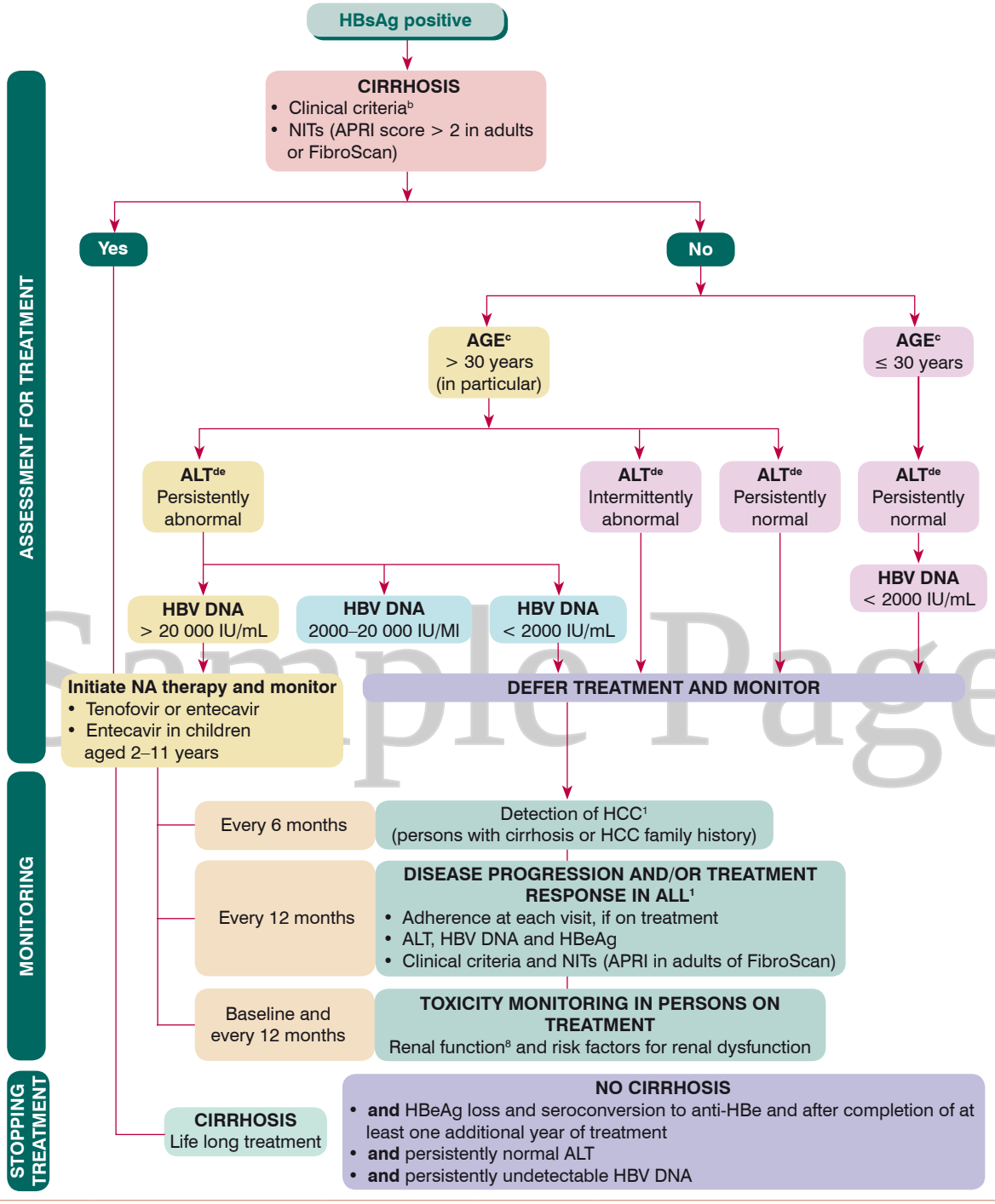


## APPENDIX 103: HYPERKALEMIA

**Causes of Hyperkalemia**

| |
|---|
| **Spurious** |
| Leakage from erythrocytes when separation of serum from clot is delayed (plasma $K^+$ normal) |
| Marked thrombocytosis or leukocytosis with release of intracellular $K^+$ (plasma $K^+$ normal) |
| Repeated fist clenching during phlebotomy, with release of $K^+$ from forearm muscles |
| Specimen drawn from arm with intravenous $K^+$ infusion |
| **Decreased excretion** |
| Kidney disease, acute and chronic |

## ALGORITHM OF WHO RECOMMENDATIONS ON THE MANAGEMENT OF PERSONS WITH CHRONIC HEPATITIS B INFECTION



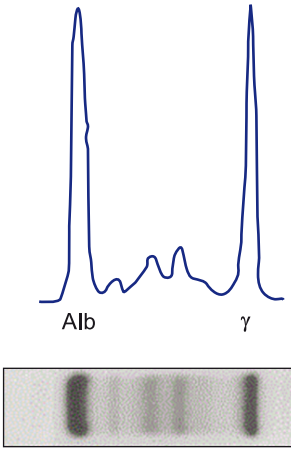
## APPENDIX 120: MULTIPLE MYELOMA

Multiple myeloma (MM) is characterized by the neoplastic proliferation of plasma cells producing a monoclonal immunoglobulin.

| | |
|---|---|
| Epidemiology | ◆ Found in all race, all geographical locations<br>◆ Men > Women (slightly)<br>◆ Blacks have nearly twice the incidence of whites<br>◆ Disease of old adults (Mean age 70 years) |
| Clinical presentations | 1. Bone pain with lytic lesions discovered on x rays or other imaging<br>2. An ↑ total serum protein concentration and/or the presence of a monoclonal protein in the urine or serum<br>3. Systemic signs or symptoms such as anemia, suggestive of malignancy<br>4. Hypercalcemia (symptomatic or incidental)<br>5. Acute renal failure with a bland urinalysis or rarely the nephrotic syndrome due to concurrent immunoglobulin light chain (AL) amyloidosis |
| Classical triad | Marrow plasmacytosis (>10%), lytic bone lesions, and a serum and/or urine M component. |

| Signs and symptoms | **Common** | **Rare** |
|---|---|---|
| | 1. Anemia – 80% (Most common sign)<br>2. Bone pain – 70% (Most common symptom)<br>3. Elevated creatinine – 48%<br>4. Fatigue/generalized weakness – 32%<br>5. Hypercalcemia – 28%<br>6. Weight loss – 24% | 1. Paresthesias -5%<br>2. Hepatomegaly - 4%<br>3. Splenomegaly - 1%<br>4. Lymphadenopathy - 1%<br>5. Fever - 0.7% |

| | |
|---|---|
| International Myeloma working group diagnostic criteria of Multiple Myeloma | **Clonal bone marrow plasma cells ≥10% or biopsy-proven bony or extramedullary plasmacytoma and any one or more of the following myeloma-defining events:** |
| | ◆ Evidence of end-organ damage that can be attributed to the underlying plasma cell proliferative disorder, specifically:<br>1. **Hypercalcemia:** serum calcium >0.25 mmol/L (>1 mg/dL) higher than the upper limit of normal or >2.75 mmol/L (>11 mg/dL)<br>2. **Renal insufficiency:** creatinine clearance <40 mL per min or serum creatinine >177 µmol/L (>2 mg/dL)<br>3. **Anemia:** hemoglobin value of >20 g/L below the lower limit of normal, or a hemoglobin value <100 g/L<br>4. **Bone lesions:** one or more osteolytic lesions on skeletal radiography, CT, or PET-CT |
| | ◆ Any one or more of the following biomarkers of malignancy:<br>◆ Clonal bone marrow plasma cell percentage* ≥60%<br>◆ Involved:uninvolved serum free light chain ratio◊ ≥100<br>◆ >1 focal lesions on MRI studies |




| | |
|---|---|
| Monoclonal proteins | Around 97% of patients with MM will have a monoclonal (M) protein produced and secreted by the malignant plasma cells, which can be detected by protein electrophoresis of the serum (SPEP) and/or of an aliquot of urine (UPEP) from a 24-hour collection |

| APPENDIX 122: NOBEL PRIZE | | | |
|---|---|---|---|
| **Year** | **Subject** | **Winner** | **Research Topic** |
| 2018 | PHYSIOLOGY/ MEDICINE | James P Allison and Tasuku Honjo | Cancer therapy by inhibition of negative immune regulation |
| | CHEMISTRY | Frances H. Arnold | Directed evolution of enzymes |
| | | George P. Smith, Sir Gregory P. Winter | Phage display of peptides and antibodies |
| | PHYSICS | Arthur Ashkin, Gerard Mourou, Donna Strickland | Groundbreaking inventions in the field of laser physics |
| 2017 | PHYSIOLOGY/ MEDICINE | Jeffrey C. Hall, Michael Rosbash and Michael W. Young | Molecular mechanisms controlling the circadian rhythm |
| | CHEMISTRY | Jacques Dubochet, Joachim Frank, Richard Henderson | Cryo-electron microscopy for the high-resolution structure determination of biomolecules in solution |
| | PHYSICS | Rainer Weiss, Barry C. Barish, Kip S. Thorne | LIGO detector and the observation of gravitational waves |
| 2016 | PHYSIOLOGY/ MEDICINE | Yoshinori Ohsumi | Mechanisms for autophagy |
| | CHEMISTRY | Jean Pierre Sauvage, Sir J. Fraser Stoffart, Bernard L. Feringa | Design and synthesis of molecular machines |
| | PHYSICS | David J. Thouless, F. Duncan M. Haldane, J. Michael Kosterlitz | Theoretical discoveries of topological phase transitions and topological phases of matter" |
| 2015 | PHYSIOLOGY/ MEDICINE | William C. Campbell & Satoshi Ōmura | Novel therapy against infections caused by roundworm parasites |
| | | Youyou Tu | Novel therapy against Malaria |
| | CHEMISTRY | Tomas Lindahl, Paul Modrich and Aziz Sancar | Mechanistic studies of DNA repair |
| | PHYSICS | Takaaki Kajita, Arthur B. McDonald | Discovery of neutrino oscillations, which shows that neutrinos have mass |
| 2014 | PHYSIOLOGY/ MEDICINE | John O'Keefe, May-Britt Moser, Edvard I. Moser | Cells that constitute a positioning system in the brain |
| | CHEMISTRY | Eric Betzig, Stefan W. Hell and William E. Moerner | Development of super-resolved fluorescence microscopy |
| | PHYSICS | Isamu Akasaki, Hiroshi Amano and Shuji Nakamura | For the invention of efficient blue light-emitting diodes which has enabled bright and energy-saving white light sources |
| 2013 | PHYSIOLOGY/ MEDICINE | James E. Rothman, Randy W. Schekman, Thomas C. Südhof | Machinery regulating vesicle traffic, a major transport system in our cells |
| | CHEMISTRY | Martin Karplus, Michael Levitt, Arieh Warshel | Development of multiscale models for complex chemical systems |
| | PHYSICS | François Englert, Peter W. Higgs | Origin of mass of subatomic particles |

## APPENDIX 3: ABDOMINAL INCISIONS

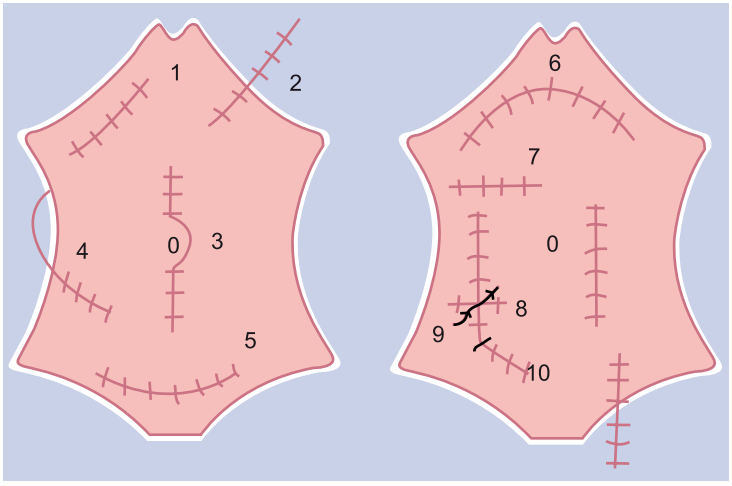



**Fig: Commonly used abdominal incisions**

**Commonly used abdominal incisions**

1. Kocher's incision
2. Thoracoabdominal incision
3. Midline incision
4. Loin (Muscle splitting) incision
5. Pfannenstiel incision
6. Gable incision
7. Transverse muscle splitting incision
8. Lanz (Muscle splitting) incision
9. Gridiron (Muscle splitting) incision
10. Rutherford Morrison (Muscle cutting) incision
11. Paramedian incision
12. McEvedy incision

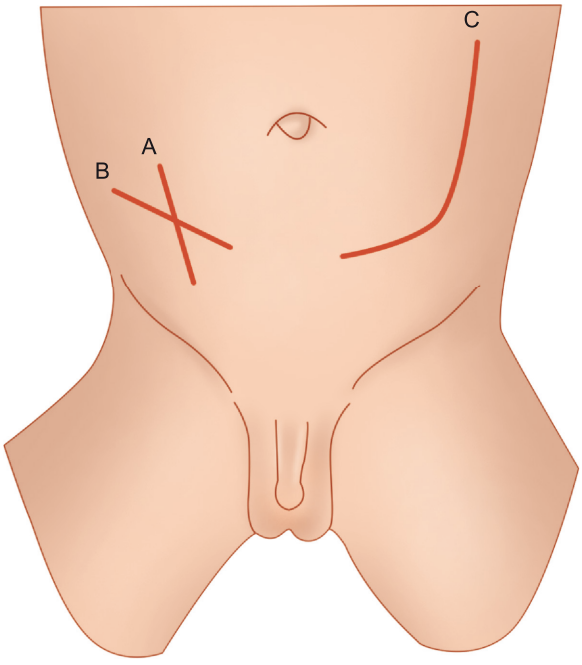



**Fig: Incisions used in Open Appendectomy**

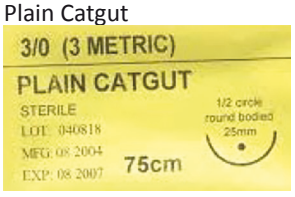
**Incisions used in Open Appendectomy:**

1. Gridiron (Muscle splitting) incision- Right angled to Spino-umbilical line at Mc Burney's point
2. Lanz (Muscle splitting) incision- Transverse incision at McBurney's point
3. Rutherford Morrison (Muscle cutting) incision (Shown on left side for clarity)

**Layers encountered in muscle splitting incision of Open appendectomy are:**

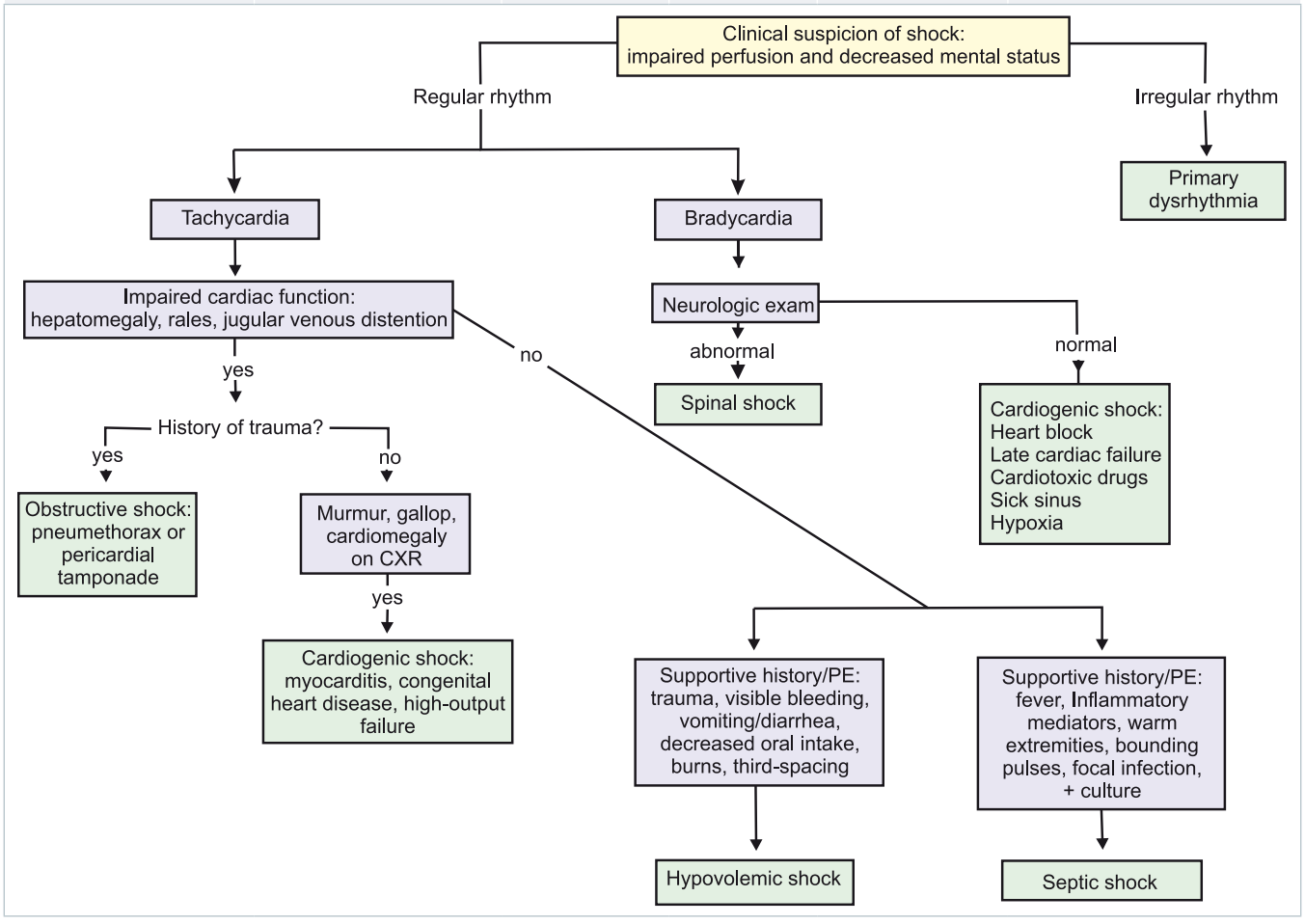
Skin
Subcutaneous fat
Scarpa's fascia
External oblique aponeurosis
Internal Oblique
Transverse abdominis
Fascia transversalis
Pre peritoneal fat
Parietal Peritoneum

## APPENDIX 4: CHARACTERISTICS OF ABSORBABLE SUTURE MATERIALS

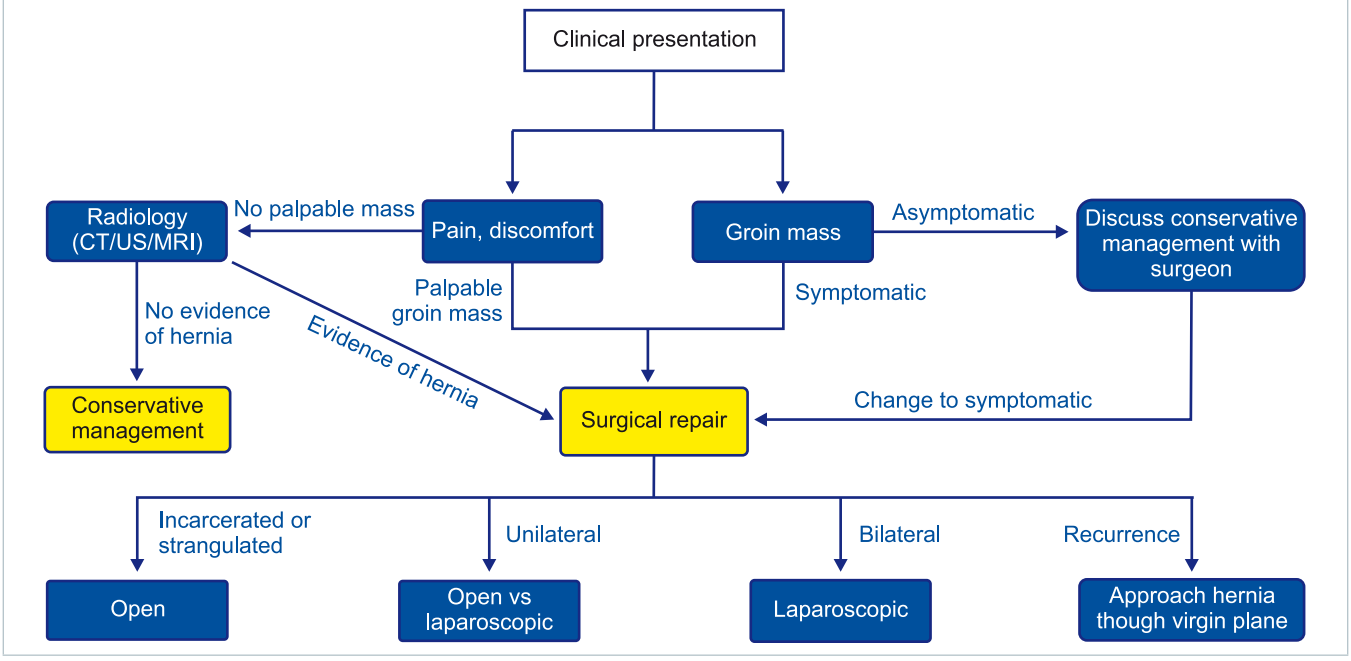
| Suture (trade name) | Manufacturing process | Effective strength (d) | Complete absorption (d) | Absorption profile | | Application |
|---|---|---|---|---|---|---|
| | | | | Tissue reactivity | Handling | |
| Plain Catgut   | Collagen from sheep intestine submucosa | 4–10 | 70 | High | Poor | Used for quick-healing mucosa |

## APPENDIX 17: HEMODYNAMIC PROFILES OF SHOCK

| Physiologic variable | Preload | Pump function | | Afterload | Tissue perfusion |
|---|---|---|---|---|---|
| **Clinical measurement** | **Pulmonary capillary wedge pressure** | **Cardiac output** | **Heart rate** | **Systemic vascular resistance** | **Mixed venous oxyhemoglobin saturation** |
| Hypovolemic | ↔ (early) or ↓ (late) | ↔ (early) or ↓ (late) | ↑ | ↑ | >65% (early) or <65% (late) |
| Cardiogenic | ↑ | ↓ | ↑ | ↑ | <65% |
| Distributive (septic shock, anaphylactic, neurogenic shock) | ↔ (early) or ↓ (late) | ↑ or ↓ (occasionally) | ↑ (No change or ↓in neurogenic shock | ↓ | >65% |
| Obstructive | | | | | |
| PE, PH, tension pneumothorax | ↔ (early) or ↓ (late) | ↔ (early) or ↓ (late) | ↑ | ↑ | >65% |
| Pericardial tamponade | ↑ | ↓ | ↑ | ↑ | <65% |



## APPENDIX 49: APPROACH OF HERNIA



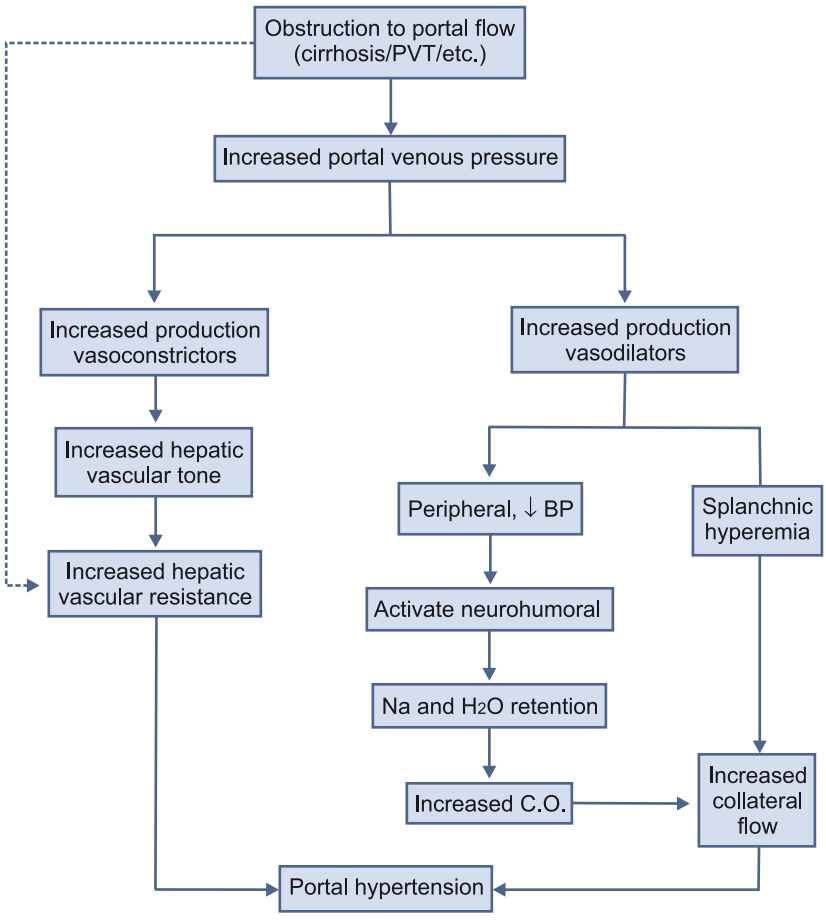

## APPENDIX 50: EPIGASTRIC HERNIA

| | |
|---|---|
| Location | These arise through the ***midline raphe (linea alba)*** anywhere ***between the xiphoid process and the umbilicus***, usually midway. |
| Defect | ◆ The region of this midline raphe is termed the linea alba, and the rectus muscles are situated just lateral to the linea alba. In this area, there is no muscle layer to protect against herniation of intra-abdominal contents through defects in the midline fascia. **The midline defect is usually elliptical in nature, with the long axis oriented transversely.**<br>◆ Epigastric hernias begin with a transverse split in the midline raphe so, in contrast to umbilical hernias, the defect is elliptical.<br>◆ Epigastric hernia defects are usually less than 1 cm in maximum diameter and commonly contain only extraperitoneal fat which gradually enlarges, spreading in the subcutaneous plane to resemble the shape of a mushroom. When very large they may contain a peritoneal sac but rarely any bowel. |
| Clinical features | The patients are often fit, healthy males between 25 and 40 years of age. These hernias can be very painful even when the swelling is the size of a pea due to the fatty contents becoming nipped sufficiently to produce partial strangulation. The pain may mimic that of a peptic ulcer but symptoms should not be ascribed to the hernia until gastrointestinal pathology has been excluded. A soft midline swelling can often be felt more easily than it can be seen. It may be locally tender. It is unlikely to be reducible because of the narrow neck. It may resemble a lipoma. A cough impulse may or may not be felt. |
| Management | Very small epigastric hernias disappear spontaneously. Small to moderate sized hernias without a peritoneal sac are not inherently dangerous and surgery should only be offered if the hernia is sufficiently symptomatic. More than one hernia may be present. The most common cause of 'recurrence' is failure to identify a second defect at the time of original repair. |

## APPENDIX 81: PORTAL HYPERTENSION

**Pathophysiology**

**PATHOPHYSIOLOGY OF PORTAL HYPERTENSION**




**Clinical features**

- Variceal bleeding is one of the most lethal complications of portal hypertension
- Ascites develops in patients with cirrhosis at a more advanced stage than may be the case for variceal bleeding
- Liver failure and encephalopathy are common complications of portal hypertension
- Hepatocellular carcinoma (HCC) is an increasingly frequent complication of cirrhosis seen in patients with portal hypertension

**Etiology**

| Prehepatic | Intrahepatic | Posthepatic |
|---|---|---|
| Portal or splenic vein thrombosis<br>Extrinsic portal vein compression<br>Arteriovenous fistula | Cirrhosis: multiple etiologies<br>Schistosomiasis<br>Congenital hepatic fibrosis<br>Rare causes | Budd-Chiari syndrome<br><br>Constrictive pericarditis |

**Evaluation**

| Endoscopy | Imaging | LFT |
|---|---|---|
| Size of varices<br>Extent of varices<br>Risk factor, red color signs<br>Portal gastropathy | Doppler ultrasound<br>CT scan<br>HVPG and imaging<br>Angiography | Clinical: ascites, encephalopathy, jaundice, muscle wasting<br>Laboratory data<br>Child's score<br>MELD score |

| APPENDIX 100: CLASSIFICATION OF PRIMARY GASTROINTESTINAL STROMAL TUMORS BY RISK OF METASTASIS | | |
|---|---|---|
| **Risk Category** | **Size** | **Mitotic Count** |
| Very Low | <2 cm | <5 per 50 HPFs* |
| Low | 2–5 cm | <5 per 50 HPFs |
| Intermediate | <5 cm | 6–10 per 50 HPFs |
| | 5–10 cm | <5 per 50 HPFs |
| High | >5 cm | >5 per 50 HPFs |
| | >10 cm | Any mitotic rate |
| | Any size | >10 per 50 HPFs |

| APPENDIX 101: ULCERATIVE COLITIS VS CROHN'S DISEASE | | |
|---|---|---|
| | **Ulcerative Colitis** | **Crohn's Disease** |
| **EPIDEMIOLOGY** | | |
| Incidence (North America) per person-years | 2.2–14.3/100,000 | 3.1–14.6/100,000 |
| Age of onset | 15–30 & 60–80 | 15–30 & 60–80 |
| Ethnicity | Jewish > Non-Jewish Caucasian > African American > Hispanic > Asian | Jewish > Non-Jewish Caucasian > African American > Hispanic > Asian |
| Male: Female ratio | 1:1 | 1.1–1.8:1 |
| Smoking | May prevent disease | May cause disease |
| Oral contraceptives | No increased risk | Odds ratio 1.4 |
| Appendectomy | Protective | Not protective |
| Monozygotic twins | 6% concordance | 58% concordance |
| Dizygotic twins | 0% concordance | 4% concordance |
| **PATHOLOGY** | | |
| Distribution | Diffuse (Pancolitis) | Skip lesions |
| Inflammation | Limited to mucosa | Transmural |
| Pseudopolyps | Marked | No to slight |
| Ulcers | Superficial | Deep linear |
| Granuloma | No | Yes (50%) |
| Fistula/Sinuses | No | Yes |
| **CLINICAL** | | |
| Gross blood in stool | Yes | Occasionally |
| Mucus | Yes | Occasionally |
| Systemic symptoms | Occasionally | Frequently |
| Pain | Occasionally | Frequently |
| Abdominal mass | Rarely | Yes |
| Significant perineal disease | No | Frequently |
| Fistulas | No | Yes |
| Small-intestinal obstruction | No | Frequently |
| Colonic obstruction | Rarely | Frequently |
| Response to antibiotics | No | Yes |

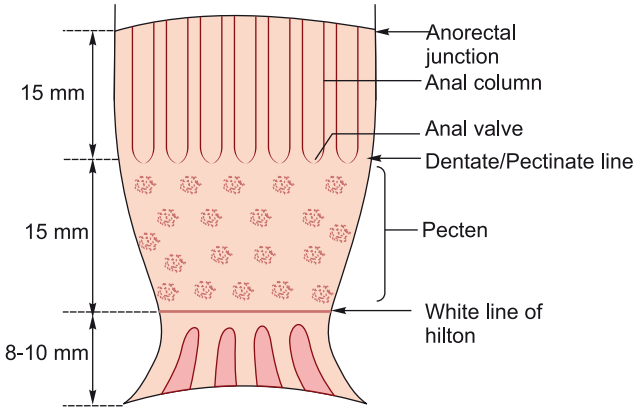
| | | | |
|---|---|---|---|
| Children (10% have lead points) | Meckel's diverticulum (Most common), followed by Polyp and duplication cyst | Hemangioma, Inverted appendix stump, Anastomotic suture line | Ectopic pancreatic tissue |

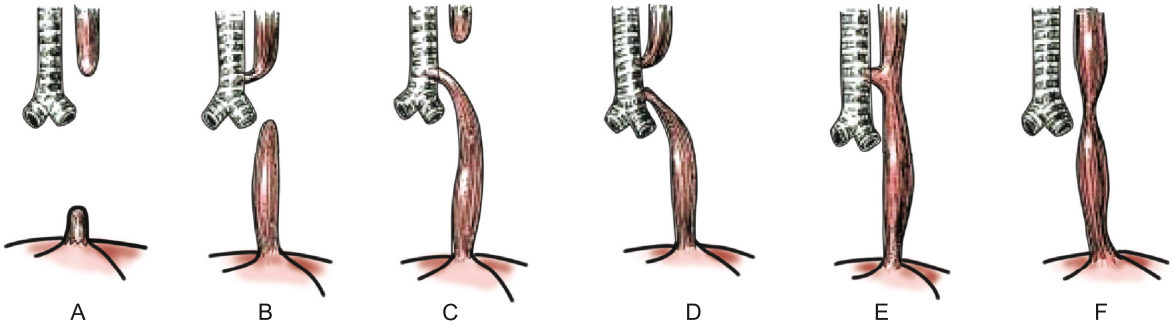
**Note:**
1. In large bowel lead point is malignant in up to 80% of cases and in small intestine the lead point is mostly benign (malignant in 1/3 cases)
2. In patients younger than 2 years, lead points are identified in less than 4% of cases. Lead points are more common in children older than 2 yr of age; the older the child, the higher the risk of a lead point

## APPENDIX 109: HAEMORRHOIDS

| | **Internal Haemorrhoids** | **External Haemorrhoids** |
|---|---|---|
| | **Above pectinate line** | **Below pectinate line** |
| Embryological origin | Endoderm (Cloaca) | Ectoderm |
| Nerve supply | Visceral afferent nerves: an incision or a needle insertion in this region is **painless**. Sensitive only to stretching | **Inferior rectal nerves -S2, S3, S4** (AKA inferior anal nerves, inferior hemorrhoidal nerve) a branch of Pudendal nerve containing **somatic sensory fibers**. Quite sensitive to pain, touch, and temperature |
| Arterial supply | **Superior rectal (Hemorrhoidal) artery** (Br of inferior mesenteric) | Middle rectal artery (Br of internal iliac artery), Inferior rectal artery (Br of internal pudendal) |
| Venous drainage (internal rectal venous plexus drains in both directions from the level of the pectinate line) | Internal rectal venous plexus drains into **superior rectal vein** (tributary of inferior mesenteric vein) and the **portal system** | Internal rectal venous plexus drains into **inferior rectal veins** and **Middle rectal vein** (tributaries of the caval venous system) around the margin of the external anal sphincter |
| Lymphatic drainage | Internal iliac nodes | Superficial inguinal nodes |
| Epithelium | Simple columnar | Stratified squamous (Non keratinized above line of Hilton, Keratinized below line of Hilton) |
| Associated skin Tag | No | Often |
| DRE | Cannot be detected | Can be detected |
| Ligation as management | Done as internal haemorrhoids are painless | Cannot be done as external haemorrhoids are painful |

# The Dentate Line is also called as Pectinate Line
## Internal hemorrhoids ("piles") are prolapses of the rectal mucosa containing the normally dilated veins of the internal rectal venous plexus
External hemorrhoids are thromboses (blood clots) in the veins of the external rectal venous plexus and are covered by skin.
The anastomoses among the superior, middle, and inferior rectal veins form clinically important communications between the portal and the systemic venous systems. The superior rectal vein drains into the inferior mesenteric vein, whereas the middle and inferior rectal veins drain through the systemic system into the inferior vena cava

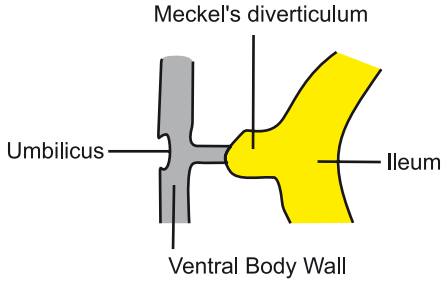


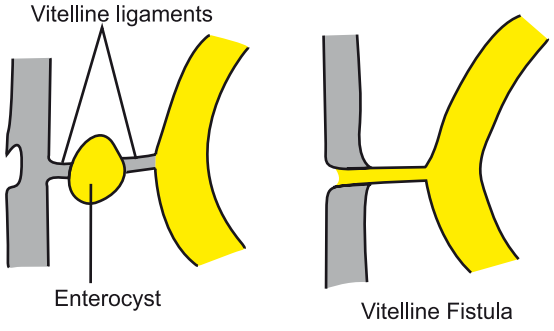



| | |
|---|---|
| **Associated Anomalies** | The etiology of the disturbed embryogenesis is presently unknown. Roughly one third of infants with EA or TEF have low birth weight, and two thirds of infants have associated anomalies. There is a nonrandom, nonhereditary association of anomalies in patients with EA or TEF that must be considered under the acronym VATER (vertebral, anorectal, tracheal, esophageal, renal or radial limb). Another acronym that is commonly used is VACTERL (vertebral, anorectal, cardiac, tracheal, esophageal, renal, and limb). |

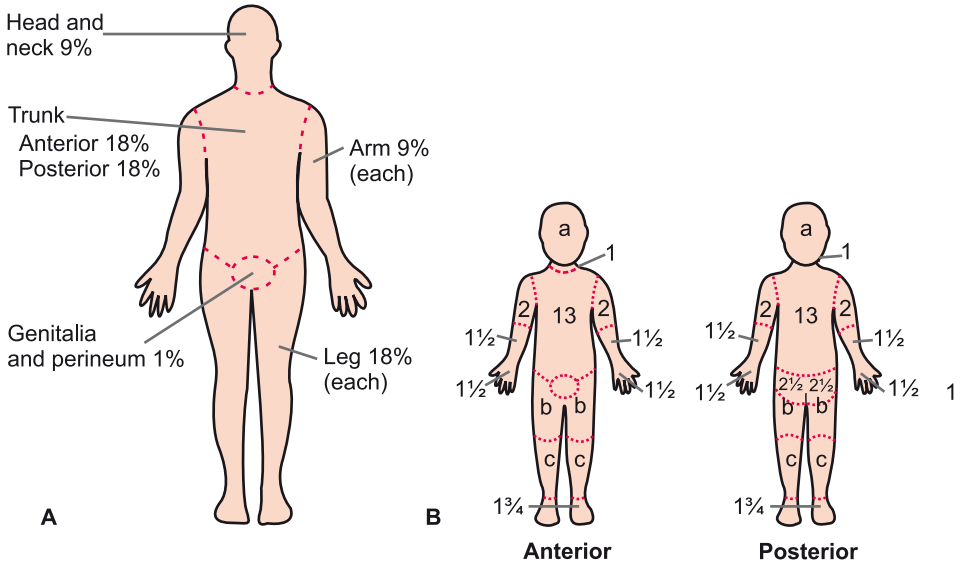
## APPENDIX 120: MECKEL'S DIVERTICULUM

| | |
|---|---|
| Definition | A Meckel's diverticulum is a persistent remnant of the vitellointestinal duct and is present in about 2% of the population. It is the most common congenital anomaly of the alimentary tract |
| Embryology | In fetal life the umbilicus is connected to the gut by the vitellointestinal duct. Further duct becomes totally obliterated and vanishes. The bowel end (Proximal end) of the duct may persist as a Meckel's diverticulum. |




| | |
|---|---|
| Epidemiology | Present in 2% population |
| Anatomy | ◈ It is found on the antimesenteric side of the ileum, commonly at 60 cm **(2 foot) from the ileocaecal valve** and is classically 5 cm long **(2 inches)**<br>◈ A Meckel's diverticulum contains all three coats of the bowel wall and has its own blood supply. |




Relative percentage of body surface area (% BSA) affected by growth

| Age | 0-1 | 1-4 | 5-9 | 10-14 | 15 |
|---|---|---|---|---|---|
| A – ½ of head | 9½% | 8½% | 6½% | 5½% | 4½% |
| B – ½ of one thigh | 2¾% | 3¼% | 4% | 4¼% | 4½% |
| C – ½ of one leg | 2½% | 2½ % | 2¾% | 3% | 3¼% |

**(B) For burns in children:** In children younger than 3 years old, the head accounts for a larger relative surface area and should be taken into account when estimating burn size. Diagrams such as the **Lund and Browder chart** give a more accurate accounting of the true burn size in children. It is the **most accurate method** and can also be used for adults

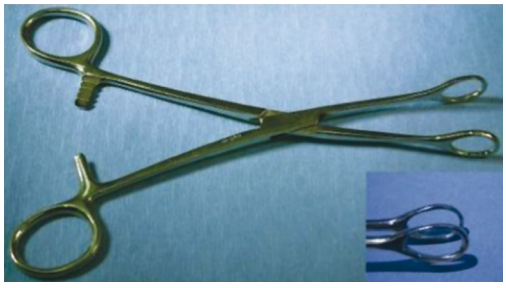
**Footnote:** Superficial Burns (First Degree) are not included in the assessment of the TBSA of a burn victim

## APPENDIX 134: GUIDELINES FOR REFERRAL TO A BURN CENTER

| |
|---|
| Partial-thickness burns greater than 10% TBSA |
| Burns involving the face, hands, feet, genitalia, perineum, or major joints |
| Third-degree burns in any age group |
| Electrical burns, including lightning injury |
| Chemical burns |
| Inhalation injury |
| Burn injury in patients with complicated pre-existing medical disorders |
| Patients with burns and concomitant trauma in which the burn is the greatest risk. If the trauma is the greater immediate risk, the patient may be stabilized in a trauma center before transfer to a burn center. |
| Burned children in hospitals without qualified personnel for the care of children |
| Burn injury in patients who will require special social, emotional, or rehabilitative intervention |

**Footnote:**

- TBSA = total body surface area.
- If BSA of burn is >15% for adult or >10% for child, patient requires hospitalization for intravenous fluid resuscitation

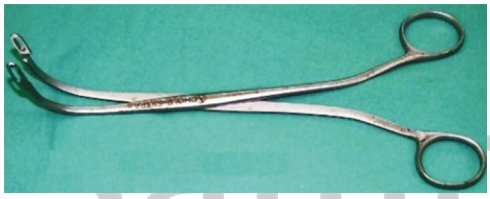


**RAMPLEY'S SWAB (SPONGE) HOLDING FORCEPS:**

- Long instrument (9 and 1/2 inch long) with finger bows, long shaft with catch, box joint, and a pair of blades
- Tip of blades are oval and **fenestrated with transverse serrations** on inner aspect, to hold swab firmly without slipping.
- Long instrument helps to clean operative area without touching the unsterile area (no touch technique)
- Sterilized by autoclaving
- Used for preoperative **cleaning (scrubbing)** of skin of operative area with aseptic solutions
- Additionally can be used in **holding fundus of gall bladder, tongue, cervix and for removing laminated daughter cysts during hydatid cyst removal**

**LISTER'S SINUS FORCEPS**

- Long slender instrument with pairs of blade having transverse serrations only on tip and their tips are blunt (olive tipped)
- Shaft have no catch to prevent vital structures from crushing
- Used to break loculi and to introduce corrugated rubber tubes or roller pack in abscess cavity and exploring sinus tract

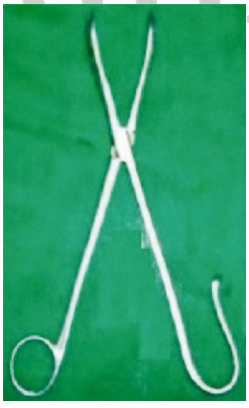


**DESJARDIN'S CHOLEDOCHOLITHOTOMY FORCEPS**

**Parts:** Long curved blades with no serrations, flat and fenestrated tip with no serrations for stone holding, finger bows, screw type joint, shaft (curved with no catch to avoid crushing of stone while removal)

**Uses:**

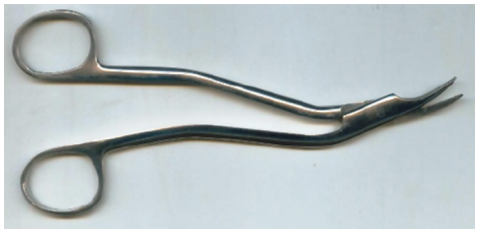
1. During choledocholithotomy it is inserted in CBD and stones are removed.
2. Can also be used in nephrolithotomy, pyelolithotomy and ureterolithotomy



**SUPRAPUBIC CYSTOLITHOTOMY FORCEPS**

**Parts:** shaft (handle) is peculiar with one finger bow for thumb and one incomplete ring like a hook for remaining fingers, for adequate grip without crushing stone. Blades are spoon shaped with concave inside and fine spicules or blunt serrations for stone holding without crushing. No catch

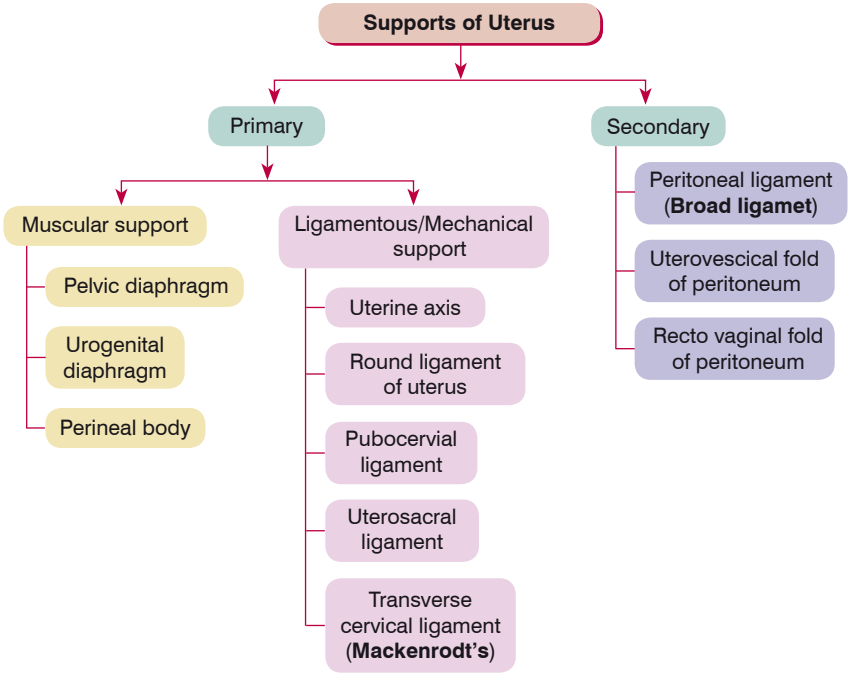
**Uses:** To remove bladder stones (vesicle calculus) during suprapubic cystolithotomy.



**HEATH'S SUTURE CUTTING SCISSORS**

- Fine scissors curved at an angle
- Small blades with serrations to grip sutures while cutting
- Used to cut skin sutures after wound healing

## SUPPORTS OF UTERUS



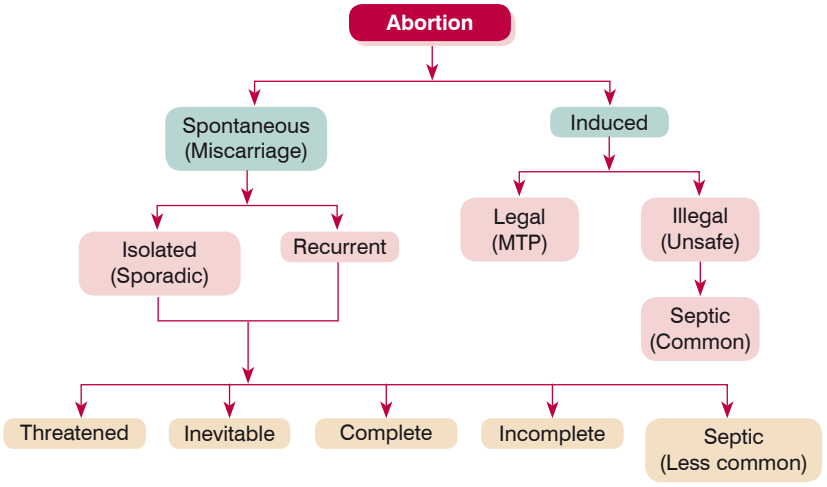

### Primary Supports of Uterus

| | |
|---|---|
| **Muscular or active supports:** | ◆ Pelvic diaphragm (formed by Levator ani muscle)<br>◆ Perineal body<br>◆ Pyramidal shape<br>◆ Situated between vagina and Anal canal, about 1.25 cm in front of anus.<br>◆ 9 muscles meet here:<br>**3 unpaired**<br>– External anal sphincter<br>– Bulbospongiosus<br>– Longitudinal muscle coat of rectal ampulla & anal canal<br>**3 Paired**<br>– 2 Levator Ani<br>– 2 Superficial transverse perineal muscle<br>– 2 Deep transverse perineal mucle<br>◆ Urogenital diaphragm |
| **Fibromuscular or mechanical support** | 1. Uterine axis<br>2. Round ligament of uterus<br>**3. Major Ligaments; Triradiate ligament**<br>a) Pubocervical ligament<br>b) Uterosacral ligament<br>c) Cardinal ligaments (or Mackenrodt's ligament/ lateral cervical ligament/ transverse cervical ligament) |

### Secondary Supports of Uterus

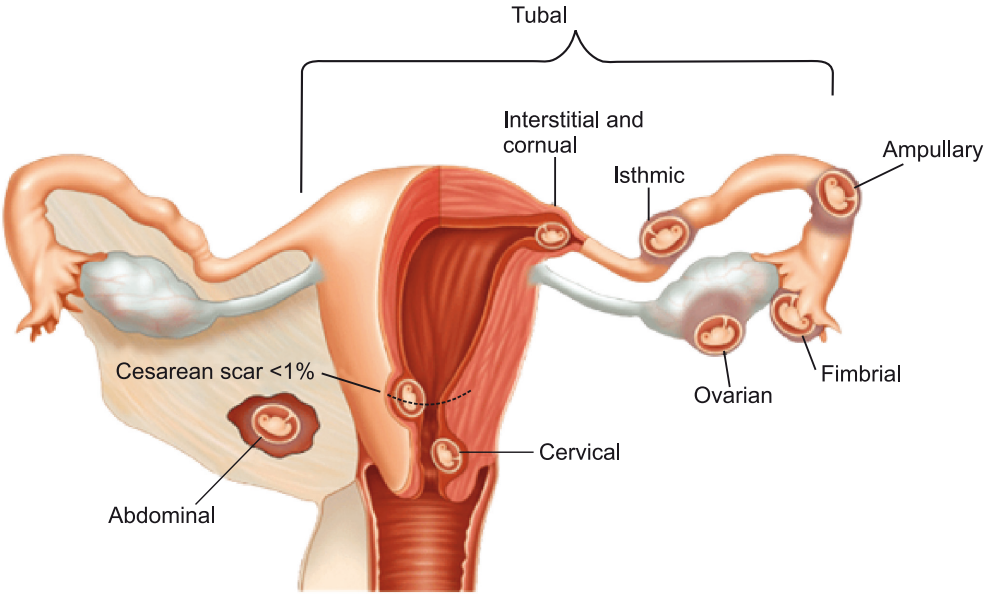
1. Broad ligament
2. Utero vaginal fold of peritoneum
3. Rectovaginal fold of peritoneum

## APPENDIX 63: TYPES OF ABORTION




| | Missed | Threatened | Inevitable | Incomplete | Complete |
|---|---|---|---|---|---|
| | Historically, the term was used to describe dead products of conception that were retained for days, wks, or even months in the uterus with a closed cervical os. Now it is used interchangeably with early pregnancy loss or wastage | Process of abortion has started but recovery is possible. | POC is inside the uterus but os is open which results in complete abortion almost always. | The fetus and the placenta may remain entirely within the uterus or partially extrude through the dilated os. | Expulsion of the entire pregnancy is already completed. |
| Clinical picture | Minimal or absent bleeding as it is a USG diagnosis now. | bloody vaginal discharge or bleeding and /or pain | Gross rupture of the membranes along with cervical dilatation | Bleeding that follows partial or complete placental separation and dilation of the cervical os | Heavy bleeding, cramping, and passage of tissue or a fetus is common. |
| Uterine size | Due to early detection usually corresponds in present times. | Corresponds | Corresponds or less. | Smaller | Smaller |
| Status of int. os | Closed | Closed | Open | Open | Closed |
| USG | Dead fetus | Fetus live, subchorionic hemorrghage+ | Fetus- live or dead | Fetus or RPOC | Empty cavity |
| Management | D&C | Conservative | Conservative if no additional Amniotic fluid has escaped and if there is no bleeding, cramping, or fever and fetus is live. | D&C, medical abortion, or expectant management in clinically stable women. | D&C if RPOC |

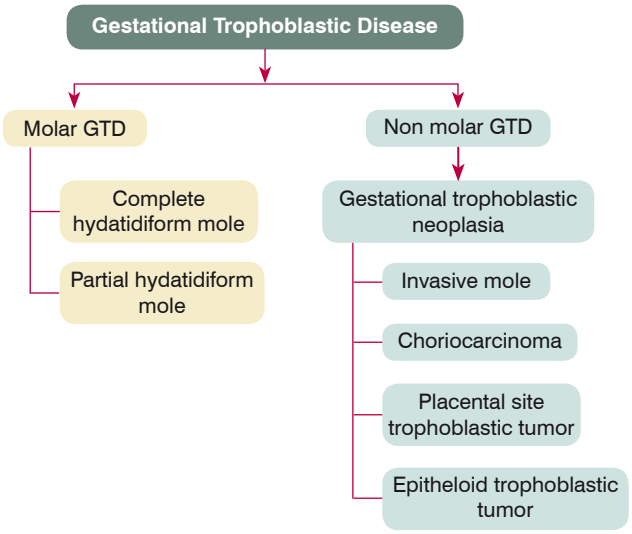
## APPENDIX 68: ECTOPIC PREGNANCY




| | |
|---|---|
| **Definition** | Fertilized ovum is implanted and develops outside the normal endometrial cavity. |
| **Site** | ◈ Isthmus: Disturbance or rupture earliest because it is the narrowest part of the tube.<br>◈ Interstitial: (pregnancy longest) may continue for 3-4 month.<br>◈ Commonest ectopic pregnancy- ***Tubal***<br>◈ Rarest ectopic pregnancy- ***Primary Abdominal***<br>◈ Commonest site in fallopian tube- ***Ampulla***<br>◈ Least common site in fallopian tube- ***Interstitial part*** |
| **Epidemiology** | Most common in age group 20-30 yrs, Nulliparous. |
| **Risk factors** | 1. History of PID<br>2. IUD use (*OCPs are protective against ectopic, however use of IUCD, Progesterone only pills & Post coital estrogen pills increases the incidence of ectopic pregnancy by decreasing tubal motility*)<br>3. History of tubal ligation or tubal reconstructive surgery of tubal endometriosis<br>4. ART particularly if the tubes are patent but damaged<br>5. Contraception failure<br>6. History of infertility<br>7. Previous ectopic pregnancy or Previous induced abortion |
| **Clinical features** | **Classical triad**: Abdominal pain (100%) + Amenorrhea (75%) + Vaginal bleeding (70%)<br>Vomiting, fainting attack, Pallor, Features of shock, Tense tumid & tender abdomen, Adenexal mass palpable (50% of cases) |
| **Clinical features of tubal rupture** | ◈ Severe lower abdominal and pelvic pain that is frequently described as sharp, stabbing, or tearing.<br>◈ Tenderness during abdominal palpation and also a tender adnexal mass could be present. Bimanual pelvic examination, especially cervical motion, causes exquisite pain.<br>◈ Blood pressure will fall and pulse will rise only if bleeding continues and hypovolemia becomes significant. |
| **Spiegelberg's criteria** | Helps to identify the ovarian pregnancy from other ectopics. Includes-<br>◈ The gestational sac is located in the region of the ovary.<br>◈ The gestational sac is attached to the uterus by the ovarian ligament.<br>◈ Ovarian tissue is histologically proven in the wall of the gestational sac.<br>◈ The oviduct on the affected side is intact (this criterion, however, holds not true for a longer ongoing ovarian pregnancy). |

## APPENDIX 81: GESTATIONAL TROPHOBLASTIC DISEASE

- Gestational trophoblastic disease (GTD) refers to a Pregnancy-related placental tumors.
- GTD is divided into ***Molar*** and ***Nonmolar*** tumors

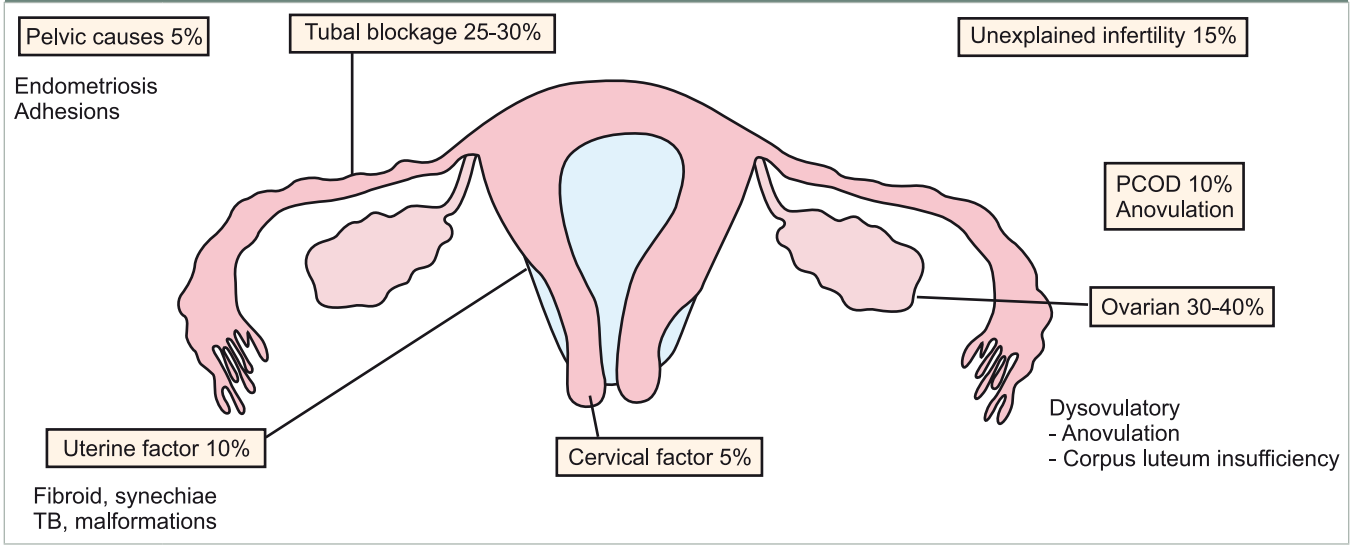



| NIH CLASSIFICATION OF GESTATIONAL TROPHOBLASTIC DISEASE | | | |
|---|---|---|---|
| I | Non-metastatic GTD | no evidence of disease outside uterus | |
| **II** | Metastatic GTD | **A.** Low-risk group | 1. Short duration (last pregnancy <4 months)<br>2. Low pretreatment HCG titre (<100 000 IU /24h urine or <40000 mIU/ml serum)<br>3. No metastasis in brain or liver<br>4. No prior chemotherapy<br>5. Antecedent pregnant event is not a term delivery (mole, ectopic pregnancy, spontaneous abortion) |
| | | **B.** High-risk group | 1. Long duration (last pregnancy >4 months)<br>2. High pretreatment HCG titre (>100 000 IU /24h urine or >40000 mIU/ml serum)<br>3. Brain or liver metastases<br>4. Significant, unsuccessful chemotherapy<br>5. Term pregnancy |

| Feature | Partial Hydatidiform Mole | Complete Hydatidiform Mole |
|---|---|---|
| **Karyotype** | 69XXY, Triploid, paternal and maternal origin | 46XX, Diploid, mostly paternal origin |
| **Immunohistochemistry** | | |
| hCG | Weak | > 2.5 multiples of the Median |
| Placental alkaline phosphatase | Strong | Weak |
| hPL | Variable | Weak |
| **Pathology** | | |
| Fetus or amnion, fetal vessels | Present | Absent |
| Hydropic villi | Variable, often focal | Pronounced, generalized |
| Trophoblastic proliferation | Focal | Variable, often marked |
| Villous scalloping | Marked | Absent |
| **Clinical** | | |

| APPENDIX 118: INFERTILITY | |
|---|---|
| Some definitions | ◆ **Infertility**: Inability of a couple to conceive within 1 year. Infertility implies a ↓in the ability to conceive and is synonymous with **sub fertility**.<br>◆ **Sterility**: An intrinsic inability to achieve pregnancy, whereas.<br>◆ **Primary infertility**: Those who have never conceived.<br>◆ **Secondary infertility**: Those who have conceived at some time in the past.<br>◆ **Fecundity**: Probability of achieving a live birth in 1 menstrual cycle.<br>◆ **Fecundability**: it is the likelihood of conception per month of exposure. Fertility, as well as infertility, of a woman or couple is best perceived as fecundability. |
| Causes | ◆ Male causes 25-40% (sole primary cause 25%, Overall contributing cause 40%)<br>◆ Female causes- 40-55%<br>◆ Combined male and female factor infertility- 20%<br>◆ Unexplained 15-20% |
| Most common | ◆ Most common cause of female sterility- ***Salpingitis.***<br>◆ Most common cause of impotence- ***Psychogenic*** |

## APPENDIX 119: FEMALE INFERTILITY

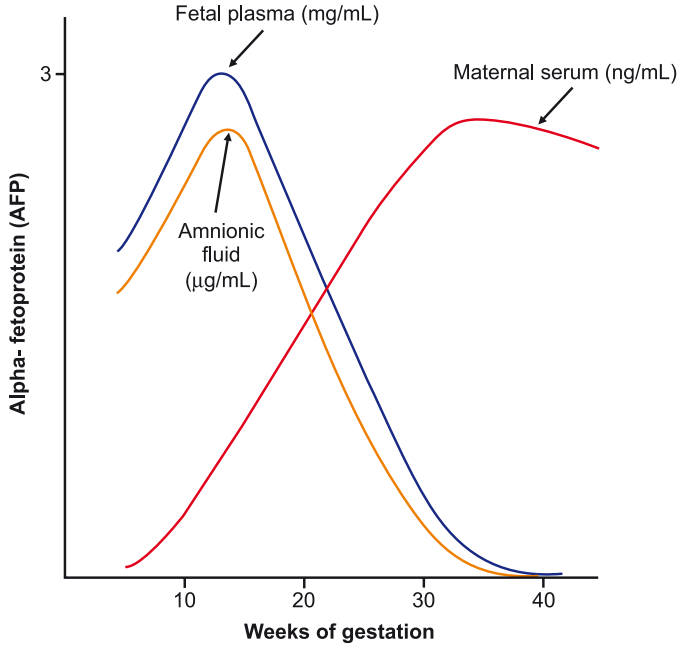


## APPENDIX 124: ALPHA FETO PROTEIN (AFP)

- ***Synthesis of AFP***: AFP is a glycoprotein synthesized by fetal yolk sac in early weeks of gestation and by the fetal GIT and liver in later part of gestation.
- AFP rapidly clears from circulation soon after birth due to short half life of 3.5 days
- Peak AFP level in:
  - Maternal serum : 32nd week (30 ng/ml)
  - Amniotic fluid/ Fetal serum: 13th week.
- Concentration of AFP in fetal serum is 1000 times > AF-AFP
- ***After 13th week***: ↓es in Fetal serum AFP & AF-AFP; ↑se in Maternal serum AFP, peak at 32 weeks.
- Main source of AF-AFP (Amniotic fluid AFP): Fetal urine.
- Fetus begins to swallow AF at 8-11 wks of gestation.
- MS-AFP is NOT elevated in meningocele as it is a closed NTD.
- If MS-AFP is high then next step is USG to determine the cause.
- **Alpha fetoprotein** levels in **amniotic fluid** show a rise and fall with gestation similar to the levels of *AFP* in fetal serum

### Abnormal AFP Levels

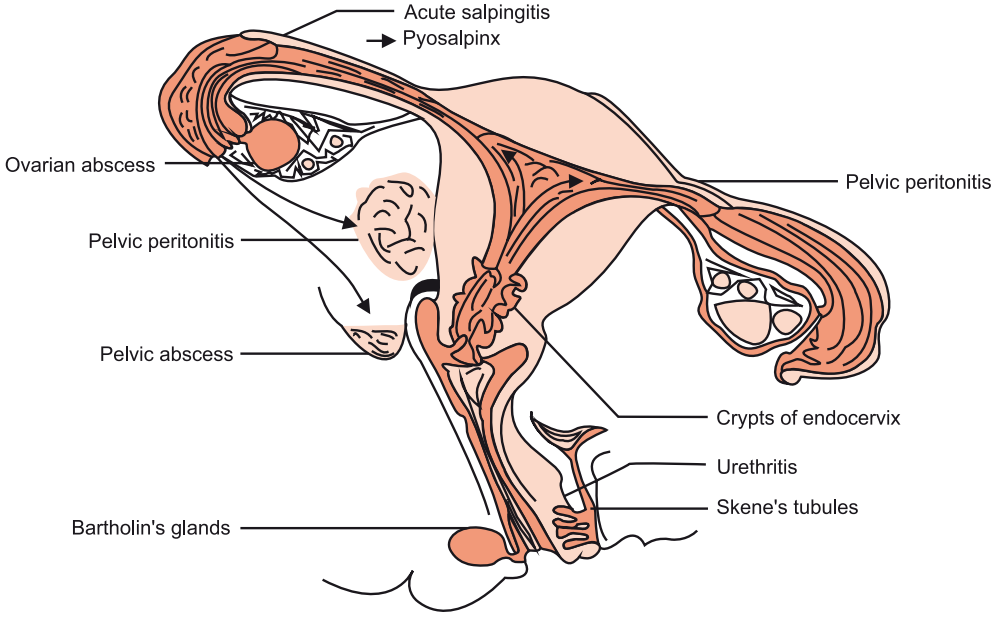
| Increase | Decrease |
|---|---|
| 1. Wrong gestational age | 1. Obesity |
| 2. Open neural tube defects (NTDs) | 2. Diabetes |
| 3. Multiple pregnancy | 3. Trisomies (Down's syndrome & Turners syndrome) |
| 4. Esophageal or duodenal atresia | 4. Gestational trophoblastic disease. |
| 5. Ventral wall defects (Gastroschisis & Omphalocele) | 5. Over estimation gestational age |
| 6. Rh isoimmunisation | 6. Missed abortion/ Fetal death |
| 7. IUFD (Inrtauterine fetal death) | 7. Gestational trophoblastic disease |
| 8. Renal anomalies | |
| 9. Sacro-coccygeal teratoma | |
| 10. Bladder extrophy | |
| 11. Amniotic band syndrome | |
| 12. Cystic hygroma | |



## APPENDIX 165: PELVIC INFLAMMATORY DISEASE (PID)

- PID implies inflammation of the upper genital tract involving the fallopian tubes & ovaries.
- Lesion is often B/L, but one tube may be more affected than the other.
- Gonococci (m/c) and chlamydia travel up the genital tract along the mucous membrane to reach the fallopian tubes and cause salpingo-oophoritis.
- Sperms also help in transportation of these organisms in a piggy-back fashion. Hence the absence of gonococcal inflammatory disease in a woman whose husband is azoospermic.
- In virgin girl PID is tubercular in nature.
- Ulceration of mucosa leads to adhesions, tubal blockage and narrowing of lumen.
- Chlamydia infection (obligate gram-ve intracellular organisms) remains asymptomatic in the endocervix or produces minimum symptoms, and therefore the infection goes unnoticed and untreated, but the damage it causes to the tube is more devastating than with gonorrhoea.
- The cervix and the urethra are the common sites where chlamydia lodge and ascend upwards.
- Use of IUCD ↑es risk for PID while barrier method prevents STD and PIDs.
- Higher rate of bacterial vaginosis is found in woman with PID.

The only exception of PID to both tubes and ovary involvement is seen in mumps where the ovary is selectively attacked




| Stages of PID | |
|---|---|
| ***Stage I*** | Acute salpingitis without peritonitis |
| ***Stage II*** | Acute salpingitis with peritonitis |
| ***Stage III*** | Acute salpingitis with superimposed tubal occlusion or tubo-ovarian complex |
| ***Stage IV*** | Ruptured tubo-ovarian abscess |
| ***Stage V*** | Tubercular salpingitis |

**Clinical Features**

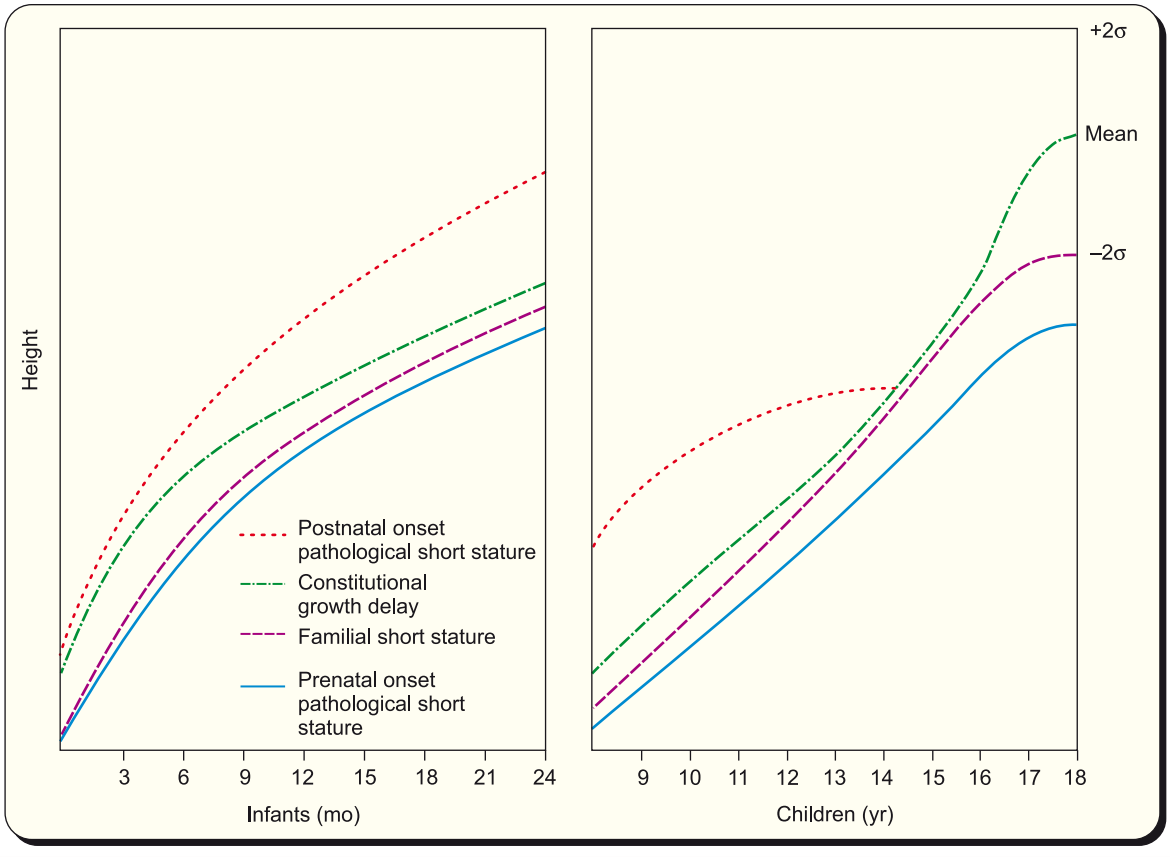
- In acute PID m/c complaint is lower abdominal pain.
- Classic triad of PID: ***Pelvic pain + Cervix motion tenderness + Adenexal tenderness.***
- In chronic PID constant low abdominal pain which gets worse before menses.
- O/E '***Frozen pelvis***' is found.

## APPENDIX 20: TYPES AND CAUSES OF SHORT STATURE

Linear growth problems are more likely to be due to congenital, constitutional, familial, or endocrine causes than to nutritional deficiency.

1. In **endocrine disorders**, length or height declines first or at the same time as weight; weight for height is normal or elevated.
2. In **nutritional insufficiency,** weight declines before length and weight for height is low (unless there has been chronic stunting).
3. In **congenital pathologic short stature,** an infant is born small and growth gradually tapers off throughout infancy. Causes include chromosomal abnormalities (Turner syndrome, trisomy 21), perinatal infection (TORCH), teratogens (phenytoin, alcohol), and extreme prematurity.
4. In **constitutional growth delay**, weight and height decrease near the end of infancy, parallel the norm through middle childhood, and accelerate toward the end of adolescence. Adult size is normal.
5. In **familial short stature,** both the infant and the parents are small; growth runs parallel to and just below the normal curves. In familial short stature, the bone age is normal (comparable to chronological age).

Figure depicts typical growth curves for four classes of decreased linear growth.




## APPENDIX 21: CLINICAL FEATURES OF COMMON CAUSES OF SHORT STATURE

| Cause | Family history | Growth pattern, clinical features and puberty | Bone age | Remarks |
|---|---|---|---|---|
| Constitutional delay | Often present | Slow from birth, immature but appropriate with late but spontaneous puberty | Moderate delay | Often difficult to differentiate from GH deficiency<br>Growth velocity measurement vital |

| Z-score | Growth indicators | | | |
|---|---|---|---|---|
| | Length/height-for-age | Weight-for-age | Weight-for-length/ height | BMI-for-age |
| **Above 3** | *See note 1* | *See note 2* | *Obese* | *Obese* |
| **Above 2** | | | *Overweight* | *Overweight* |
| **Above 1** | | | *Possible risk of overweight* | *Possible risk of overweight* |
| **0 (median)** | | | | |
| **Below – 1** | | | | |
| **Below – 2** | *Stunted* | *Underweight* | *Wasted* | *Wasted* |
| **Below – 3** | *Severely stunted* | *Severely underweight* | *Severely wasted* | *Severely wasted* |
| Wasting = Acute malnutrition; Stunting = Chronic malnutrition | | | | |

## APPENDIX 37: CLASSIFICATION OF MALNUTRITION IN CHILDREN

**1. McLaren's classification (Kanawati AA, McLaren DS)**

| Mid arm-head circumference ratio | Interpretation |
|---|---|
| > 0.310 | Normal |
| 0.310 – 0.280 | Grade I: mild malnutrition |
| 0.279 – 0.250 | Grade II: moderate malnutrition |
| < 0.250 | Grade III: severe malnutrition |

**2. Gomez Classification**

The child's weight is compared to that of a normal child (50th percentile) of the same age. It is useful for population screening and public health evaluations.

Percent of reference weight for age = ((patient weight) / (weight of normal child of same age)) x 100

| Percent of reference weight for age | Interpretation |
|---|---|
| 90 - 110% | Normal |
| 75 - 89% | Grade I: mild malnutrition |
| 60 - 74% | Grade II: moderate malnutrition |
| < 60% | Grade III: severe malnutrition |

**3. Wellcome Classification:** evaluates the child for edema and with the Gomez classification system.

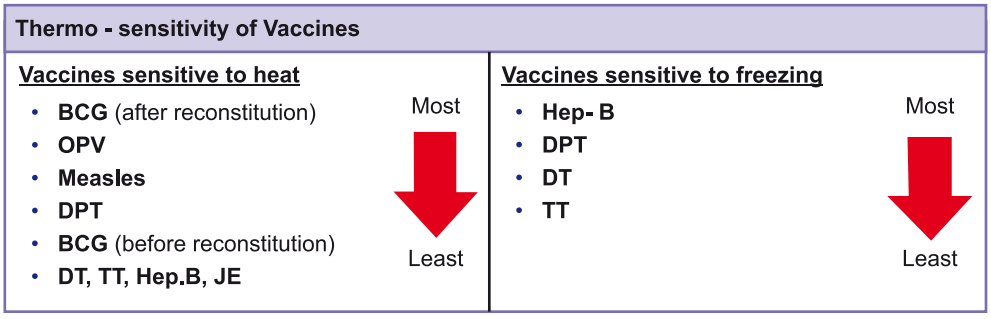
| Nutritional status | Expected weight for age | Presence of edema |
|---|---|---|
| Normal | > 80 % | No |
| Under nutrition | 60 - 80 % | No |
| Kwashiorkor | 60 - 80 % | Yes |
| Marasmus | < 60 % | No |
| Marasmic-kwashiorkor | < 60 % | Yes |

**4. Waterlow Classification/ WHO Classification:** Chronic malnutrition results in stunting. Malnutrition also affects the child's body proportions eventually resulting in body wastage.

Percent weight for height = ((weight of patient)/(weight of a normal child of the same height)) x 100

Percent height for age = ((height of patient) / (height of a normal child of the same age)) x 100

| | Weight for Height (wasting) | Height for Age (stunting) |
|---|---|---|
| Normal | > 90 % | > 95 |
| Mild | 80 - 90 % | 90 - 95 |
| Moderate | 70 – 80 | 85 - 90 |

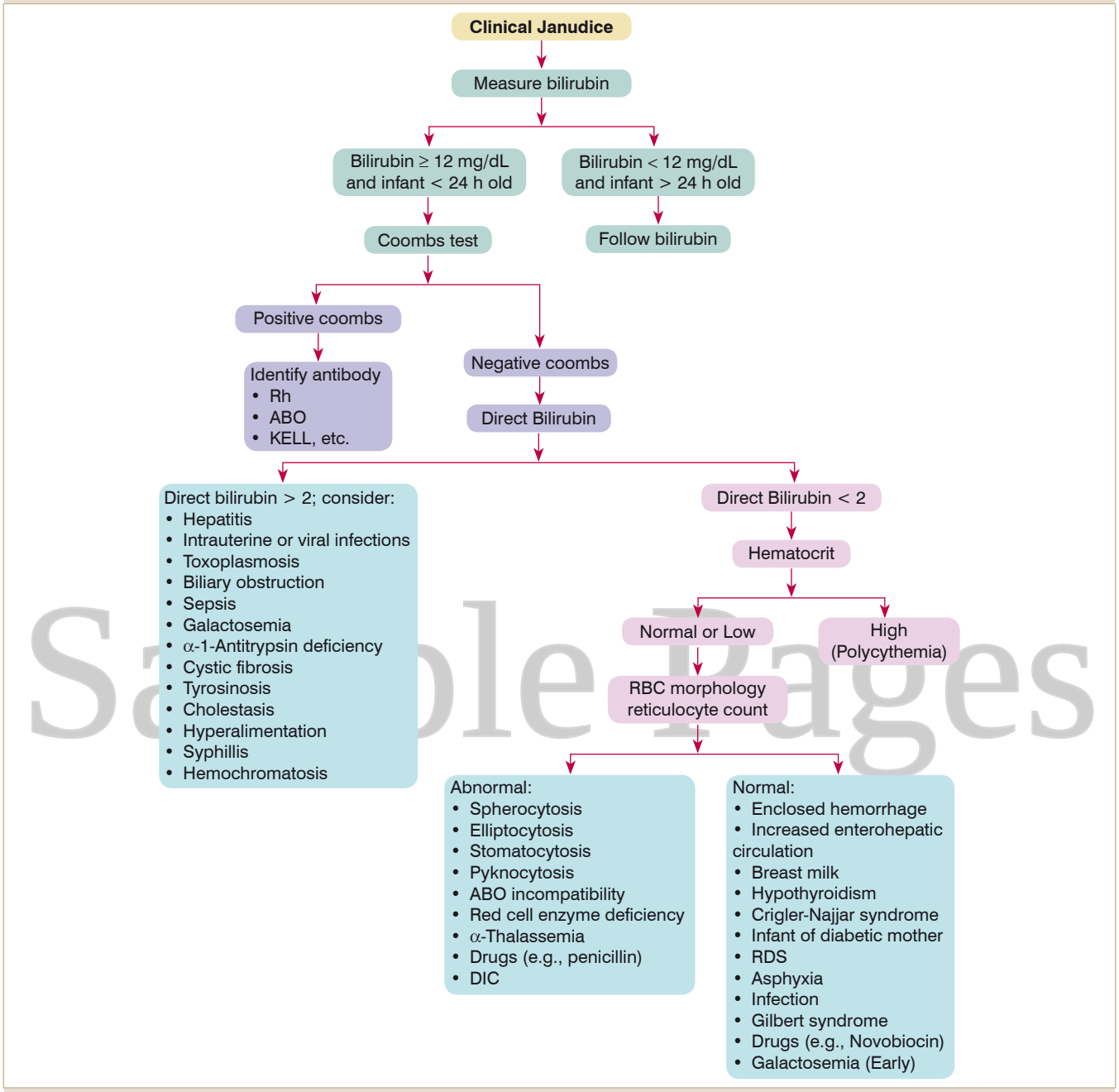



| APPENDIX 45: NATIONAL IMMUNIZATION SCHEDULE INDIA | | | | |
|---|---|---|---|---|
| **Vaccine** | **When to give** | **Dose** | **Route** | **Site** |
| **For Pregnant Women** | | | | |
| TT -1 | early pregnancy | 0.5ml | intra muscular | Upper arm |
| TT -2 | 4 weeks after 1st dose of TT* | 0.5ml | intra muscular | Upper arm |
| TT booster | If received 2 TT doses in a pregnancy within the last 3yrs | 0.5ml | intra muscular | Upper arm |
| **For Infants** | | | | |
| BCG | At birth or as early as possible till 1 year of age | 0.1ml (0.05ml until 1month age) | Intra-dermal | Left Upper Arm |
| Hepatitis B | At birth or as early as possible within 24 hours | 0.5 ml | Intra-muscular | Antero-lateral side of mid-thigh |
| OPV -0 | At birth or as early as possible within first 15 days | 2 drops | Oral | Oral |
| OPV -1, 2, 3 | 6 weeks, 10 weeks & 14 weeks | 2 drops | Oral | Oral |
| DPT- 1, 2, 3 | 6 weeks, 10 weeks & 14 weeks | 0.5 ml | Intra-muscular | Antero-lateral side of mid-thigh |
| Hepatitis B- 1, 2, &3**** | 6 weeks, 10 weeks & 14 weeks | 0.5 ml | Intra-muscular | Antero-lateral side of mid-thigh |
| Measles | 9 completed months - to 12 months. Give up to 5yrs if not received at 9 - 12 months age | 0.5 ml | Sub-cutaneous | Right upper arm |
| Vitamin A (1st dose) | At 9 months with measles | 1ml (1lakh IU) | Oral | Oral |
| **For Children** | | | | |
| DPT booster | 16-24 months | 0. 5 ml | Intra- muscular | Antero-lateral side of mid-thigh |
| OPV Booster | 16-24 months | 2 drops | Oral | Oral |
| JapaneseEncephalitis** | 16-24 months with DPT/OPV booster | 0.5 ml | Sub-cutaneous | Left Upper Arm |
| Measles | 16 - 24 months age | 0.5 ml | Sub-cutaneous | Right upper arm |
| Vitamin A***(2nd to 9th dose) | 16 months with DPT/OPV booster. Then, one dose every 6 months upto the age of 5 years. | 2 ml (2 lakh IU) | Oral | Oral |
| DT booster | 5- 6 years | 0.5 ml | Intra-muscular | Upper arm |
| TT | 10 years &16 years | 0.5 ml | Intra-muscular | Upper arm |

| APPENDIX 63: DIAGNOSIS AND TREATMENT OF CONGENITAL ADRENAL HYPERPLASIA | | | |
|---|---|---|---|
| **Disorder** | **Signs And Symptoms** | **Laboratory Findings** | **Therapeutic Measures** |
| 21-Hydroxylase deficiency, nonclassic form | May be asymptomatic; precocious adrenarche, hirsutism, acne, menstrual irregularity, infertility | ↑ Baseline and ACTH-stimulated 17-hydroxyprogesterone<br>↑ Serum androgens | Suppression with glucocorticoids |
| 11β-Hydroxylase deficiency | Glucocorticoid deficiency | ↓ Cortisol, ↑ ACTH | Glucocorticoid (hydrocortisone) replacement |
| | | ↑↑ Baseline and ACTH-stimulated 11-deoxycortisol and deoxycorticosterone | |
| | Ambiguous genitalia in females | ↑ Serum androgens | Vaginoplasty and clitoral recession |
| | Postnatal virilization in males and females | ↑ Serum androgens | Suppression with glucocorticoids |
| | Hypertension | ↓ Plasma renin, hypokalemia | Suppression with glucocorticoids |
| 3β-Hydroxysteroid dehydrogenase deficiency, classical form | Glucocorticoid deficiency | ↓ Cortisol, ↑ ACTH<br>↑↑ Baseline and ACTH-stimulated Δ5 steroids (pregnenolone, 17-hydroxy pregnenolone, DHEA) | Glucocorticoid (hydrocortisone) replacement |
| | Mineralocorticoid deficiency (salt-wasting crisis) | Hyponatremia, hyperkalemia<br>↑ Plasma renin | Mineralocorticoid (fludrocortisone) replacement; sodium chloride supplementation |
| | Ambiguous genitalia in females and males | ↑ DHEA, ↓ androstenedione, testosterone, and estradiol | Surgical correction of genitals and sex hormone replacement as necessary, consonant with sex of rearing |
| | Precocious adrenarche, disordered puberty | ↑ DHEA, ↓ androstenedione, testosterone, and estradiol | Suppression with glucocorticoids |
| 17α-Hydroxy-lase/17,20-lyase deficiency | Cortisol deficiency (corticosterone is an adequate glucocorticoid) | ↓ Cortisol, ↑ ACTH<br>↑ DOC, corticosterone<br>Low 17α-hydroxylated steroids; poor response to ACTH | Glucocorticoid (hydrocortisone) administration |
| | Ambiguous genitalia in males | ↓ Serum androgens; poor response to hCG | Orchidopexy or removal of intra-abdominal testes; sex hormone replacement consonant with sex of rearing |
| | Sexual infantilism | ↓ Serum androgens or estrogens | Sex hormone replacement consonant with sex of rearing |
| | Hypertension | ↓ Plasma renin; hypokalemia | Suppression with glucocorticoids |

| APPENDIX 64: CAUSES OF HIRSUTISM | |
|---|---|
| Gonadal hyperandrogenism | Ovarian hyperandrogenism<br>Polycystic ovary syndrome/functional<br>Ovarian hyperandrogenism<br>Ovarian steroidogenic blocks<br>Syndromes of extreme insulin resistance<br>Ovarian neoplasms |

## NEONATAL JAUNDICE WORKUP



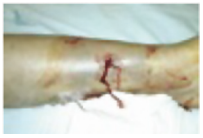
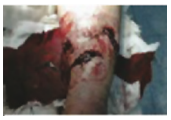
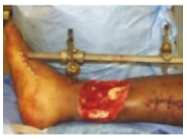
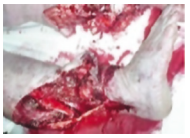



## INITIATION OF THERAPY IN NEONATAL JAUNDICE

| Group | Age | Phototherapy Cut off Bilirubin | Exchange transfusion Cut off Bilirubin |
|---|---|---|---|
| Group 1: Gestation ≥ 38 weeks and medically well | 12 hours | 9 mg/dL | 17.70 mg/dL |
| | 24 hours | 11.60 mg/dL | 19.00 mg/dL |
| Group 2: Gestation ≥ 38 weeks and clinical risk factors | 12 hours | 7.60 mg/dL | 15.10 mg/dL |
| | 24 hours | 9.80 mg/dL | 16.60 mg/dL |
| Group 3: Gestation 35 to 37.9 weeks and medically well | 12 hours | 7.60 mg/dL | 15.10 mg/dL |
| | 24 hours | 9.80 mg/dL | 16.60 mg/dL |

| | | |
|---|---|---|
| Colles' fracture | Distal radius fracture with dorsal angulation, impaction and radial drift | Fall on outstretched hand |
| Cotton's fracture | Trimalleolar fracture of ankle | |
| Clay shoveller's fracture | Stress avulsion fracture of Spinous process of C6, C7 or T1 | Forced hyper flexion of neck |
| Chopart's fracture-dislocation | Foot dislocation through talonavicular and calcaneocuboid joints with associated fractures, usually after ankle twisting. Treated in a non-weight bearing cast for 6-8 weeks | |
| Chauffeur's fracture | Intra-articular fracture of radial styloid | Forced ulnar deviation of the wrist causing avulsion of the radial styloid |
| Chance fracture | Horizontal fracture of vertebral body | Hyper flexion of spine, seen in car accidents when lap belts were used |
| Duverney fracture | Isolated fracture of the iliac wing | Direct trauma |
| Essex-Lopresti fracture | Comminuted radial head fracture with interosseous membrane disruption and distal radioulnar joint subluxation | Fall from height |
| Gosselin fracture | V-shaped distal tibia fracture extending into the tibial plafond | |
| Galeazzi fracture | Radius shaft fracture with dislocation of distal radioulnar joint | Blow to forearm |
| Holdsworth fracture | Unstable spinal fracture-dislocation at the thoracolumbar junction | |
| Hume fracture | Olecranon fracture with anterior dislocation of radial head | |
| Hill-Sachs fracture | Impacted posterior humeral head fracture occurring during anterior shoulder dislocation | |
| Hangman's fracture | Fracture of both pedicles of $C_2$ | Distraction and extension of neck (judicial hanging) |
| Jones fracture | Fracture of base of 5th metatarsal extending into intermetatarsal joint | Inversion of ankle (pronator brevis pull) |
| Jefferson fracture | Burst fracture of ring of atlas i.e. 1$^{st}$ cervical vertebra | Compression of neck |
| Lisfranc fracture | Fracture dislocation of midfoot | Forced plantar flexion of foot or dropping heavy weight on foot |
| Le Fort's fracture of the ankle | Vertical fracture of distal fibula with avulsion of medial malleolus | |
| Le Fort fractures | Series of facial fractures | Direct trauma to face |
| Moore's fracture | Distal radius fracture with ulnar dislocation and entrapment of styloid process under annular ligament | |
| Monteggia fracture | Proximal ulna fracture with dislocation of radial head | Blow to forearm |
| March fracture | Stress fracture of 2$^{nd}$ /3$^{rd}$ metatarsal shaft | Heavy or unaccustomed exercise |
| Malgaigne's fracture | Vertical pelvic fracture through both pubic rami and the ilium or sacroiliac joint with vertical displacement | High energy impact to pelvis (front to back) |
| Maisonneuve fracture | Spiral fracture of proximal fibula | External rotation of ankle |
| Pipkin fracture-dislocation | Posterior dislocation of hip with avulsion fracture of fragment of femoral head by the ligamentum teres | Impact to the knee with the hip flexed (dashboard injury) |
| Pilon (Hammer) fracture | Intra-articular fracture of tibial plafond. Usually but not always with fibular fracture | High velocity injuries |
| Pott's fracture | Bimalleolar fracture of the ankle | Eversion of ankle |
| Rolando fracture | Intra articular T or Y shaped Comminuted fracture of base of 1$^{st}$ metacarpal | Axial load along the metacarpal causing splitting of the proximal articular surface |

## APPENDIX 16: GUSTILO AND ANDERSON OPEN FRACTURE CLASSIFICATION

| Gustilo Type | I | II | IIIA | IIIB | IIIC |
|---|---|---|---|---|---|
| Images |  |  |  |  |  |
| Energy | Low energy | Moderate | High | High | High |
| Wound Size | < 1 cm | > 1 cm | >10 cm | >10 cm | >10 cm |
| Soft Tissue | Minimal | Moderate | Extensive | Extensive | Extensive |
| Contamination | Clean | Moderate contamination | Extensive | Extensive | Extensive |
| Fracture Pattern | Simple fracture pattern with minimal comminution | Moderate comminution | Severe comminution or segmental fractures | Severe comminution or segmental fractures | Severe comminution or segmental fractures |
| Periosteal Stripping | No | No | Yes | Yes | Yes |
| Skin Coverage | Local coverage | Local coverage | Local coverage including | Requires free tissue flap or rotational flap coverage | Typically requires flap coverage |
| Neurovascular Injury | Normal | Normal | Normal | Normal | Exposed fracture with arterial damage that requires repair |



## APPENDIX 17: COLLES' FRACTURE

Described by **Abraham Colles** in 1814. Fracture of distal radius at corticocancellous junction which is typically dorsally displaced and angulated

| | |
|---|---|
| Mechanism | ◆ Fracture is also caused by a forced dorsiflexion of the wrist<br>◆ Occurs in pts > 50 years of age(**Post menopausal elderly women**)<br>◆ Fall on out stretched hand<br>◆ Dorsal surface undergoes compression while volar surface undergoes tension |
| Displacements | ◆ **Mnenonic: SLIP** (L and P comes twice, so total 6 displacements)<br>1. Supination (External rotation)<br>2. Lateral displacement<br>3. Lateral tilt/angulation<br>4. Impaction<br>5. Posterior/ Dorsal displacement<br>6. Posteriro/ Dorsal tilt |
| Deformity | **Dinner fork/ Silver fork/ Spoon shaped deformity** |
| Complications | 1. Finger and joints stiffness is most common complication<br>2. Malunion is 2nd **most common**<br>3. **Sudeck's osteo dystrophy (colles # is MC cause of Sudeck of upper limb)**<br>4. Shoulder hand syndrome<br>5. Rupture of extensor pollicis tendon<br>6. Carpal tunnel syndrome<br>7. Carpal instability<br>8. Triangular fibro cartilage complex (TFCC) injury<br>9. Delayed and non union are rare |

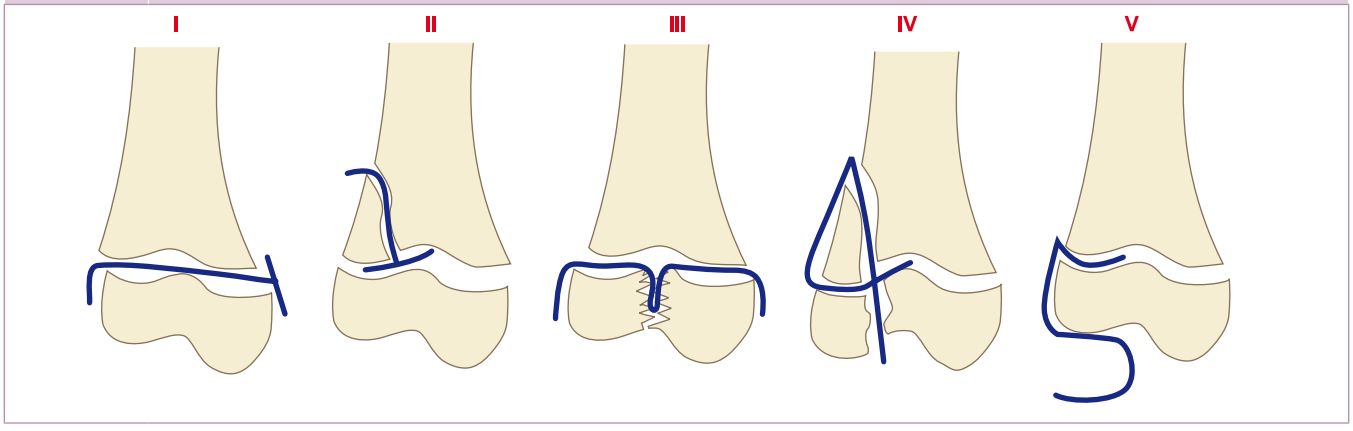
| | |
|---|---|
| Treatment | 1. Side-arm skin traction for Type 1 injury (**Dunlop traction**)<br>2. Overhead skeletal traction for Type 1 injury<br>3. **CRPP** (closed reduction percutaneous pinning) for type 2 injury<br>4. **ORIF** (open reduction and internal fixation) for type 3 injury. performed emergently (<8 hours) or urgently (≤24 hours) or after the swelling has decreased, but not later than 5 days after injury because the possibility of myositis ossificans apparently increases after that time |
| Early Complications | 1. **Brachial artery injury** (earlier 5%, now a days < 1%) - perform angiography, or doppler<br>2. Compartment syndrome (uncommon)<br>3. **Nerve injury:** Most commonly **median nerve (particularly the anterior interosseous branch)**. Most nerve injuries are associated with type III displaced supracondylar fractures. The radial nerve lies posterolateral to the supracondylar fractures thus less commonly involved. Ulnar nerve which is posteriorly located is uncommonly injured. Conclusion: **MEDIAN > ULNAR > RADIAL**<br>4. **Neuropraxia**—is reported to occur in 3% to 22% |
| Late complications | 1. **Malunion**<br>2. **Cubitus varus** (carrying angle < 5 degrees) and Cubitus valgus (carrying angle > 15 degrees) (**Cubitus varus AKA Gun stock deformity is far more common**). Cubitus varus is the most common angular deformity that results from supracondylar fractures in children. The most common cause is malunion of a supracondylar fracture.<br>3. **Cubitus valgus**, although mentioned in the literature as causing tardy ulnar nerve palsy, rarely occurs and is more often caused by nonunion of lateral condylar fractures.<br>4. **Tardy ulnar nerve palsy** (Not due to supracondylar fracture per se but its complication as valgus)<br>5. Elbow stiffness and myositis ossifficans |
| Management of cubitus varus | Three basic types of osteotomies have been described: a medial opening wedge osteotomy with a bone graft, an oblique osteotomy with derotation, and a lateral closing wedge osteotomy (easiest, safest and most stable osteotomy) |



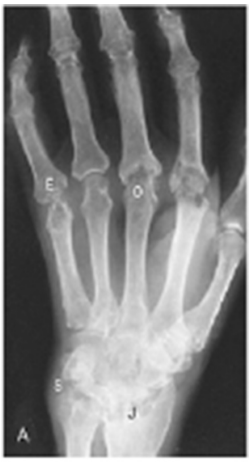
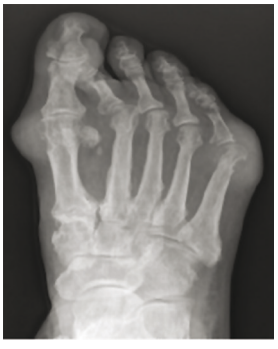
## APPENDIX 21: SALTER HARRIS CLASSIFICATION

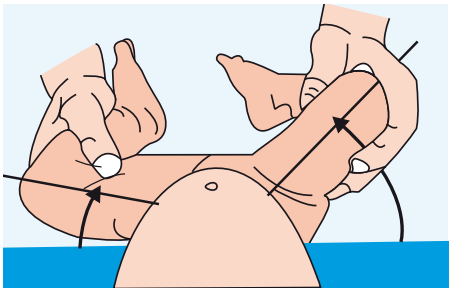
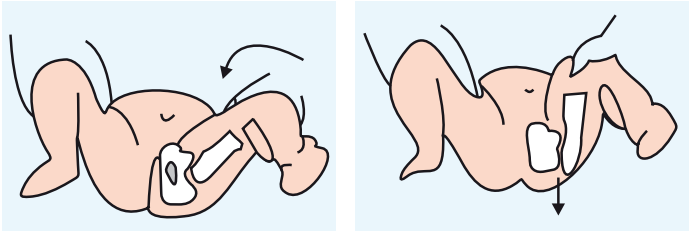
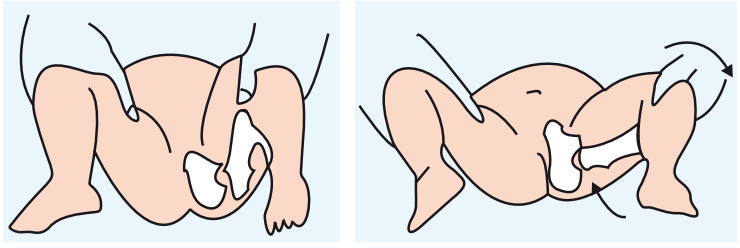
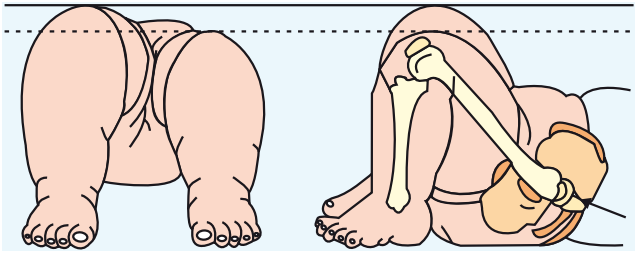
A Salter–Harris fracture is a fracture that involves the epiphyseal plate or growth plate of a bone. It is a common injury found in children, occurring in 15% of childhood long bone fractures

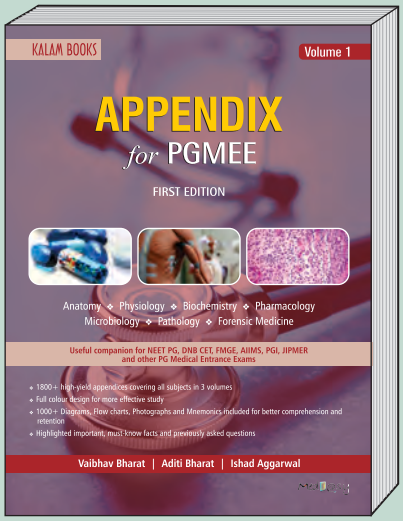
| | |
|---|---|
| Type I | Fracture through the physeal plate (often not detected radiographically) |
| Type II | Fracture through the metaphysis and physis (most common; up to 75% of all physeal fractures) |
| Type III | Fracture through the epiphysis and physis |
| Type IV | Fracture through the metaphysis, physis and epiphysis |
| Type V | Crush injury involving part or all of the physis |



## APPENDIX 33: ARTHRITIS SUMMARY

| | Comments | Type & M.C Joints involved | Less commonly involved | Spared | Nature | X ray appearance |
|---|---|---|---|---|---|---|
| **Rheumatoid Arthritis** | Generalized connective tissue disorder that selectively targets synovial tissue, particularly in the peripheral joints of the hands and feet.<br>40-50 yrs, F>M | Symmetrical polyarteritis.<br>**MC Joints:** Wrist, ***MCP, PIP,*** elbow, knee, ankle, MTP | Hip, TMJ, Subtalar, forefoot. upper cervical spine (facet jt) with atlantoaxial subluxation | Lumbar spine, DIP | Erosive-painful | -**Z deformity** (radial deviation of wrist & ulnar deviation of digits)<br>**-Swan neck deformity** (hyperextended PIP & flexion of DIP)<br>**-Boutonniere deformity** (flexion of PIP & Extension of DIP)<br>**-Wind swept deformity** (valgus of toes of one foot & varus of other)<br>-Hitch- Hiker thumb<br>-Hammer toe |
| | |  <br>The figure shows soft tissue swelling (*S*), juxta-articular osteoporosis (*O*), uniform loss of joint space (*J*), and marginal erosions (*E*). | | | | |
| **Osteoarthritis** | Degenerative athritis. **MC form of arthritis** Elderly | Assymetrical Poly> Pauci> Monoarticular.<br>**MC Joints: Knee** is the most common joint involved in OA & **Genu Varum** (Bow leg) is the most common deformity seen in OA of the knee.<br>Oher common Jts-DIP, PIP, 1st CMC, spine, Hip, Feet | Glenohumeral, Acromioclavicular, Tibiotalar, TMJ, Sacroiliac | Wrist, MP jt, MCP jt | May be erosive | -Heberdens node (DIP)<br>-Bouchard's node (PIP)<br>-Loose bodies<br>-Osteophytes<br>-Subchondral cyst & sclerosis |
| **Gout** | <u>Crystal arthropathy</u> due to deposition of monosodium urate-MSU crystals in & around the joints. >40 yrs, M>F | Monoarticular. **MC Joints: *MTP of great toe*** (MC & earliest jt known as **podagra**), tendons (not muscles) & bursae | Ankle, finger jt, olecranon | | Erosive-painful | **- <u>Joint effusion (earliest sign)</u>**<br>-Tophi of articular cartilage of small joints of hand and foot,<br>**-Martel's or G sign** (punched out cysts or deep bony erosions with overhanging margins) |
| | |  <br>Figure shows the characteristic involvement of 1st meta tarso-phalangeal joint with presence of lytic lesion with sclerotic margins & over hanging edges | | | | |

| | |
|---|---|
| **ASSYMETRICAL ABDUCTION** |  |
| | Most sensitive sign of **Developmental dysplasia of hip (DDH)** across all age groups. |
| **BARLOW TEST** |  Barlow Test |
| | **Indication:** DDH<br>**Procedure:** adducting the hip (bringing the thigh towards the midline) while applying light pressure on the knee, directing the force posteriorly.<br>**Interpretation:** If the hip is dislocatable - that is, if the hip can be popped out of socket with this maneuver - the test is considered positive |
| **ORTOLANI'S TEST** | **Indication:** DDH - to confirm Barlow's test<br>It relocates the dislocation of the hip joint that has just been elicited by the Barlow maneuver |
| |  Ortolani Test |
| | **Technique:** It is performed by an examiner first flexing the hips and knees of a supine infant to 90 degrees, then with the examiner's index fingers placing anterior pressure on the greater trochanters, gently and smoothly abducting the infant's legs using the examiner's thumbs.<br>**Interpretation:** A positive sign is a distinctive 'clunk' which can be heard and felt as the femoral head relocates anteriorly into the acetabulum |
| **GALEAZZI TEST/ ALLIS SIGN** |  |
| | **Indication:** DDH<br>**Technique:** It is performed by flexing an infant's knees in the supine position so that the ankles touch the buttocks.<br>**Interpretation:** If the knees are not level then the test is positive, indicating a potential congenital hip malformation |

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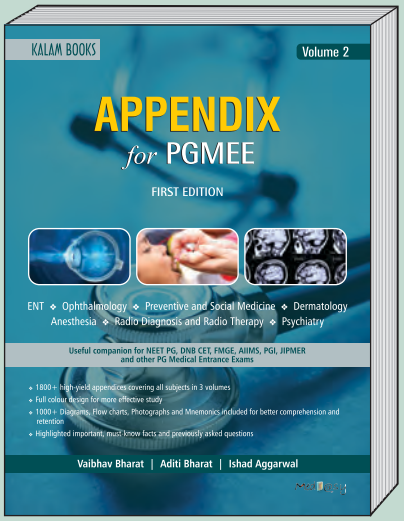


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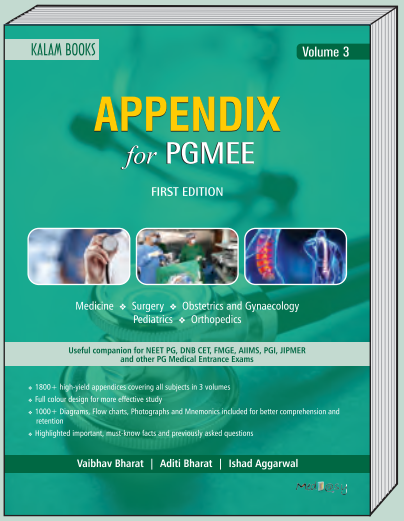


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