

APPENDIX

for PGMEE

FIRST EDITION



Medicine ❖ Surgery ❖ Obstetrics and Gynaecology
Pediatrics ❖ Orthopedics

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Vaibhav Bharat | Aditi Bharat | Ishad Aggarwal

APPENDIX

for PGMEE

Volume 3

First Edition

Dr Vaibhav Bharat

MBBS, DNB General Surgery
Director, MedE@sy

Dr Aditi Bharat

MBBS, MD Anaesthesiology (TATA Memorial Hospital, Mumbai)

Dr Ishad Aggarwal

MBBS, MD Dermatology (IPGIMER, Kolkata)

Edited by

Dr Harshvardhan Bharadwaj

MBBS, M.Med, DA

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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 juggernaut scale and precision.

With changing pattern of PGMEET 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 PGMEET
By Team MedE@sy

Sample Pages

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Sickle- β thalassemia	S β	FS	FS	0	>3.5	2 to 15	80 to 92	0
Sickle- β^+ thalassemia	S β^+	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; $\delta\beta$: 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 coproporphyrin (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

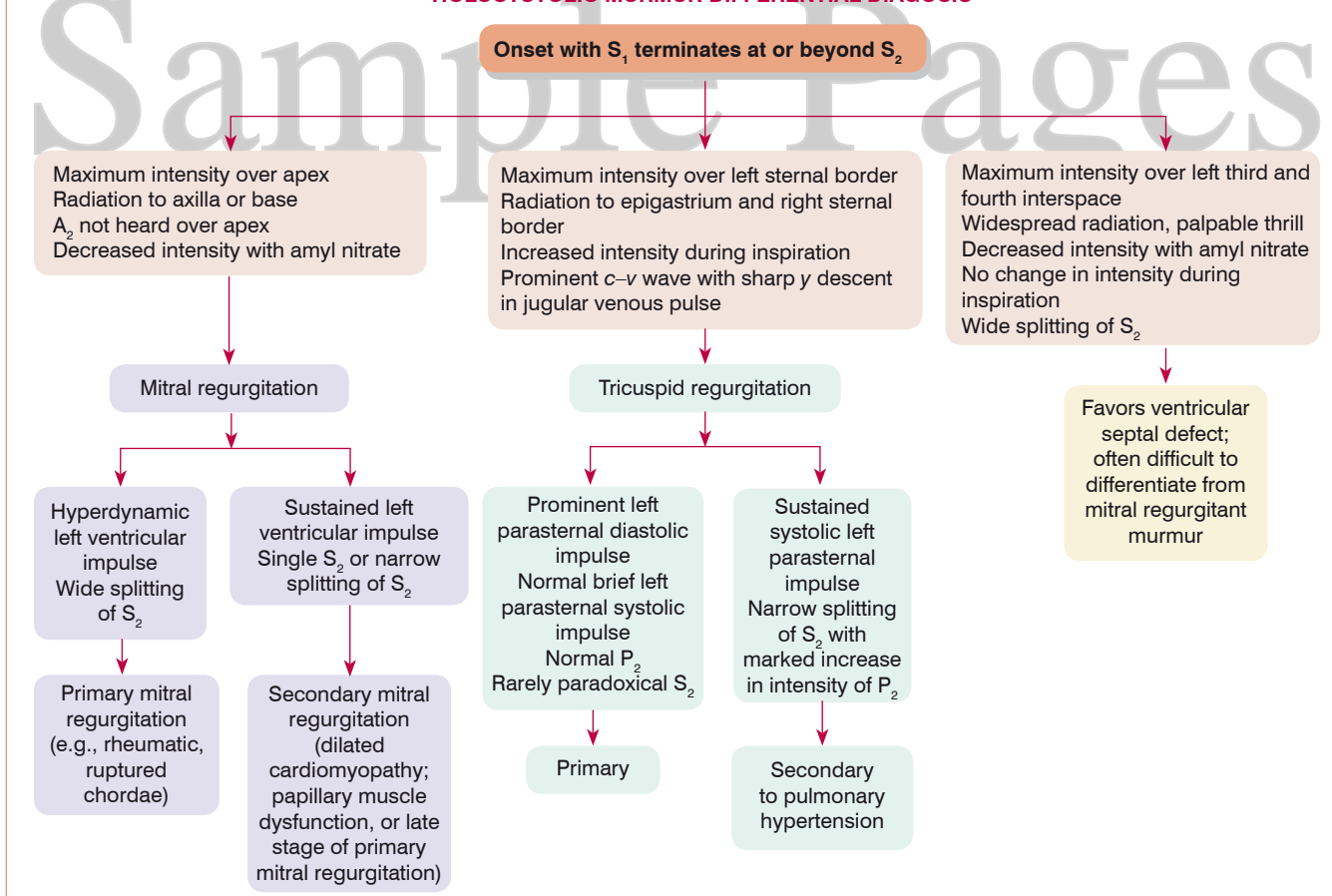
HMB synthase; also known as PBG deaminase

Abbreviations: AD, autosomal dominant; ALA, 5-aminolevulinic acid; AR, autosomal recessive; COPRO I, coproporphyrin I; COPRO III, coproporphyrin 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

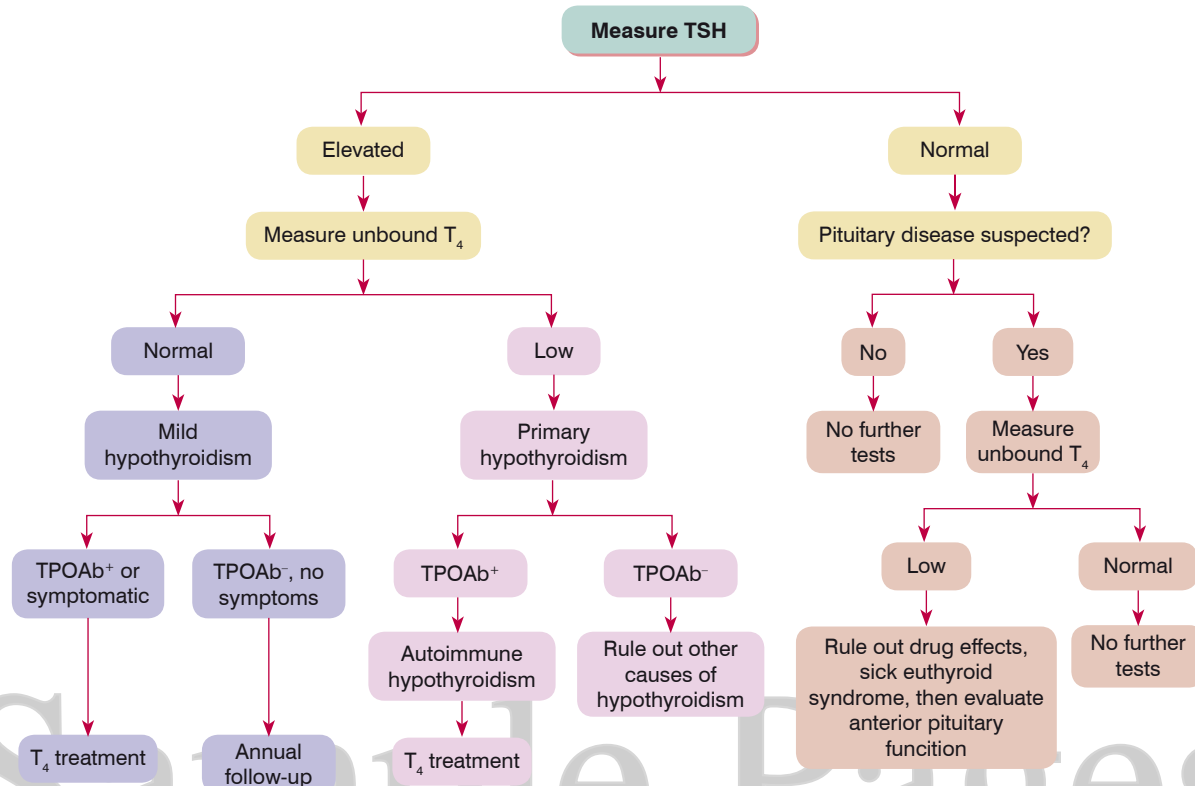
Condition	Description
Tricuspid insufficiency	Can be best heard over the 4th left sternal border. The intensity can be accentuated following inspiration (Carvallo's sign) due to increased regurgitant flow in right ventricular volume. TR is most often secondary to pulmonary hypertension. Primary tricuspid 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. S_4 is often audible in patients with <i>acute</i> 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 S_2 is normal. This fact can be used to distinguish from pulmonary stenosis, which has a wide splitting S_2 . When the shunt becomes reversed ("Eisenmenger syndrome"), the murmur may be absent and S_2 can become markedly accentuated and single.

HOLOSYSTOLIC MURMUR DIFFERENTIAL DIAGNOSIS



APPENDIX 83: HYPOTHYROIDISM

EVALUATION OF HYPOTHYROIDISM



Signs and Symptoms of Hypothyroidism (Descending Order of Frequency)

Symptoms

Tiredness, weakness
 Dry skin
 Feeling cold
 Hair loss
 Difficulty concentrating and poor memory
 Constipation
 Weight gain with poor appetite
 Dyspnea
 Hoarse voice
 Menorrhagia (later oligomenorrhea or amenorrhea)
 Paresthesia
 Impaired hearing

Signs

Dry coarse skin; cool peripheral extremities
 Puffy face, hands, and feet (myxedema)
 Diffuse alopecia
 Bradycardia
 Peripheral edema
 Delayed tendon reflex relaxation
 Carpal tunnel syndrome
 Serous cavity effusions

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 $\geq 6.5\%$ ^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

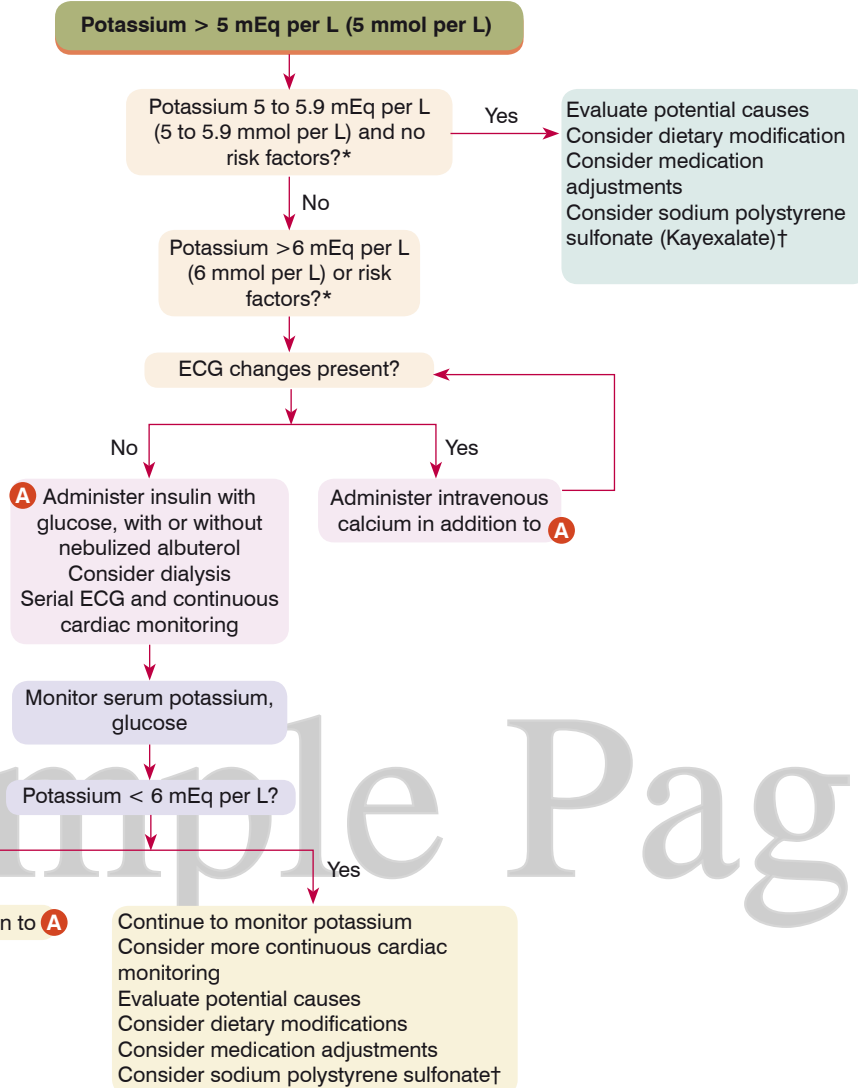
Footnote:

- ❖ 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
<ul style="list-style-type: none"> ❖ Fatigue, lack of energy ❖ Weight loss, Anorexia ❖ Myalgia, Joint pain ❖ Fever ❖ Normochromic anemia, lymphocytosis, eosinophilia ❖ Slightly increased TSH ❖ Hypoglycemia ❖ Low blood pressure, postural hypotension ❖ Hyponatremia (due to loss of feedback inhibition of AVP release) 	<ul style="list-style-type: none"> ❖ Abdominal pain, nausea, vomiting ❖ Dizziness, postural hypotension ❖ Salt craving ❖ Low blood pressure, postural hypotension ❖ Increased serum creatinine ❖ Hyponatremia ❖ Hyperkalemia 	<ul style="list-style-type: none"> ❖ Lack of energy ❖ Dry and itchy skin (in women) ❖ Loss of libido ❖ 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

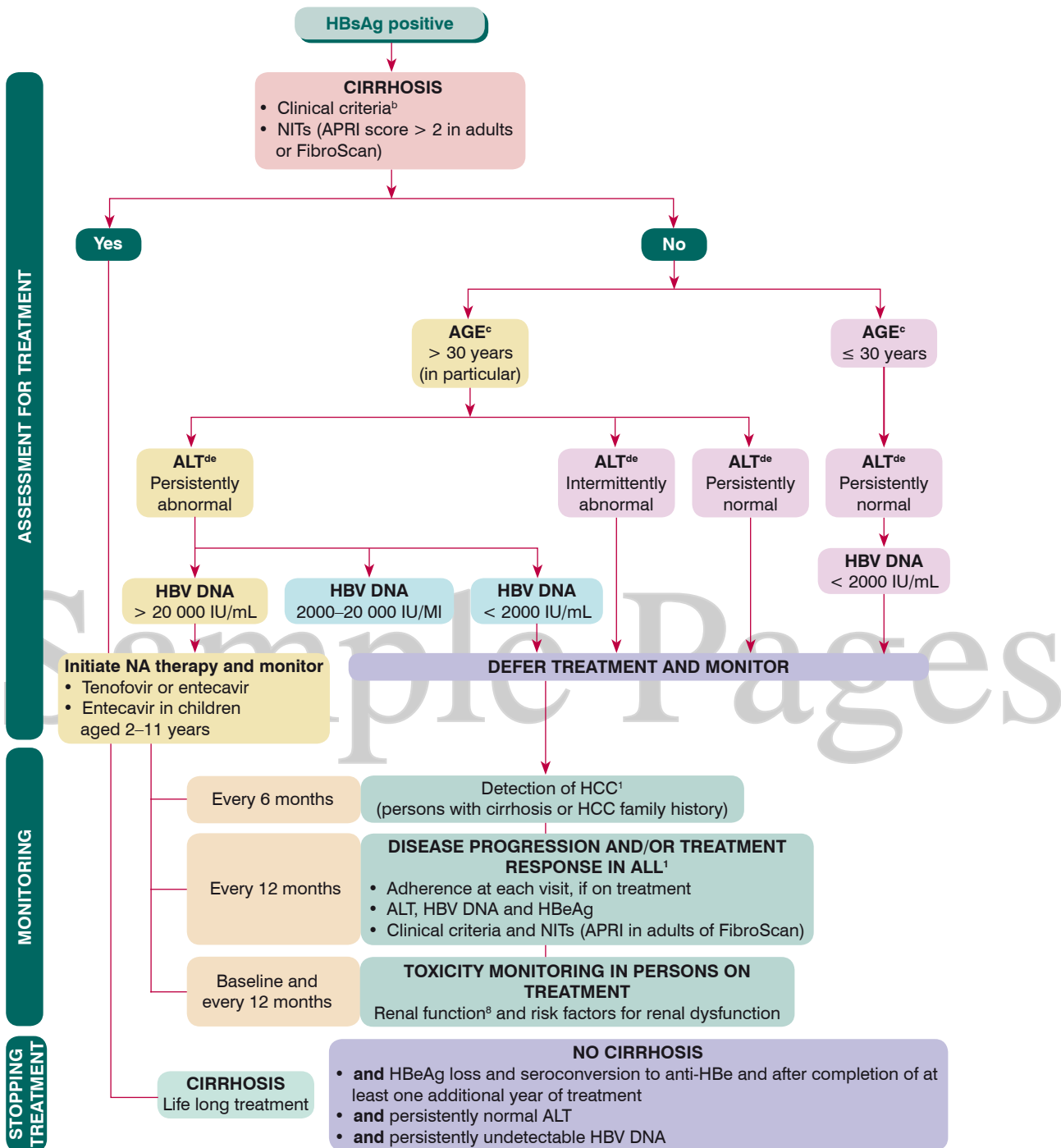
Spurious

- 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

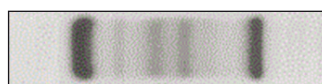
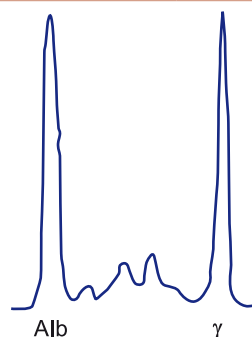
ALGORITHM OF WHO RECOMMENDATIONS ON THE MANAGEMENT OF PERSONS WITH CHRONIC HEPATITIS B INFECTION



APPENDIX 120: MULTIPLE MYELOMA

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

Epidemiology	<ul style="list-style-type: none"> Found in all race, all geographical locations Men > Women (slightly) Blacks have nearly twice the incidence of whites Disease of old adults (Mean age 70 years) 	
Clinical presentations	<ol style="list-style-type: none"> Bone pain with lytic lesions discovered on x rays or other imaging An ↑ total serum protein concentration and/or the presence of a monoclonal protein in the urine or serum Systemic signs or symptoms such as anemia, suggestive of malignancy Hypercalcemia (symptomatic or incidental) 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
	<ol style="list-style-type: none"> Anemia – 80% (Most common sign) Bone pain – 70% (Most common symptom) Elevated creatinine – 48% Fatigue/generalized weakness – 32% Hypercalcemia – 28% Weight loss – 24% 	<ol style="list-style-type: none"> Paresthesias -5% Hepatomegaly - 4% Splenomegaly - 1% Lymphadenopathy - 1% Fever - 0.7%
International Myeloma working group diagnostic criteria of Multiple Myeloma	<p>Clonal bone marrow plasma cells ≥10% or biopsy-proven bony or extramedullary plasmacytoma and any one or more of the following myeloma-defining events:</p> <ul style="list-style-type: none"> Evidence of end-organ damage that can be attributed to the underlying plasma cell proliferative disorder, specifically: <ol style="list-style-type: none"> 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) Renal insufficiency: creatinine clearance <40 mL per min or serum creatinine >177 μmol/L (>2 mg/dL) Anemia: hemoglobin value of >20 g/L below the lower limit of normal, or a hemoglobin value <100 g/L Bone lesions: one or more osteolytic lesions on skeletal radiography, CT, or PET-CT Any one or more of the following biomarkers of malignancy: <ul style="list-style-type: none"> Clonal bone marrow plasma cell percentage* ≥60% Involved:uninvolved serum free light chain ratio^o ≥100 >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
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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 Stoddart, 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

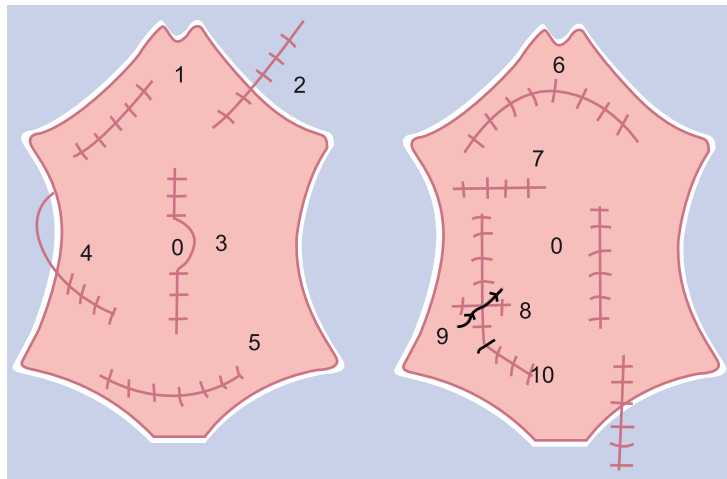


Fig: Commonly used abdominal incisions

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

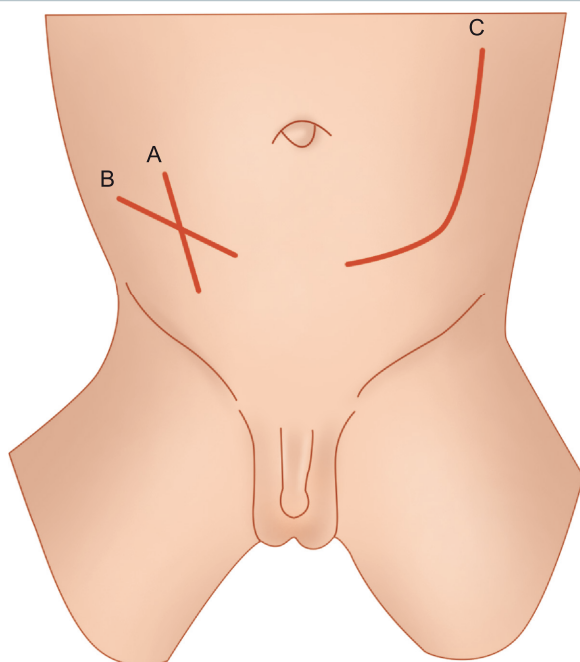


Fig: Incisions used in Open Appendectomy

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:

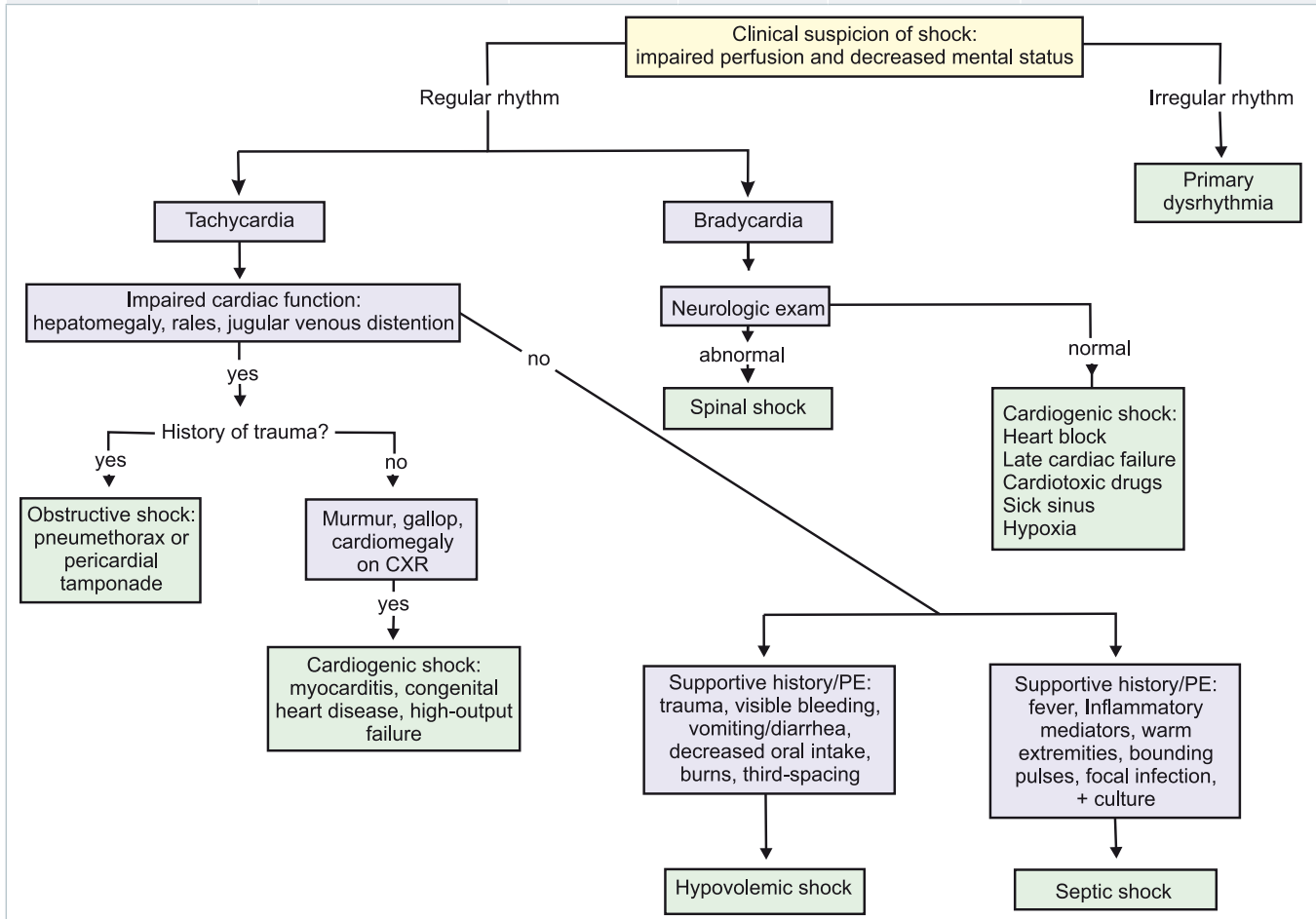
Skin
 Subcutaneous fat
 Scarpa's fascia
 External oblique aponeurosis
 Internal Oblique
 Transverse abdominis
 Fascia transversalis
 Pre peritoneal fat
 Parietal Peritoneum

APPENDIX 4: CHARACTERISTICS OF ABSORBABLE SUTURE MATERIALS

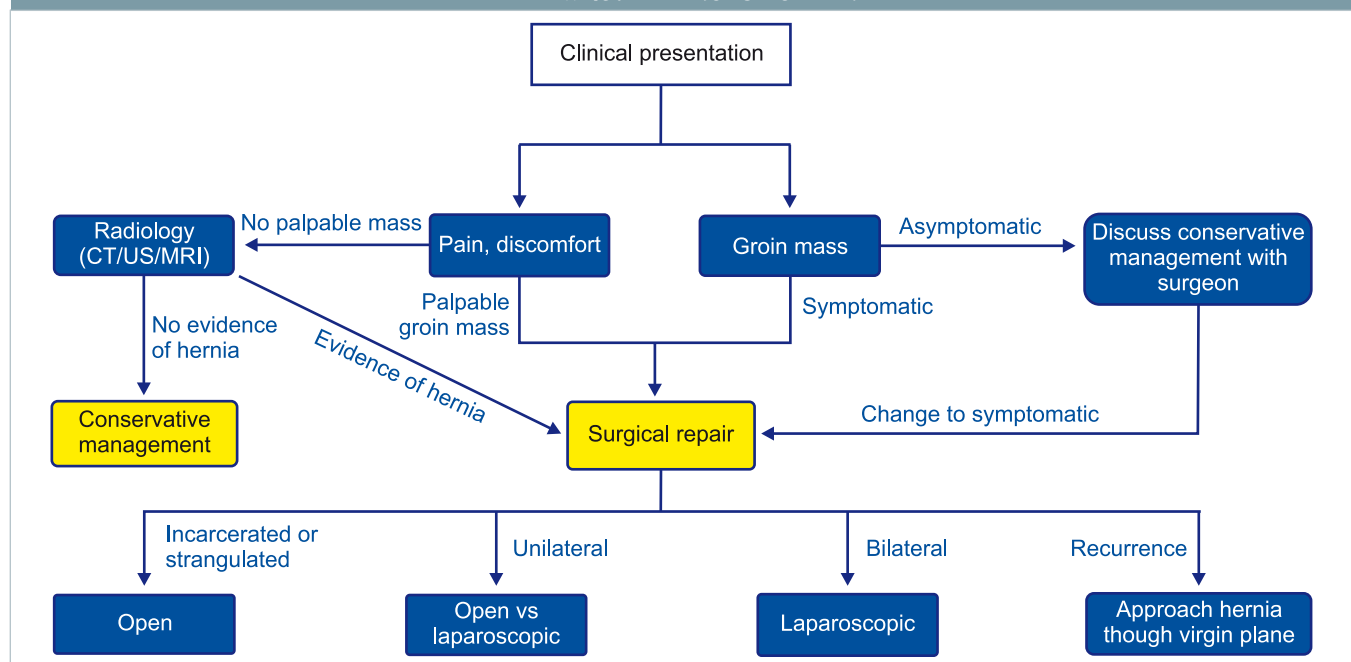
Suture (trade name)	Manufacturing process	Effective strength (d)	Complete absorption (d)	Absorption profile		Application
				Tissue reactivity	Handling	
Plain Catgut 3/0 (3 METRIC) PLAIN CATGUT STERILE LOT: 040818 MFG: 08 2004 EXP: 08 2007 75cm	Collagen from sheep intestine submucosa	4–10	70	High	Poor	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	<ul style="list-style-type: none"> ♦ 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. ♦ Epigastric hernias begin with a transverse split in the midline raphe so, in contrast to umbilical hernias, the defect is elliptical. ♦ 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	<div><p>PATHOPHYSIOLOGY OF PORTAL HYPERTENSION</p><pre>graph TD A[Obstruction to portal flow (cirrhosis/PVT/etc.)] --> B[Increased portal venous pressure] B --> C[Increased production vasoconstrictors] B --> D[Increased production vasodilators] C --> E[Increased hepatic vascular tone] E --> F[Increased hepatic vascular resistance] F --> H[Portal hypertension] D --> G[Peripheral, ↓ BP] D --> I[Splanchnic hyperemia] G --> J[Activate neurohumoral] J --> K[Na and H2O retention] K --> L[Increased C.O.] L --> M[Increased collateral flow] I --> M M --> H H -.-> A</pre></div>		
Clinical features	<ul style="list-style-type: none">◆ 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 Portal or splenic vein thrombosis Extrinsic portal vein compression Arteriovenous fistula	Intrahepatic Cirrhosis: multiple etiologies Schistosomiasis Congenital hepatic fibrosis Rare causes	Posthepatic Budd-Chiari syndrome Constrictive pericarditis
Evaluation	Endoscopy Size of varices Extent of varices Risk factor, red color signs Portal gastropathy	Imaging Doppler ultrasound CT scan HVPG and imaging Angiography	LFT Clinical: ascites, encephalopathy, jaundice, muscle wasting Laboratory data Child’s score 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
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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

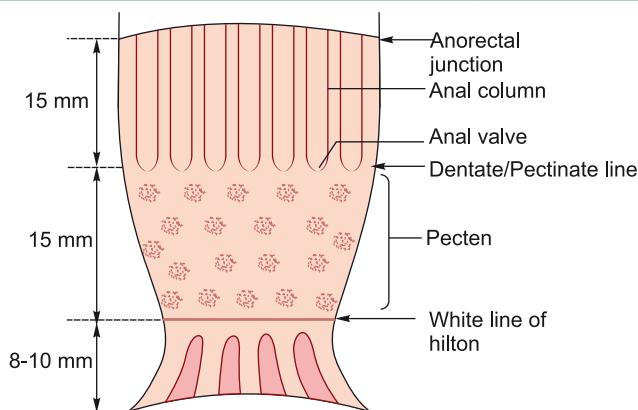
APPENDIX 109: HAEMORRHOIDS

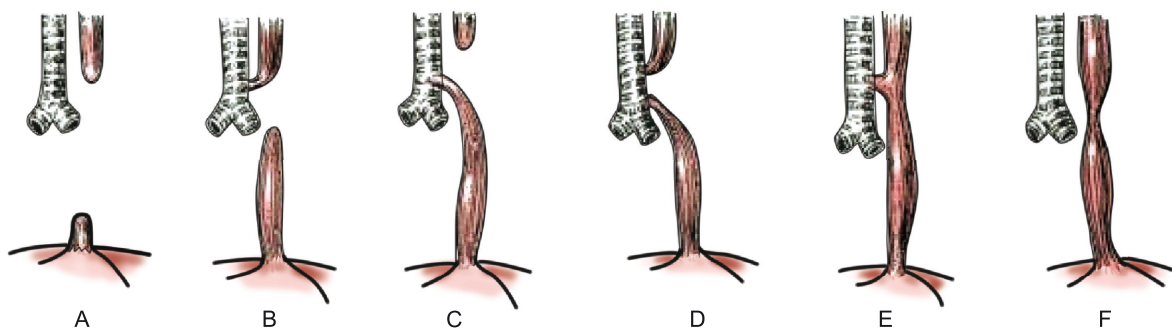
	Internal Haemorrhoids	External Haemorrhoids
	Above pectinate line	Below pectinate line
Embryological origin	Endoderm (Cloaca)	Ectoderm
Nerve supply	Visceral afferent nerves: an incision or a needle insertion in this region is painless . Sensitive only to stretching	Inferior rectal nerves -S2, S3, S4 (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

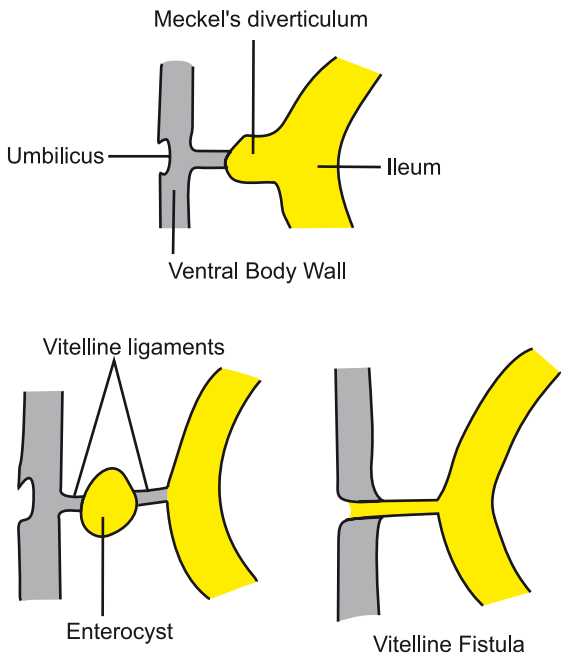




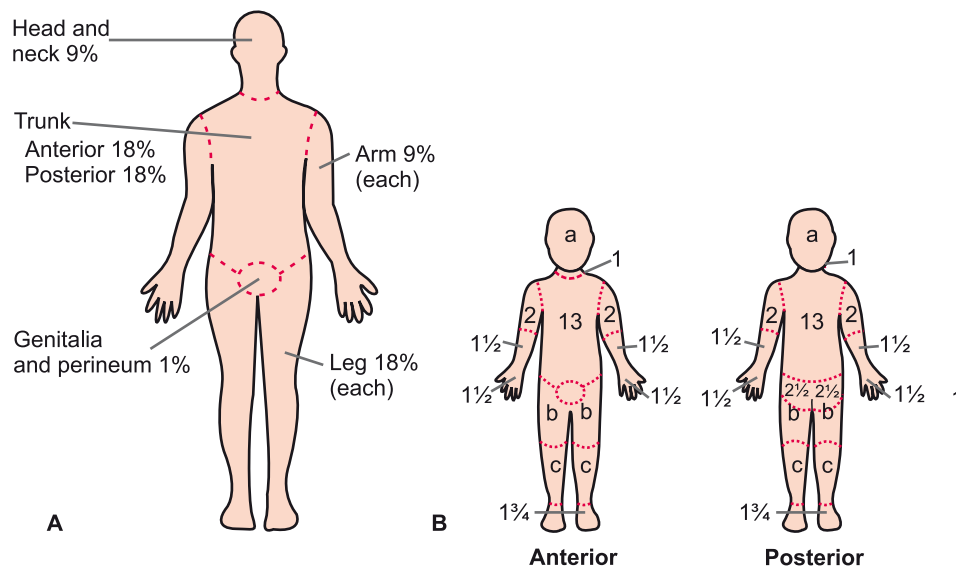
Associated Anomalies The etiology of the disturbed embryogenesis is presently unknown. Roughly one third of infants with EA or TEF have low birth weight, and two thirds of infants have associated anomalies. There is a nonrandom, nonhereditary association of anomalies in patients with EA or TEF that must 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	<ul style="list-style-type: none">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)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.

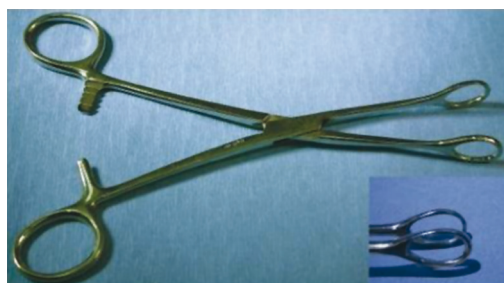
Burned children in hospitals without qualified personnel for the care of children

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

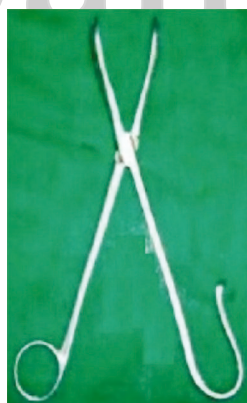


DESJARDIN'S CHOLEDOCHOLITHOTOMY FORCEPS

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

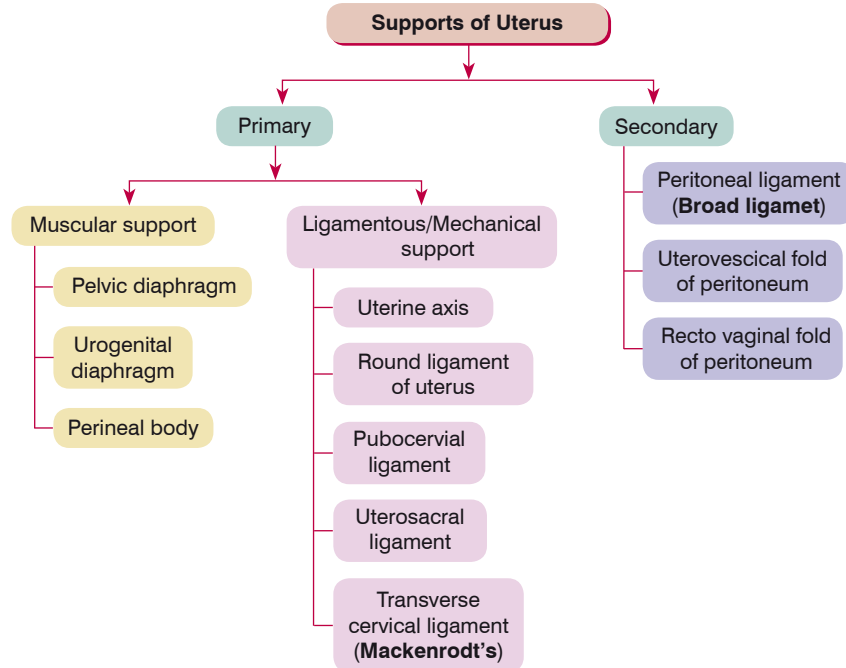
Uses: To remove bladder stones (vesicle calculus) during suprapubic cystolithotomy.



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)
- ◆ Perineal body
- ◆ Pyramidal shape
- ◆ Situated between vagina and Anal canal, about 1.25 cm in front of anus.
- ◆ 9 muscles meet here:
 - 3 unpaired**
 - External anal sphincter
 - Bulbospongiosus
 - Longitudinal muscle coat of rectal ampulla & anal canal
 - 3 Paired**
 - 2 Levator Ani
 - 2 Superficial transverse perineal muscle
 - 2 Deep transverse perineal muscle
- ◆ Urogenital diaphragm

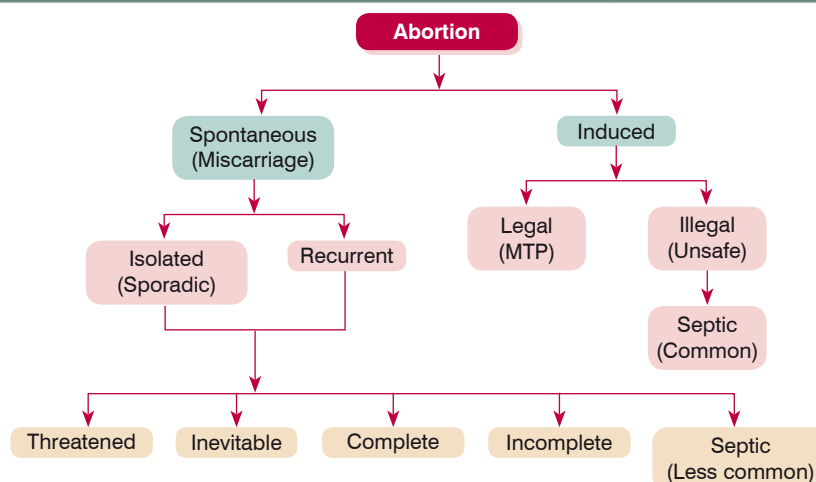
Fibromuscular or mechanical support

1. Uterine axis
2. Round ligament of uterus
- 3. Major Ligaments; Triradiate ligament**
 - a) Pubocervical ligament
 - b) Uterosacral ligament
 - c) Cardinal ligaments (or Mackenrodt's ligament/ lateral cervical ligament/ transverse cervical ligament)

Secondary Supports of Uterus

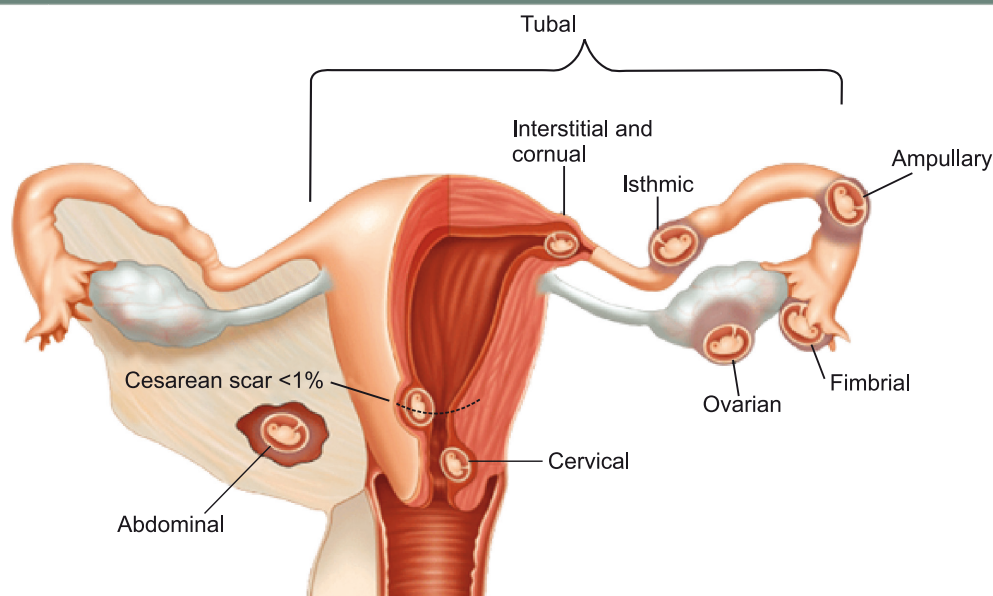
1. Broad ligament
2. Utero vaginal fold of peritoneum
3. Rectovaginal fold of peritoneum

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 hemorrhage+	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

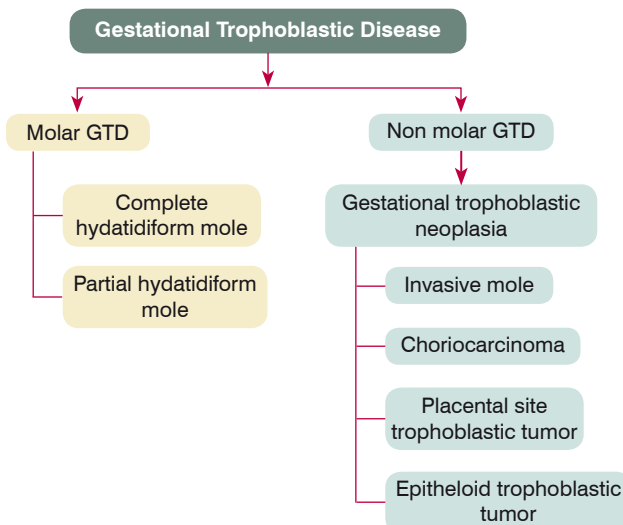
APPENDIX 68: ECTOPIC PREGNANCY



Definition	Fertilized ovum is implanted and develops outside the normal endometrial cavity.
Site	<ul style="list-style-type: none"> ♦ Isthmus: Disturbance or rupture earliest because it is the narrowest part of the tube. ♦ Interstitial: (pregnancy longest) may continue for 3-4 month. ♦ Commonest ectopic pregnancy- Tubal ♦ Rarest ectopic pregnancy- Primary Abdominal ♦ Commonest site in fallopian tube- Ampulla ♦ Least common site in fallopian tube- Interstitial part
Epidemiology	Most common in age group 20-30 yrs, Nulliparous.
Risk factors	<ol style="list-style-type: none"> 1. History of PID 2. IUD use (<i>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</i>) 3. History of tubal ligation or tubal reconstructive surgery of tubal endometriosis 4. ART particularly if the tubes are patent but damaged 5. Contraception failure 6. History of infertility 7. Previous ectopic pregnancy or Previous induced abortion
Clinical features	Classical triad: Abdominal pain (100%) + Amenorrhea (75%) + Vaginal bleeding (70%) Vomiting, fainting attack, Pallor, Features of shock, Tense tumid & tender abdomen, Adnexal mass palpable (50% of cases)
Clinical features of tubal rupture	<ul style="list-style-type: none"> ♦ Severe lower abdominal and pelvic pain that is frequently described as sharp, stabbing, or tearing. ♦ Tenderness during abdominal palpation and also a tender adnexal mass could be present. Bimanual pelvic examination, especially cervical motion, causes exquisite pain. ♦ 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- <ul style="list-style-type: none"> ♦ The gestational sac is located in the region of the ovary. ♦ The gestational sac is attached to the uterus by the ovarian ligament. ♦ Ovarian tissue is histologically proven in the wall of the gestational sac. ♦ 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

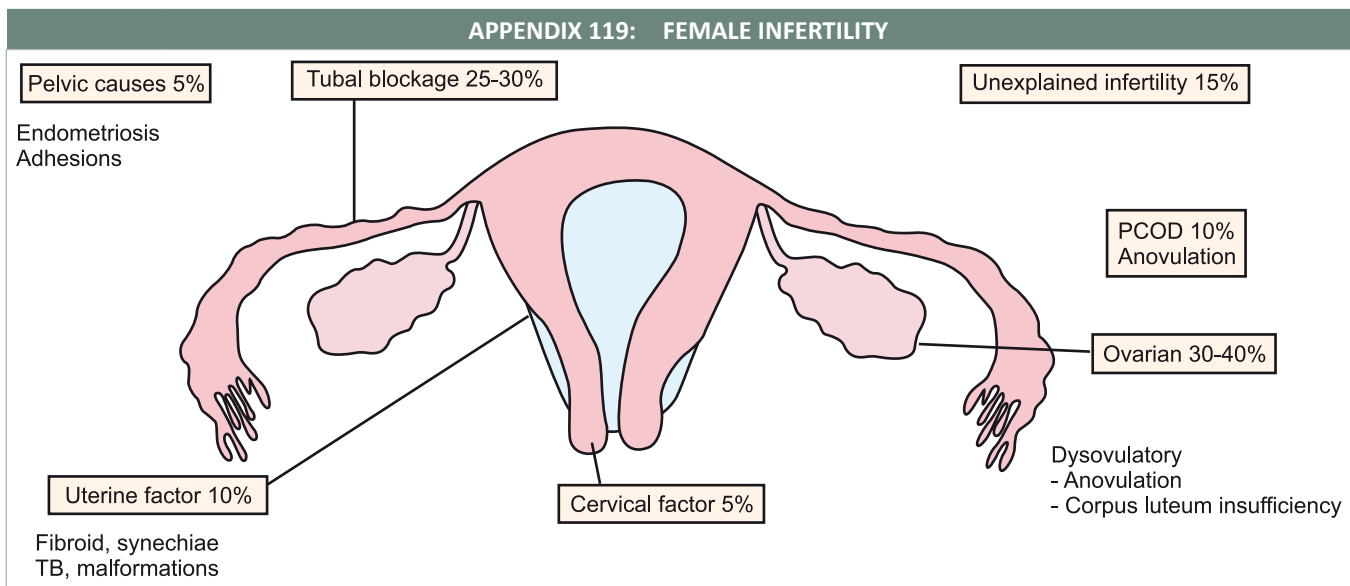
I	Non-metastatic GTD	no evidence of disease outside uterus		
II	Metastatic GTD	A. Low-risk group	1. Short duration (last pregnancy <4 months) 2. Low pretreatment HCG titre (<100 000 IU /24h urine or <40000 mIU/ml serum) 3. No metastasis in brain or liver 4. No prior chemotherapy 5. Antecedent pregnant event is not a term delivery (mole, ectopic pregnancy, spontaneous abortion)	
		B. High-risk group	1. Long duration (last pregnancy >4 months) 2. High pretreatment HCG titre (>100 000 IU /24h urine or >40000 mIU/ml serum) 3. Brain or liver metastases 4. Significant, unsuccessful chemotherapy 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	<ul style="list-style-type: none"> ◆ Infertility: Inability of a couple to conceive within 1 year. Infertility implies a ↓ in the ability to conceive and is synonymous with sub fertility. ◆ Sterility: An intrinsic inability to achieve pregnancy, whereas. ◆ Primary infertility: Those who have never conceived. ◆ Secondary infertility: Those who have conceived at some time in the past. ◆ Fecundity: Probability of achieving a live birth in 1 menstrual cycle. ◆ 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	<ul style="list-style-type: none"> ◆ Male causes 25-40% (sole primary cause 25%, Overall contributing cause 40%) ◆ Female causes- 40-55% ◆ Combined male and female factor infertility- 20% ◆ Unexplained 15-20%
Most common	<ul style="list-style-type: none"> ◆ Most common cause of female sterility- Salpingitis. ◆ 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

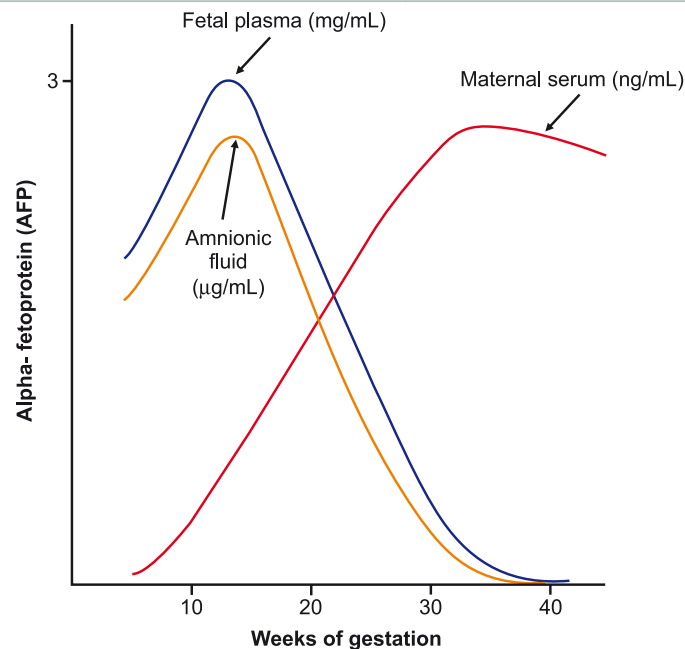
Abnormal AFP Levels

Increase

1. Wrong gestational age
2. Open neural tube defects (NTDs)
3. Multiple pregnancy
4. Esophageal or duodenal atresia
5. Ventral wall defects (Gastroschisis & Omphalocele)
6. Rh isoimmunisation
7. IUFD (Intrauterine fetal death)
8. Renal anomalies
9. Sacro-coccygeal teratoma
10. Bladder extrophy
11. Amniotic band syndrome
12. Cystic hygroma

Decrease

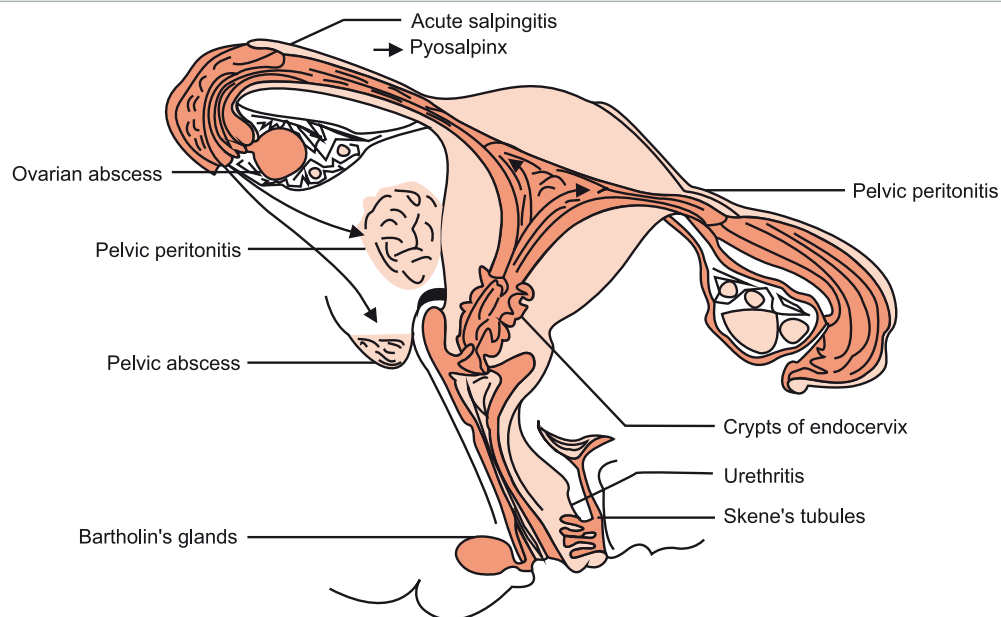
1. Obesity
2. Diabetes
3. Trisomies (Down's syndrome & Turners syndrome)
4. Gestational trophoblastic disease.
5. Over estimation gestational age
6. Missed abortion/ Fetal death
7. Gestational trophoblastic disease



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

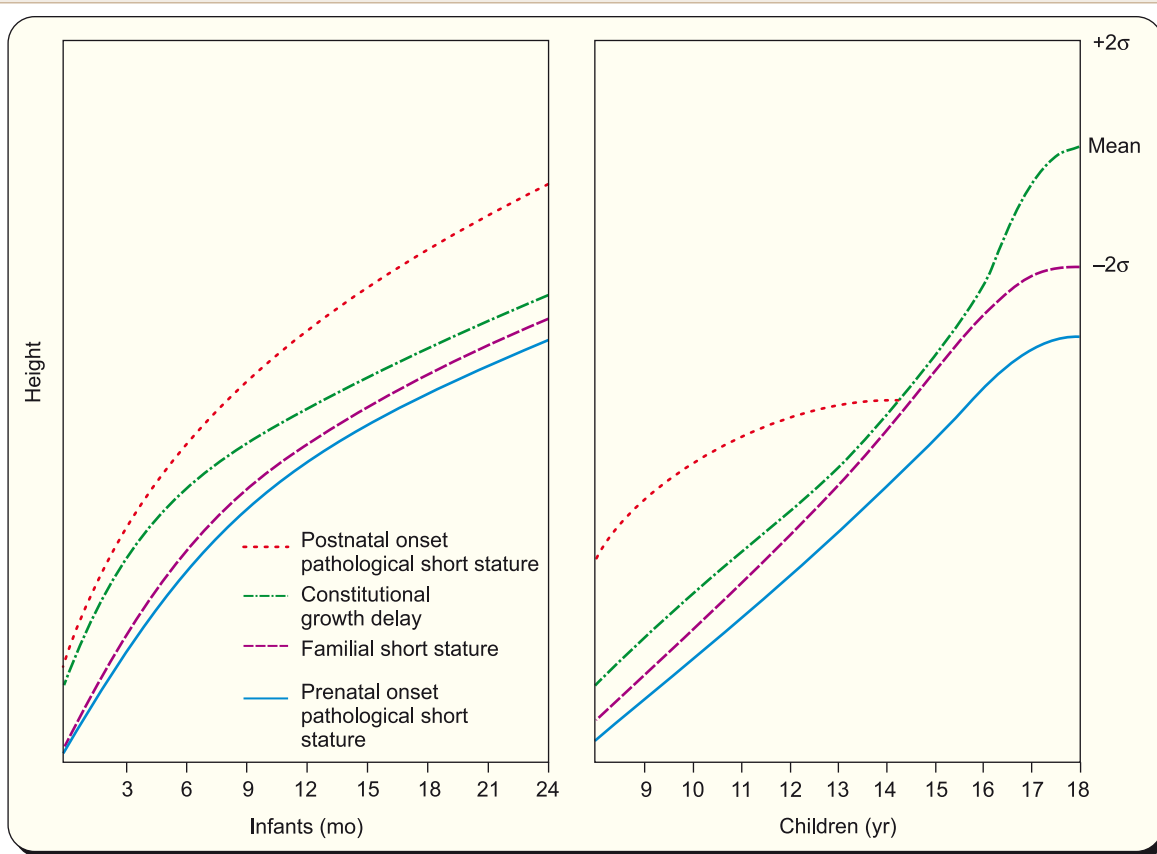
- ◆ In acute PID m/c complaint is lower abdominal pain.
- ◆ Classic triad of PID: **Pelvic pain + Cervix motion tenderness + Adnexal tenderness.**
- ◆ In chronic PID constant low abdominal pain which gets worse before menses.
- ◆ O/E '**Frozen pelvis**' is found.

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 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

Thermo - sensitivity of Vaccines	
Vaccines sensitive to heat <ul style="list-style-type: none"> • BCG (after reconstitution) • OPV • Measles • DPT • BCG (before reconstitution) • DT, TT, Hep.B, JE 	Most  Least
Vaccines sensitive to freezing <ul style="list-style-type: none"> • Hep- B • DPT • DT • TT 	Most  Least

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*** (2 nd 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

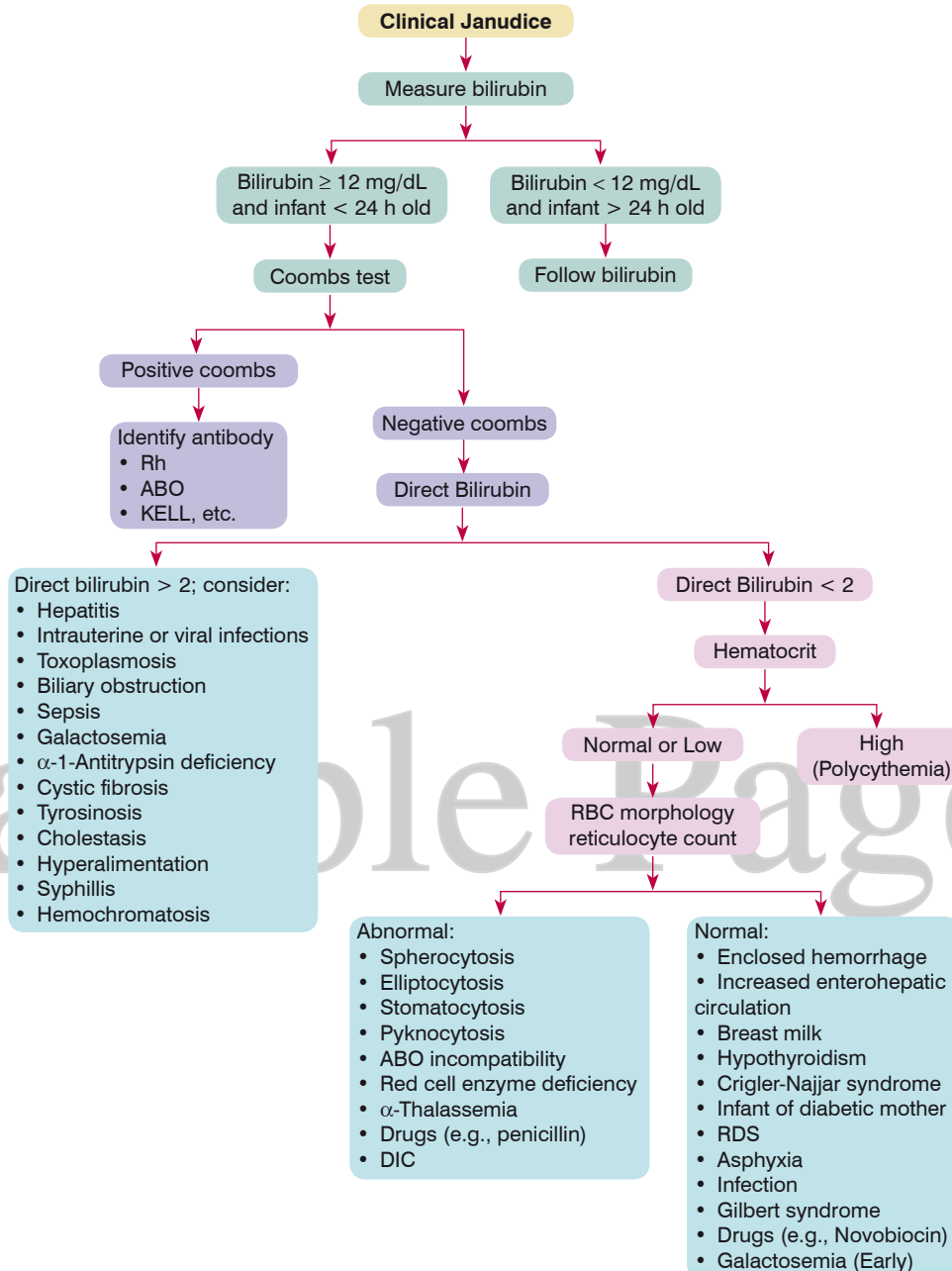
APPENDIX 63: DIAGNOSIS AND TREATMENT OF CONGENITAL ADRENAL HYPERPLASIA

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 ↑ Serum androgens	Suppression with glucocorticoids
11β-Hydroxylase deficiency	Glucocorticoid deficiency	↓ Cortisol, ↑ ACTH ↑↑ Baseline and ACTH-stimulated 11-deoxycortisol and deoxycorticosterone	Glucocorticoid (hydrocortisone) replacement
	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 ↑↑ Baseline and ACTH-stimulated Δ5 steroids (pregnenolone, 17-hydroxy pregnenolone, DHEA)	Glucocorticoid (hydrocortisone) replacement
	Mineralocorticoid deficiency (salt-wasting crisis)	Hyponatremia, hyperkalemia ↑ 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-Hydroxylase/17,20-lyase deficiency	Cortisol deficiency (corticosterone is an adequate glucocorticoid)	↓ Cortisol, ↑ ACTH ↑ DOC, corticosterone 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 Polycystic ovary syndrome/functional Ovarian hyperandrogenism Ovarian steroidogenic blocks Syndromes of extreme insulin resistance Ovarian neoplasms
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NEONATAL JAUNDICE WORKUP





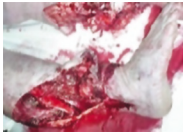


INITIATION OF THERAPY IN NEONATAL JAUNDICE

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

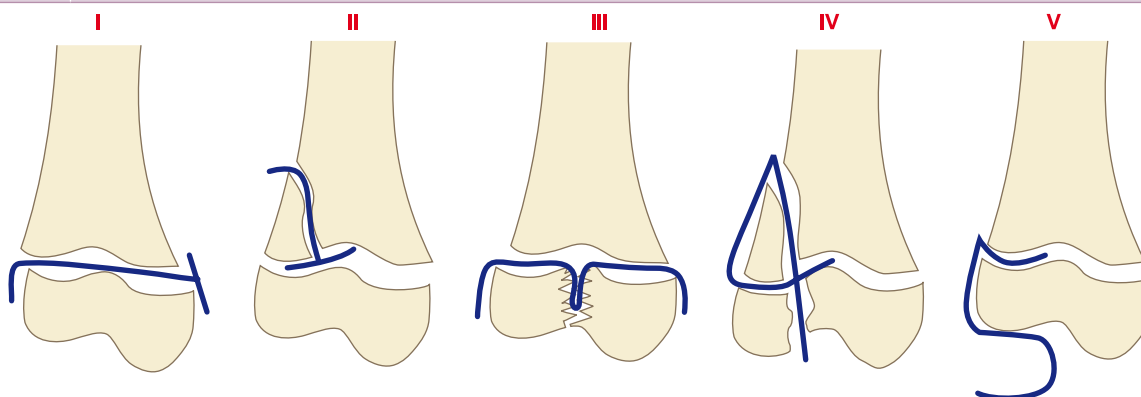
Mechanism	<ul style="list-style-type: none"> ◆ Fracture is also caused by a forced dorsiflexion of the wrist ◆ Occurs in pts > 50 years of age (Post menopausal elderly women) ◆ Fall on out stretched hand ◆ Dorsal surface undergoes compression while volar surface undergoes tension
Displacements	<ul style="list-style-type: none"> ◆ Mnemonic: SLIP (L and P comes twice, so total 6 displacements) <ol style="list-style-type: none"> 1. Supination (External rotation) 2. Lateral displacement 3. Lateral tilt/angulation 4. Impaction 5. Posterior/ Dorsal displacement 6. Posterio/ Dorsal tilt
Deformity	Dinner fork/ Silver fork/ Spoon shaped deformity
Complications	<ol style="list-style-type: none"> 1. Finger and joints stiffness is most common complication 2. Malunion is 2nd most common 3. Sudeck's osteo dystrophy (colles # is MC cause of Sudeck of upper limb) 4. Shoulder hand syndrome 5. Rupture of extensor pollicis tendon 6. Carpal tunnel syndrome 7. Carpal instability 8. Triangular fibro cartilage complex (TFCC) injury 9. Delayed and non union are rare

Treatment	<ol style="list-style-type: none"> 1. Side-arm skin traction for Type 1 injury (Dunlop traction) 2. Overhead skeletal traction for Type 1 injury 3. CRPP (closed reduction percutaneous pinning) for type 2 injury 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	<ol style="list-style-type: none"> 1. Brachial artery injury (earlier 5%, now a days < 1%) - perform angiography, or doppler 2. Compartment syndrome (uncommon) 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 4. Neuropraxia—is reported to occur in 3% to 22%
Late complications	<ol style="list-style-type: none"> 1. Malunion 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. 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. 4. Tardy ulnar nerve palsy (Not due to supracondylar fracture per se but its complication as valgus) 5. Elbow stiffness and myositis ossificans
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

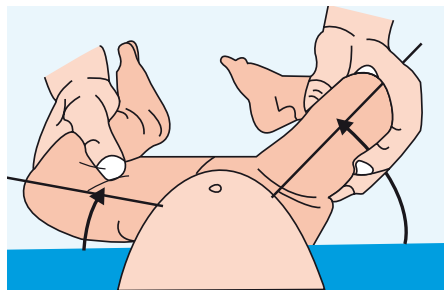
A Salter–Harris fracture is a fracture that involves the epiphyseal plate or growth plate of a bone. It is a common injury found in children, occurring in 15% of childhood long bone fractures

Type I	Fracture through the physal plate (often not detected radiographically)
Type II	Fracture through the metaphysis and physis (most common; up to 75% of all physal 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

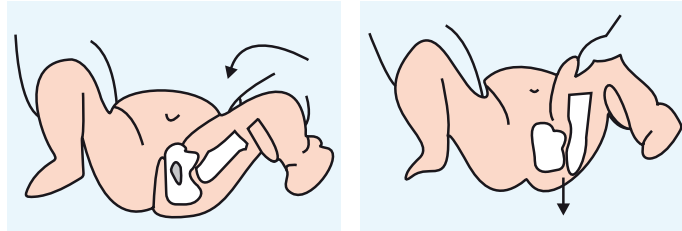


APPENDIX 33: ARTHRITIS SUMMARY

	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. 40-50 yrs, F>M	Symmetrical polyarthritis. 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) - Swan neck deformity (hyperextended PIP & flexion of DIP) - Boutonniere deformity (flexion of PIP & Extension of DIP) - Wind swept deformity (valgus of toes of one foot & varus of other) -Hitch- Hiker thumb -Hammer toe
		 <p>The figure shows soft tissue swelling (S), juxta-articular osteoporosis (O), uniform loss of joint space (J), and marginal erosions (E).</p>				
Osteoarthritis	Degenerative arthritis. MC form of arthritis Elderly	Assymetrical Poly> Pau-ci> Monoarticular. 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. Other 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) -Bouchard's node (PIP) -Loose bodies -Osteophytes -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	- Joint effusion (earliest sign) -Tophi of articular cartilage of small joints of hand and foot, - Martel's or G sign (punched out cysts or deep bony erosions with overhanging margins)
		 <p>Figure shows the characteristic involvement of 1st meta tarso-phalangeal joint with presence of lytic lesion with sclerotic margins & over hanging edges</p>				

ASYMMETRICAL ABDUCTION

Most sensitive sign of **Developmental dysplasia of hip (DDH)** across all age groups.

BARLOW TEST

Barlow Test

Indication: DDH

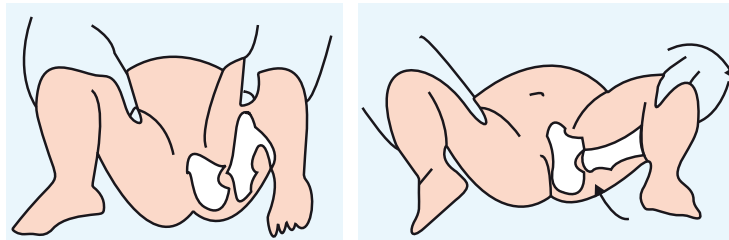
Procedure: adducting the hip (bringing the thigh towards the midline) while applying light pressure on the knee, directing the force posteriorly.

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

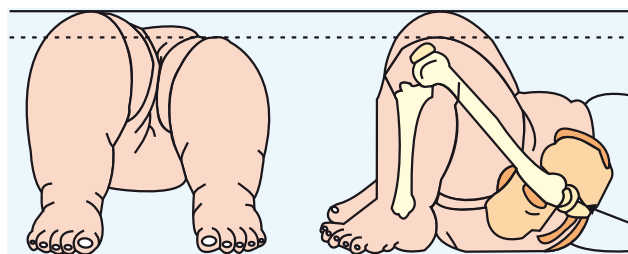
It relocates the dislocation of the hip joint that has just been elicited by the Barlow maneuver



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.

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

Technique: It is performed by flexing an infant's knees in the supine position so that the ankles touch the buttocks.

Interpretation: If the knees are not level then the test is positive, indicating a potential congenital hip malformation



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