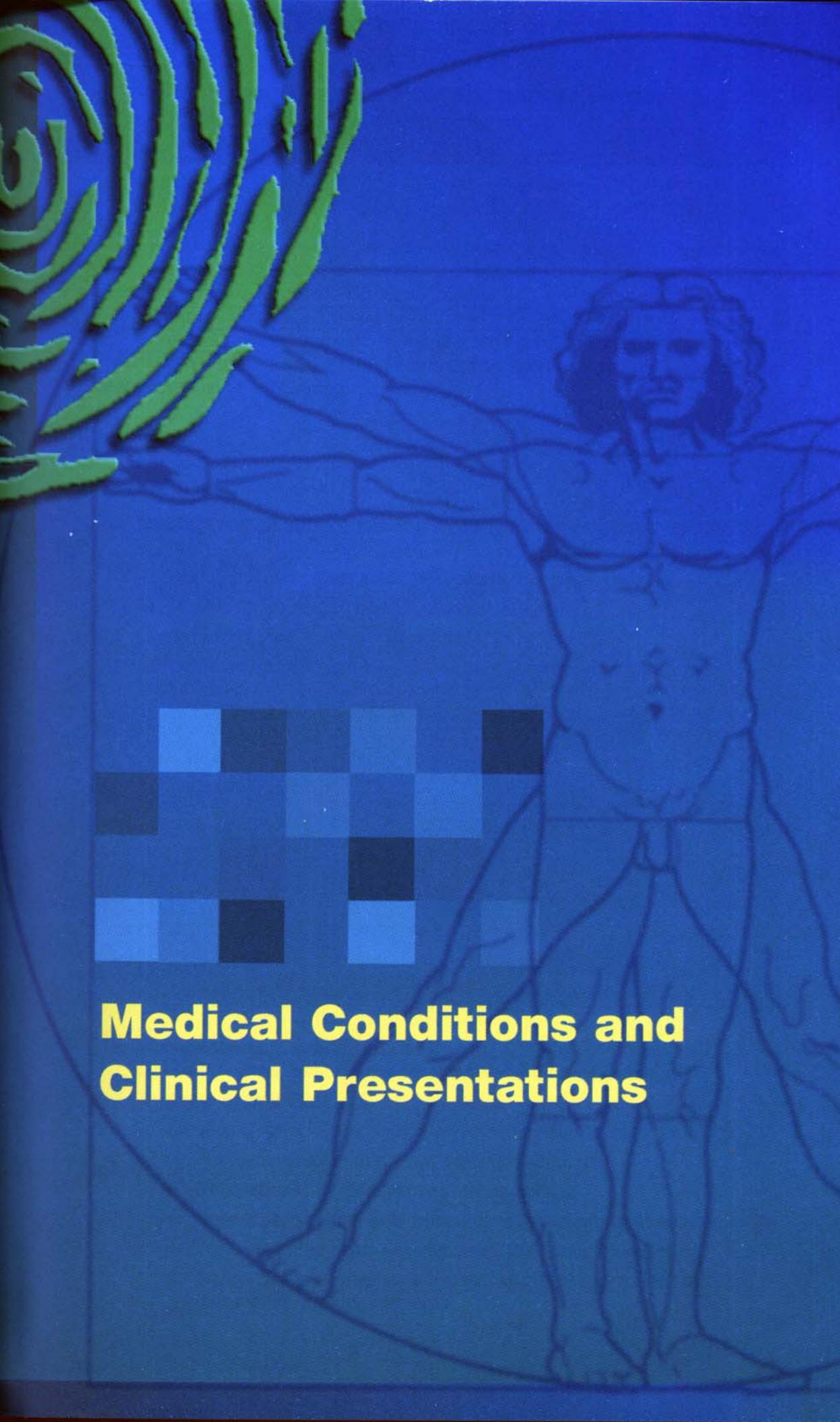




Anthology of Medical Conditions

- Integrated multidisciplinary index of clinical conditions and presentations
- Guidelines for clinical problem solving
- Legal, ethical and organisational aspects of clinical medicine



Medical Conditions and Clinical Presentations

001 Abdominal Distension/Ileus (Ascites, Bowel Obstruction)

Overview

Abdominal distension is common and may indicate the presence of serious intra-abdominal or systemic disease. Clinically, distension is separable into two main types of presentation: **Acute** distension (usually painful and predominantly surgical), or **Chronic/Subacute** distension (usually painless and associated with obstetrical/gynaecological and medical causes). Remember all the 'F's, which cover many of the causes over both groups: **Fluid, Flatus, Faeces, Fat, Fetus**, large **Focal** masses, and the **False** impressions of pseudopregnancy.

Causes

1) Acute abdominal distension

a) Mechanical intestinal obstruction (luminal, wall, extrinsic – usually painful)

The most common causes are **adhesions, tumours, hernias, volvulus**

- Gastric outlet obstruction – 'pyloric' stenosis
- Small intestine – adhesions, hernias, tumours etc.
- Large intestine – tumours, diverticular disease, volvulus



Abdominal distension – pyloric stenosis (adult)

b) Non-mechanical intestinal obstruction

(acute intestinal pseudo-obstruction – sometimes painless)

- Acute gastric dilatation
- Small and large intestine – 'paralytic' or 'spastic' ileus
 - Transient postoperative physiological ileus
 - Secondary to peritonitis from any cause
 - Secondary to ischaemia (mesenteric infarction, ischaemic enteritis)
 - Toxic inflammatory bowel disease (IBD) (toxic megacolon)
 - Secondary to severe systemic illness or retroperitoneal pathology (haemorrhage, tumours, etc.)

2) Chronic abdominal distension (usually painless)

- a) Ascites
- b) Faecal impaction
- c) Large abdominal or pelvic mass
 - Hepatosplenomegaly
 - Neoplasms
 - Abdominal aneurysms
 - Ovarian cysts
 - Gross bladder distension
- d) Pregnancy (exclude first in females)
- e) Subjective (false pregnancy, irritable bowel syndrome, obesity)



Abdominal distension – ascites

3) Chronic nonmechanical intestinal pseudo-obstruction

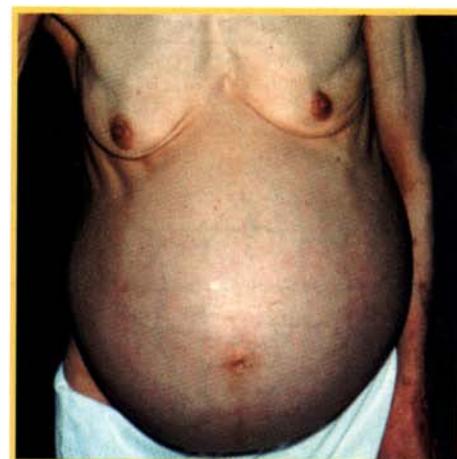
- a) Enteric nervous system (amyloidosis, diabetes, paraneoplastic)
- b) Extrinsic nervous system (stroke, spinal cord injury, multiple sclerosis, Parkinson disease, autonomic dysfunction)
- c) Smooth muscle (scleroderma)

Key Objectives

- Differentiate between causes of abdominal distension based on history and physical findings.
- Identify causes of acute abdominal distension requiring urgent early care.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate clinically the aetiology of abdominal distension.
 - Elicit information on risk factors which would predispose to the various causes for abdominal distension.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret abdominal X-rays and other appropriate investigations used in cases of abdominal distension.
 - Recommend paracentesis when indicated and interpret the results.
- Conduct an effective plan of management for a patient with abdominal distension/ileus:
 - Outline the short term medical and surgical management of patients with gaseous distension.
 - Contrast the immediate and long term management of a patient with cirrhotic ascites versus malignant ascites.
 - Select patients in need of specialised care.



Abdominal distension – ovarian cyst

Overview

Most abdominal masses represent significant underlying disease requiring complete investigation. Intra-abdominal masses must be differentiated from abdominal wall masses. The large number of possible causes can be made more manageable by focusing on the site and physical characteristics of the mass. (See #002A Epigastric Mass – #002F Left Iliac Fossa Mass.)

Causes

1) Organomegaly

- a) Hepatomegaly
- b) Splenomegaly
- c) Enlarged kidneys
- d) Retroperitoneal masses

2) Gastrointestinal system masses

- a) Gastric, colonic, appendiceal
- b) Stool
- c) Pancreatic (pseudocyst)
- d) Gallbladder mass

3) Vascular (abdominal aortic aneurysm)

4) Gynaecologic / Pelvic

- a) Pregnant uterus
- b) Ovarian mass
- c) Enlarged bladder

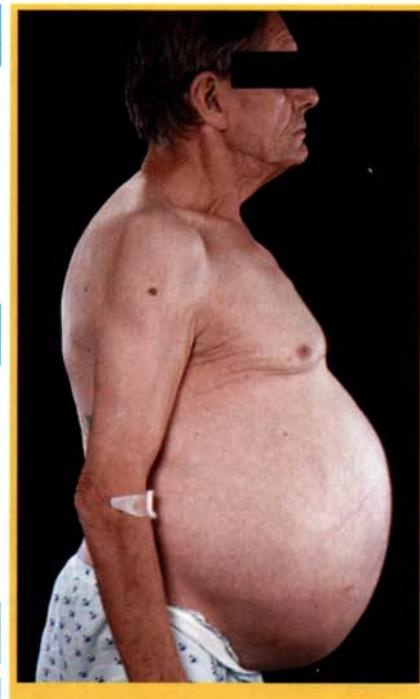
5) Lymphadenopathy

- a) Lymphomas
- b) Metastases
- c) Infectious causes

6) Masses in abdominal wall

(see #002G Abdominal Hernia)

- a) Rectus sheath haematoma
- b) Desmoid tumour
- c) Umbilical lump/discharge



Retroperitoneal liposarcoma

- Intertrigo, concretions, granulomas
- Malignant nodule (Sister Mary Joseph nodule)
- Pilonidal sinus
- Endometriosis
- Embryological remnants (vitelline duct, urachus)

Key Objectives

- Distinguish the cause of an abdominal mass based on history and physical findings.
- Distinguish intra-abdominal from abdominal wall masses.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Exclude pregnancy as cause of the abdominal mass.
 - Determine which patients are likely to have a neoplasm causing the abdominal mass.
 - Determine the physical characteristics enabling diagnosis of masses arising in specific organs.
 - Describe the risk factors which would predispose to the various causes for abdominal mass.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret abdominal imaging (radiography, computed tomography (CT) scan, ultrasound, etc.) in patients with an abdominal mass.
 - Interpret and discuss the role of serum tumour marker testing.
- Conduct an effective plan of management for a patient with an abdominal mass:
 - Discuss the medical and surgical management of patients with an abdominal mass.
 - Select patients in need of specialised care.

002A Epigastric Mass

Overview

Epigastric masses are most commonly due to pathology in stomach or liver; or may indicate retroperitoneal pathology (aneurysm, pancreatic or lymph node mass).

Causes

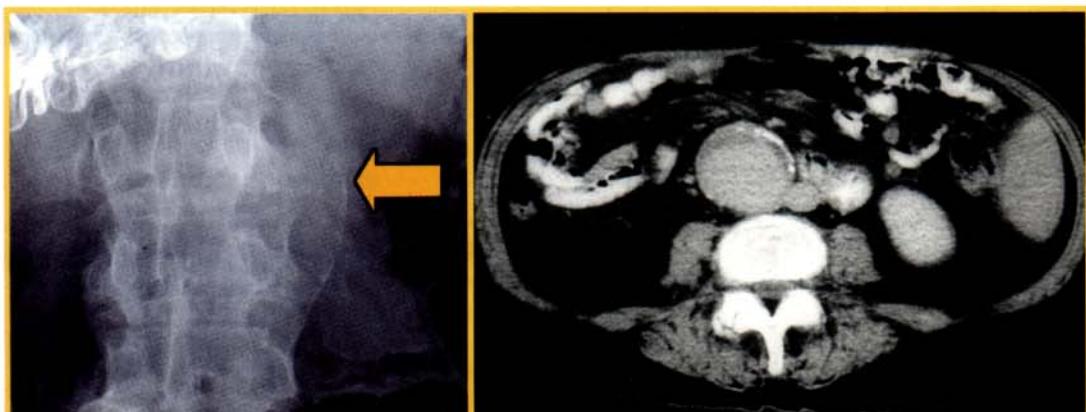
- 1) **Abdominal aortic aneurysm**
- 2) **Gastric mass (neoplasm, gastric dilatation)**
- 3) **Liver mass (left lobe)**
- 4) **Pancreatic mass (pseudocyst, tumour)**
- 5) **Lymph node mass (para-aortic nodes)**

Key Objective

- Distinguish the cause of an epigastric mass based on history and physical findings.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Identify site of origin and likely cause of an epigastric mass based on clinical findings.
- Interpret the critical clinical and laboratory findings in diagnosis.
- Outline an effective management plan for a patient with an epigastric mass.



Aortic aneurysm

002B Right Hypochondrial Mass

Overview

Masses in the right hypochondrium arise most commonly from the liver or gallbladder. The normal liver is impalpable, except in children. The liver becomes palpable when abnormally firm and enlarged or when it contains a mass.

Causes

1) Hepatomegaly

a) General enlargement

- Cirrhosis
- Heart failure

b) Focal masses

- Neoplasms, parasitic cysts, etc.

2) Enlarged gallbladder

a) Acute cholecystitis

b) Mucocele / Empyema

c) Courvoisier sign and law (underlying pancreatic neoplasm)

3) Hepatic flexure colonic mass

4) Right renal mass



Daughter cysts in hepatic hydatid

Key Objective

- Distinguish the cause of a right hypochondrial mass based on history and physical findings.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Identify site of origin and likely cause of a right hypochondrial mass.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis.
- Outline an effective diagnostic/management plan for a patient with a right hypochondrial mass.
- Outline the management of a patient presenting with a mass due to acute cholecystitis.
- Outline the management of a patient with a mass in the liver due to a hydatid cyst:
 - Describe the life cycle of the hydatid parasite through definitive (primary) and intermediate host cycles; describe the types of clinical presentation and methods of diagnosis and treatment of hydatid cyst disease in humans.



Primary hepatocellular carcinoma

002C Left Hypochondrial Mass

Overview

Masses in the left hypochondrium are most commonly caused by splenic enlargement, which must be differentiated from renal, colonic or pancreatic tail lesions.

Causes

- 1) Splenomegaly
- 2) Left renal mass
- 3) Colonic mass (splenic flexure)
- 4) Pancreatic tail mass

Key Objective

- Distinguish the cause of a left hypochondrial mass based on history and physical findings.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate splenic from other masses.
- Outline an effective management plan in a patient with a left hypochondrial mass due to splenomegaly.
- Outline effective diagnostic and management plans in patients with a left renal mass.



Wilms tumour

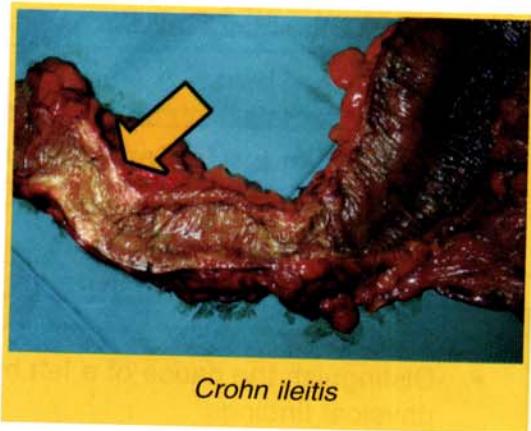
002D Right Iliac Fossa Mass

Overview

Painful right iliac fossa masses commonly are associated with appendiceal or colonic pathology and require prompt surgical assessment.

Causes

- 1) Appendiceal mass (phlegmon or abscess)**
- 2) Caecal mass (carcinoma)**
- 3) Other infectious/inflammatory causes**
 - a) Crohn disease
 - b) Actinomycosis
 - c) Ileocaecal tuberculosis (TB)
 - d) Psoas abscess
- 4) Iliac lymph node swellings**
 - a) Lymphoma
 - b) Metastatic



Key Objective

- Distinguish the cause of a right iliac fossa mass based on history and physical findings.

General/Specific Objectives

- Outline a management plan for a patient presenting with a right iliac fossa mass.
- Select and interpret imaging investigations (radiography, computed tomography (CT) scans, ultrasound, etc.) in patients presenting with a right iliac fossa mass.

002E Suprapubic/Pelvic Mass

Overview

In women a pregnant uterus must first be excluded. Other gynaecologic causes include large ovarian or uterine abdominopelvic masses. In both sexes, a distended bladder is an important cause.

Causes

1) Pregnant uterus

2) Urinary bladder

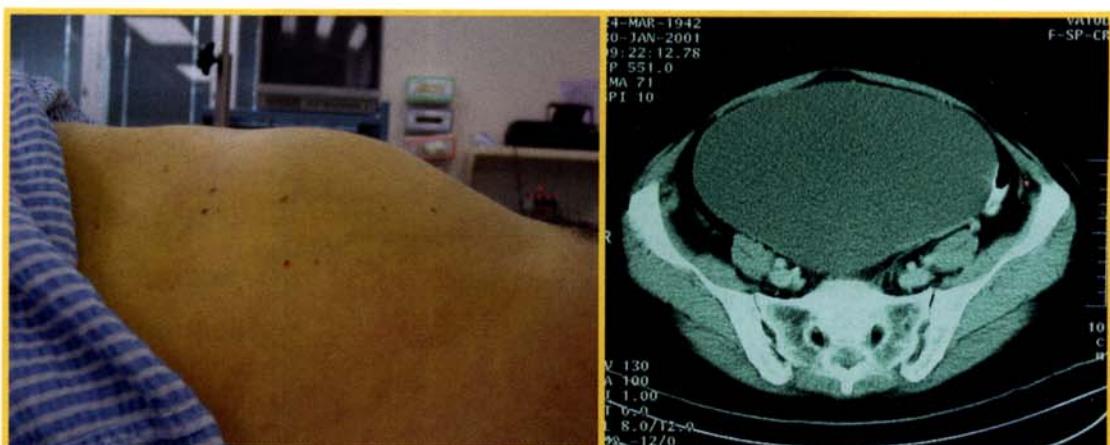
3) Ovarian mass

Key Objective

- Distinguish the cause of a suprapubic mass on the basis of history and physical findings.

General/Specific Objective

- Outline effective diagnostic/management plans for a patient with a suprapubic mass.



Suprapubic/pelvic mass – ovarian cyst

002F Left Iliac Fossa Mass

Overview

Left iliac fossa masses are most commonly due to significant colonic pathology.

Causes

1) Colonic mass

- a) Carcinoma sigmoid colon
- b) Colonic diverticular disease
- c) Faeces

2) Lymph node mass

Key Objective

- Distinguish the cause of a left iliac fossa mass based on history and physical findings.

General/Specific Objectives

- Outline effective diagnostic/management plans for a patient with a left iliac fossa mass.
- Outline the diagnostic plans for a patient presenting with left iliac fossa mass due to iliac lymph node enlargement.



Diverticular disease

002G Abdominal Hernia

Overview

Herniorrhaphy is the commonest surgical procedure performed by general surgeons. Twenty-five per cent of males will develop an inguinal hernia in their lifetime. Interference with the blood supply of the hernial contents (strangulation) is a surgical emergency.

Causes

1) Groin hernias

- a) Inguinal hernia (direct, indirect)
 - Commoner in males
- b) Femoral hernia
 - Commoner in females



Indirect inguinoscrotal
inguinal hernia

2) Umbilical hernias

- a) Congenital umbilical hernia of infancy
 - Most settle spontaneously
- b) Adult umbilical/para-umbilical hernia
 - Multiparity in females major causal factor

3) Incisional (wound) hernia

- a) Commoner with vertical incisions
- b) Wound infection commonest cause

4) Less common abdominal wall hernias

- a) Epigastric hernia (fatty hernia of linea alba)
- b) Spigelian (linea semilunaris) hernia
 - Characteristically interstitial
- c) Lumbar hernia
- d) Perineal hernia (usually after abdomino-perineal excision of rectum)



Bilateral inguinal hernias

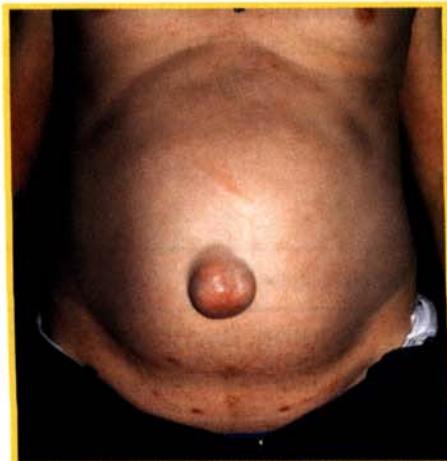
5) Diverication of recti

- a) Diffuse epigastric bulge between recti in epigastrium due to obesity and spreading of linea alba – not a true hernia

6) Internal abdominal hernias



Femoral hernia



Para-umbilical hernia

Key Objectives

- Identify abdominal wall hernias requiring immediate rather than elective repair.
- Recognise factors predictive of hernia recurrence postoperatively (such as wound sepsis, obesity, ascites and malnutrition).

General/Specific Objectives

- Through efficient, focused data gathering:
 - Recognise symptoms and signs of strangulated hernia, as opposed to irreducibility.
 - Differentiate inguinal and femoral hernias from other causes of a groin (or inguinoscrotal) mass such as lymphadenopathy, varicocele, hydrocele, saphena varix or femoral aneurysm.
 - Differentiate inguinal from femoral hernia on the basis of physical signs including visual inspection, palpation and special manoeuvres.
- Conduct an effective diagnostic and management plan for a patient with an abdominal wall hernia:
 - Identify patients in need of surgical repair (emergency and elective).
 - Identify factors predisposing to abdominal wall hernia.
 - Counsel and educate patients on the risks associated with uncorrected hernias and identify hernias at special risk of strangulation.
 - Identify possible sites of intra-abdominal herniation by considering embryology of intra-abdominal structures; and outline the potential clinical significance of intra-abdominal hernias.

002H Adrenal Mass

Overview

Adrenal masses are at times found incidentally after computed tomography (CT) scan, magnetic resonance imaging (MRI), or ultrasound examination done for unrelated reasons. The incidence is about 3% (almost 10% of autopsies). Larger masses are likely to require investigation. Functioning adrenal masses comprise a group causing important treatable causes of hypertension.

Causes

1) Non-functioning adenoma

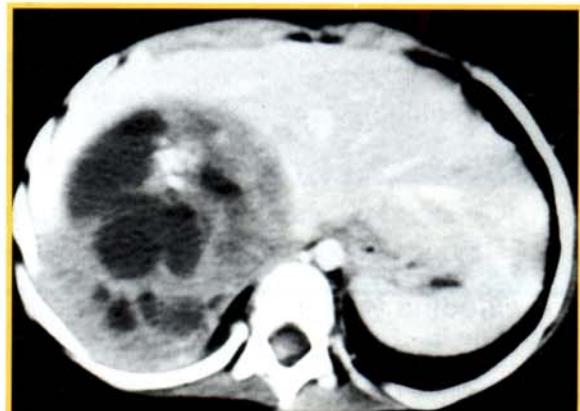
2) Functioning adenoma

- a) Cushing syndrome (glucocorticoid excess)
- b) Conn syndrome (aldosterone excess)
- c) Androgen excess
- d) Phaeochromocytoma

3) Adrenal carcinoma

4) Metastasis

5) Neuroblastoma (in children)



Adrenal neuroblastoma



Serum / Plasma

Sodium	139 mmol/L
Potassium	↓ 2.4 mmol/L
Chloride	96 mmol/L
Bicarbonate	↑ 34 mmol/L
Urea	3.8 mmol/L
Creatinine	71 μ mol/L
Ionised calcium	↓ 1.11 mmol/L
Aldosterone	↑ 2029 pmol/L
Renin	<7.0 mU/L

Adrenal mass on CT, with serum chemistry – Conn syndrome

Key Objectives

- Determine whether the mass is malignant or not.
- Determine whether the mass is functional or not.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate benign functioning adenomas from those that are non-functioning.
 - Differentiate benign from malignant masses by inquiring and examining for primary tumours which metastasise to the adrenal glands.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Select and interpret investigations for the exclusion of functioning adrenal masses.
 - List features noted on diagnostic imaging techniques suggestive of malignancy.
 - Select patients and list indications for fine needle aspiration cytological (FNAC) biopsy.
- Conduct an effective plan of management for a patient with an adrenal mass:
 - Outline initial plan of management for patients with adrenal masses which are functioning.
- Select patients in need of specialised care; list those requiring referral to endocrinology / internal medicine, and those requiring surgical referral.

003 Abdominal Pain

003A Abdominal Pain in Adults

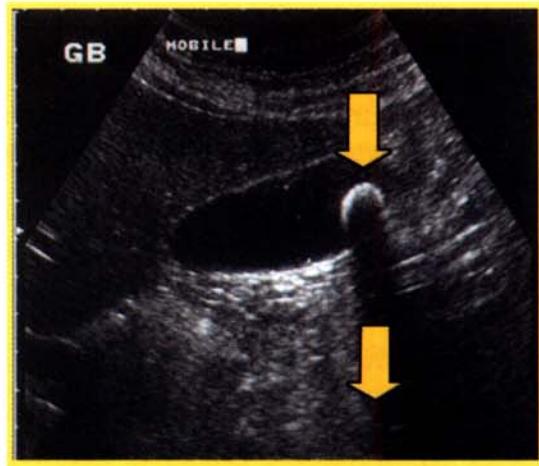
Overview

Abdominal pain may result from intra-abdominal inflammation or disorders of the abdominal wall. Pain may also be referred from sources outside the abdomen such as retro-peritoneal processes as well as intrathoracic processes. Thorough clinical evaluation is the most important 'test' in the diagnosis of acute abdominal pain. Localisation of the pain site, and awareness of characteristic symptom sequences, can reduce the list of possible causes to manageable size.

Causes

1) Right upper quadrant pain

- a) Biliary tract
 - Biliary 'colic'
 - Acute cholecystitis
 - Cholangitis
- b) Liver (hepatitis, hepatic abscess, neoplasms)
- c) Pancreatitis
- d) Renal pain (pyelonephritis, tumours, infarction and polycystic kidney)
- e) Extra-abdominal (myocardial infarction (MI), pulmonary embolus, pneumonia)



Acute cholecystitis

2) Epigastric pain

- a) Peptic ulcer disease
- b) Pancreatitis
- c) Cholangitis
- d) Functional dyspepsia
- e) Neoplasms (hepatic, gastric, pancreatic)
- f) Abdominal aortic aneurysm
- g) Epigastric hernia

3) Left upper quadrant pain

- a) Peptic ulcer disease
- b) Pancreatitis
- c) Gastric and pancreatic neoplasms
- d) Renal pain

4) Right/Left lower quadrant pain

- a) Bowel (appendicitis, colitis, diverticulitis: large and small bowel)
- b) Mesenteric lymphadenitis
- c) Gynaecologic causes (complicated ovarian cyst, ectopic pregnancy, pelvic inflammatory disease)
- d) Ureteric/Renal 'colic'
- e) Urinary retention
- f) Pyelonephritis and cystitis



Ureteric calculus – renal 'colic'

5) Generalised

- a) Functional gut disorders (functional dyspepsia, irritable bowel syndrome)
- b) Peritoneal inflammation (ruptured viscus, bacterial peritonitis)
- c) Bowel obstruction
- d) Vascular (ischaemic bowel, ruptured abdominal aortic aneurysm)
- e) Metabolic (diabetic ketoacidosis, porphyria)
- f) Neurogenic (herpes zoster)
- g) Other (Mediterranean/Malta fever (brucellosis), sickle cell crisis, etc.)

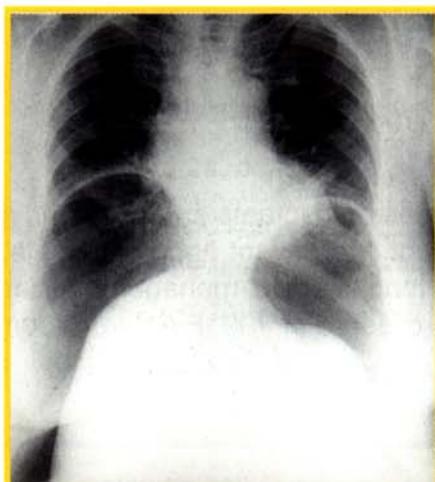


Small bowel obstruction – adhesions

6) Special group 'acute abdomen – acute abdominal surgical emergency'

- a) Perforated viscus (peptic ulcer, appendix, gallbladder, colon)
 - Perforated peptic ulcer
 - Perforated appendicitis / cholecystitis
 - Perforated colonic diverticulitis with faecal peritonitis
- b) Strangulated intestinal obstruction
- c) Ruptured aortic aneurysm
- d) Acute haemorrhagic pancreatitis (pancreatic necrosis)
- e) Ruptured ectopic pregnancy
- f) Acute massive mesenteric vascular infarction

- g) Blunt or penetrating trauma with peritonitis**
- h) Primary bacterial peritonitis**



Perforated colonic diverticulitis



*Intestinal obstruction – strangulated
groin hernia*

Key Objectives

- Recognise patients with abdominal pain who require emergency treatment, medical or surgical.
- Recognise the special group of 'acute abdomen – acute abdominal surgical emergency' where early surgical referral and exploration is likely to be required.
- Determine whether extra-abdominal causes listed above (MI, etc.) may be causing the pain.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Elicit clinical findings and their sequence which are key to establishing the most likely source of the pain.
 - Differentiate intra-abdominal from extra-abdominal or metabolic causes of acute abdominal pain.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Interpret abdominal X-rays and other imaging modalities diagnostic of various causes.
- Conduct an effective plan of management for a patient with acute abdominal pain:
 - Select patients who require emergency surgery and who require emergency medical care.
 - Outline a plan of management for common causes of abdominal pain.
 - Select patients in need of specialised care and/or further investigation.
 - Understand the common and life-threatening causes of acute abdominal pain.

003B Non-Acute/Recurrent Abdominal Pain in Infancy and Early Childhood

Overview

Abdominal pain is a common and disconcerting problem in infants and children. In an infant or child presenting with **chronic** but persistent intermittent abdominal pain, the probable causes should be rapidly identified so that directed management can be initiated. Causes of **acute** abdominal pain are considered in #003F Acute Abdominal Pain in Children.

Causes

1) Organic

a) Abdominal

- Infantile colic
- Infectious (*Giardia lamblia*)
- Obstructive (chronic constipation)
- Trauma
- Vasculitic
- Miscellaneous

b) Extra-abdominal

- Miscellaneous (peptic ulceration, intermittent porphyria)

2) Functional

a) Psychogenic (family disruption, school failure)

Key Objective

- It is important to differentiate between functional and organic causes and to recognise how causes of abdominal pain in infants and children differ from those in adulthood.

General/Specific Objectives

- Through efficient, focused data gathering:
 - In an infant, identify the acute organic causes and differentiate from infantile colic.
 - Differentiate acute from chronic and functional abdominal pain.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select appropriate laboratory investigations to differentiate those conditions requiring acute management.

- Investigate colic and functional pain with the minimum investigations to establish the diagnosis.
- Identify from investigations, the key essentials differentiating acute from chronic pain.
- Conduct an effective plan of management for a patient with abdominal pain:
 - In infants, outline the initial stages for management of acute intestinal obstruction.
 - Manage infantile colic with the infant's interests as the focus.
 - Manage functional abdominal pain with the child's interests as the focus.
 - Outline the treatment programme for a child with chronic abdominal pain utilising the support of the parents, family and community services.

003C Chronic Recurrent Abdominal Pain

Overview

Chronic and recurrent abdominal pain is a common symptom with an extensive differential diagnosis and heterogeneous pathophysiology. The history and physical examination frequently differentiate between functional and more serious underlying diseases.

Causes

1) Intestinal tract

- a) Oesophageal (oesophagitis)
- b) Gastric (peptic ulcer disease, neoplasm)
- c) Duodenal (peptic ulcer disease)
- d) Small bowel (inflammatory bowel disease (IBD), neoplasm)
- e) Colon (IBD, irritable bowel syndrome, neoplasm, diverticulosis)

2) Related organs

- a) Gallbladder / Biliary tract (cholelithiasis / biliary 'colic')
- b) Pancreas (chronic pancreatitis, neoplasm)



Bile duct stone – MRCP and ERCP

3) Other

- a) Psychogenic factors (malingering, functional dyspepsia)
- b) Abdominal wall
- c) Gynaecologic causes
- d) Visceral neoplasms/lymphomas
- e) Collagen / Vascular
- f) Lactose intolerance

Key Objective

- Recognising that visceral pain is typically poorly localised and often referred to distal sites, differentiate between various causes of chronic abdominal pain.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate between organic and non-organic causes of chronic abdominal pain.
 - Select patients in need of further laboratory and radiological investigation.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Outline the significance of common findings on ultrasound or computed tomography (CT) imaging of the abdomen as well as barium contrast studies.
- Conduct an effective plan of management for a patient with chronic abdominal pain:
 - Contrast the medical, surgical, nutritional and psychological management of chronic abdominal pain.
 - Select narcotics appropriately for patients and manage complications arising from the use of these drugs.
 - Select patients in need of referral to other healthcare professionals.
 - Counsel and provide appropriate education for patients with chronic abdominal pain syndromes.

003D Heartburn/Dyspepsia

Overview

'Heartburn' and 'dyspepsia' are common gastrointestinal complaints, since studies from various countries report an incidence of the symptoms in 20–40% of adults. Although serious complications can arise with few premonitory symptoms, appropriate management can generally avert such sequelae.

Causes

- 1) Functional dyspepsia / Somatoform disorders**
- 2) Gastro-oesophageal reflux disease with/without oesophagitis**
- 3) Motility disorders of the oesophagus**
 - a) Achalasia
 - b) Scleroderma
- 4) Peptic ulcer disease (including drugs)**
- 5) Miscellaneous (biliary disease, irritable bowel, chronic pancreatitis, diabetic gastroparesis, ischaemic heart disease, etc.)**

Key Objectives

- Diagnose somatoform disorders by inclusion rather than exclusion; always exclude the possibility of ischaemic heart disease.
- Outline conservative management measures including dietary counselling.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate between dyspepsia and chest pain or referred pain resulting from ischaemic heart disease.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - List or outline indications for radiological or endoscopic investigation of patients with symptoms suggestive of gastro-oesophageal reflux or peptic ulcer.
- Conduct an effective plan of management for a patient with 'heartburn':
 - Discuss the current concepts of pathophysiology in the management of gastro-oesophageal reflux and peptic ulcer disease.

- Counsel patients regarding lifestyle modification which can ameliorate symptoms of gastro-oesophageal reflux and peptic ulcer disease.
- Select patients in need of specialised care.



Oesophageal motility disorder



Sliding oesophageal hiatus hernia

003E Anal Pain

Overview

While almost all causes of anal pain are treatable, some can be destructive locally if left untreated. Anal pain is **not** a feature of uncomplicated haemorrhoids.

Causes

1) Identifiable

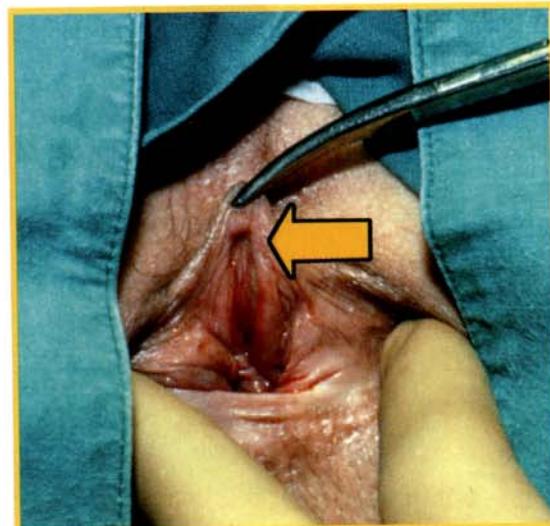
- a) Perianal haematoma
- b) Haemorrhoids (painful due to thrombosis, infection, or erosion)
- c) Anal fissure
- d) Anal ulcer / Anal cancer
- e) Perianal fistula (due to Crohn disease)
- f) Perirectal abscess



Acute perianal haematoma

2) Other

- a) Proctalgia fugax, neuropathic pain syndrome
- b) Coccygeal pain, other pelvic floor muscle syndromes



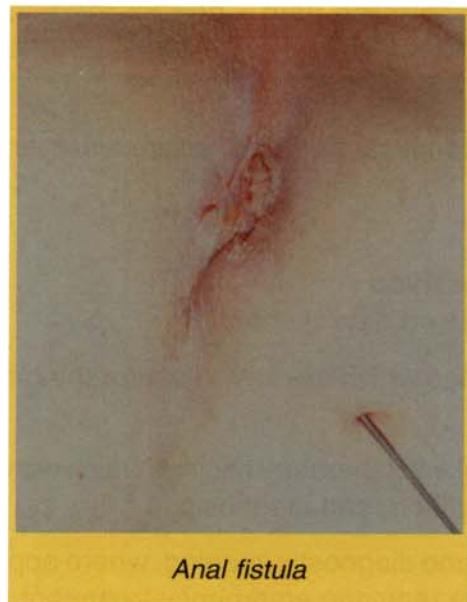
Anal fissure

Key Objective

- Perform visual inspection, palpation, and ano-rectal examination in all patients presenting with anal pain.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate between the causes of anal pain.
 - Establish whether tenesmus (an uncomfortable sense of incomplete evacuation leading to frequent, painful straining) is present.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Based on inspection, palpation and ano-rectal examination, differentiate the cause of anal pain.
- Conduct an effective plan of management for a patient with anal pain:
 - Select patients with perirectal abscess for urgent surgical treatment.
 - Counsel patients with haemorrhoids and anal fissure in the conservative treatment options including *sitz* baths, stool softeners and secondary preventive measures such as strict avoidance of constipation.
 - Select patients in need of specialised care.



003F Acute Abdominal Pain in Children

Overview

Acute abdominal pain in children may result from intra-abdominal inflammation or obstruction. Thorough clinical evaluation is usually the most important step in the diagnosis of abdominal pain so that directed management can be initiated.

Causes

1) Right lower quadrant / Left lower quadrant

- a) Appendicitis, constipation
- b) Mesenteric lymphadenitis
- c) Inflammatory bowel disease (IBD)
- d) Inguinal hernia (incarcerated)

2) Generalised

- a) Peritoneal inflammation (ruptured viscus, bacterial peritonitis)
- b) Bowel (infantile colic, gastroenteritis, bowel obstruction (intussusception, constipation))
- c) Psychosomatic
- d) Extra-abdominal (referred pain – pneumonia)

Key Objective

- Select patients with abdominal pain who require emergency treatment, medical or surgical.

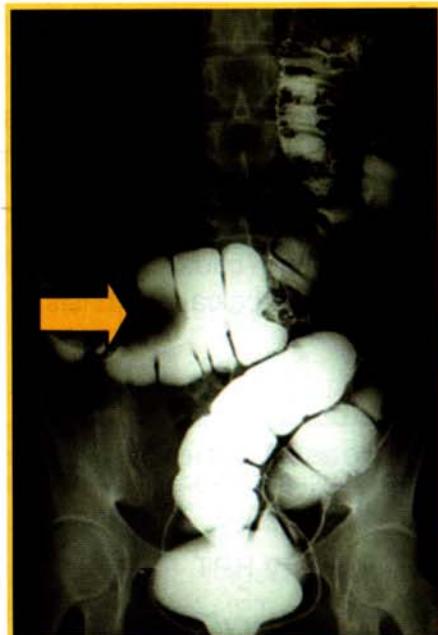
General/Specific Objectives

- Through efficient, focused data gathering:
 - Elicit clinical findings which are key to establishing the most likely source of the pain.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Select laboratory and diagnostic imaging, where appropriate, to determine whether conditions requiring emergency treatment are present.
 - Interpret abdominal X-rays.
- Conduct an effective plan of management for children with acute abdominal pain:
 - Select patients who require emergency surgery and those who require emergency medical care.

- Outline the initial plan of management in infants with acute intestinal obstruction.
- Outline a plan of management for common causes of abdominal pain (e.g. infantile colic) with the child's interest as the focus.
- Outline a plan of management for a child with acute abdominal pain using support from parents, family, and community services.
- Select patients in need of specialised care and/or further investigation.



Inguinal hernia – non-reducible



Ileocolic intussusception

004A Hypercalcaemia

Overview

Hypercalcaemia may be associated with an excess of calcium in both extracellular fluid and bone (e.g. increased intestinal absorption), or with a localised or generalised deficit of calcium in bone (e.g. increased bone resorption). This differentiation is important for both diagnostic and management reasons. Hypercalcaemia in adults requires investigation to identify the cause. Primary hyperparathyroidism (HPT) is a common cause and is differentiated from other causes such as bone malignancy by high serum parathormone.

Most patients with hypercalcaemia due to primary HPT have or will develop symptoms, the most important of which are changes in the mental state, lethargy, constipation, thirst, urinary frequency and nocturia. Surgery is indicated in the majority of patients with established primary HPT – the commonest cause is a single parathyroid adenoma. Hypercalcaemia can cause severe anatomic injury to the kidneys and, if severe, patients may develop hypercalcaemic crisis.

Causes

1) Increased bone resorption

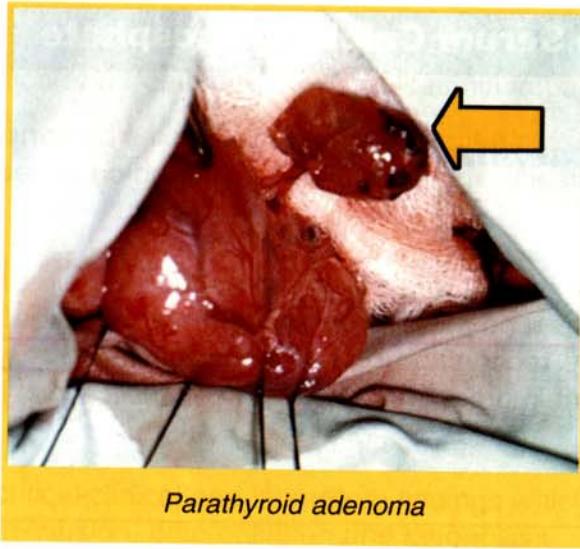
- a) Malignancy
- b) Primary HPT
- c) Secondary/Tertiary HPT
- d) Hyperthyroidism
- e) Immobilisation
- f) Paget disease of bone

2) Increased intestinal absorption

- a) Increased intake (e.g. milk alkali syndrome)
- b) Vitamin D-mediated (e.g. granulomatous diseases such as sarcoidosis)

3) Diminished excretion (familial hypocalciuric hypercalcaemia, drugs)

4) Miscellaneous



Parathyroid adenoma

Key Objective

- Formulate a management plan for hypercalcaemia consistent with its causal conditions.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Outline a diagnostic and management plan for a patient with hypercalcaemia.
 - Outline laboratory tests and diagnostic imaging findings in relation to the common causes and contrast the findings in primary HPT, secondary HPT, cancer and Paget disease.
 - Outline embryological development of parathyroids and the relevance to sites of parathyroid adenomas.
 - Differentiate hypercalcaemia caused by increased intake from that of excess bone resorption.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis.
- Conduct an effective plan of management for a patient with hypercalcaemia:
 - Select patients in need of specialised care.

004B Hypocalcaemia

Overview

Hypocalcaemia is an important and potentially serious complication from a variety of causes. Tetany, seizures, and papilloedema may occur in patients who develop acute hypocalcaemia. Early recognition of symptoms of paraesthesiae is important in diagnosis and treatment in order to prevent these complications in patients at risk.

Causes

1) Loss of calcium from the circulation

- a) Hyperphosphataemia (renal insufficiency)
- b) Pancreatitis
- c) Osteoblastic metastases
- d) Drugs (ethylene diamine tetra-acetic acid (EDTA), citrate)
- e) Rhabdomyolysis
- f) Respiratory (overbreathing) or metabolic alkalosis

2) Decreased vitamin D production or action

- a) Renal failure
- b) Rickets/Rachitis
- c) Malabsorption

3) Decreased parathyroid hormone (PTH) production or actions

- a) After thyroid/parathyroid surgery
- b) Autoimmune
- c) Diminished response

4) Hypomagnesaemia

Key Objectives

- Early recognition and treatment of hypocalcaemia in patients at risk.
- Calculate a corrected calcium concentration in the presence of hypoalbuminaemia before initiating any other investigation.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate hypocalcaemia caused by hyperphosphataemia/hypomagnesaemia from that of diminished production or action of PTH or vitamin D.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Contrast laboratory findings in the various conditions causing hypocalcaemia.
- Conduct an effective plan of management for a patient with hypocalcaemia:
 - Formulate a management plan for acute hypocalcaemia associated with either tetany or seizures.
 - Select patients in need of specialised care.

004C Hypophosphataemia / Fanconi Syndrome

Overview

Of hospitalised patients, 10–15% develop hypophosphataemia, and a small proportion have sufficiently profound depletion to lead to complications (e.g. rhabdomyolysis).

Causes

1) Gastro-intestinal

- a) Decreased dietary intake / Vomiting (prolonged, severe)
- b) Decreased absorption (chronic diarrhoea, steatorrhoea, vitamin D malabsorption)
- c) Antacids (binding of ingested and secreted phosphate)

2) Renal losses

- a) Hyperparathyroidism (HPT) (also associated with diminished vitamin D)
- b) Osmotic diuresis (salt, glucose)
- c) Primary (isolated, Fanconi syndrome)

3) Redistribution (intracellular shift)

- a) Re-feeding (stimulated by insulin)
- b) Respiratory alkalosis, acute
- c) 'Hungry-bone' syndrome

Key Objectives

- Appreciate that the likely causes are gastrointestinal, renal or redistributive.
- Select the most conservative form of therapy, since intravenous (IV) phosphate salts are potentially hazardous.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Diagnose the cause of hypophosphataemia.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - If no cause is clinically apparent, differentiate between **redistribution**, **gastrointestinal** and **renal** causes by measuring fractional urinary phosphate excretion.

- Conduct an effective plan of management for a hypophosphataemic patient:
 - State that most patients **will not** require therapy other than repair of the underlying causes.
 - Recognise that phosphate is a major intracellular anion and cellular energy store component; and is a necessary additive in prolonged parenteral nutritional therapy.
- Select patients with vitamin D deficiency for replacement with vitamin D.

005 Abnormal Serum Hydrogen Ion Concentration

Overview

The hydrogen ion content of arterial blood is normally kept blandly alkaline at about 40 nanomolar ($[H^+]$ between 36 and 44 nmol/L, which coincidentally equals a range of pH from 7.44 to 7.36), by combined renal and respiratory regulatory mechanisms together with extracellular and intracellular buffer systems.

Major adverse consequences may occur with severe acidaemia and alkalaemia despite absence of specific symptoms (the range of extracellular hydrogen ion content compatible with life is approximately 20–200 nmol/L. This 10-fold change is expressed in the pH notation as a change of one pH unit, e.g. from 7.8 to 6.8). The reversible reaction $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$ forms the basis of renal and respiratory control. The diagnosis of acid-base disorders depends on recognition of the clinical setting and appropriate laboratory studies. It is crucial to distinguish acidaemia and alkalaemia due to metabolic causes from those due to respiratory (gaseous) causes; especially important is detecting the presence of both. Management of the underlying causes and not simply of the change in hydrogen ion concentration is essential.

Causes

1) 005A Metabolic acidosis

a) Addition of acid (high anion gap)

- Endogenous acids (lactic acidosis, ketoacidosis, renal failure)
- Exogenous acids (methanol, ethylene glycol, salicylate, etc.)

b) Loss of alkali/base (normal anion gap)

- Gastrointestinal bicarbonate loss (e.g. diarrhoea, small bowel or pancreatic fistula)
- Renal bicarbonate loss (e.g. renal tubular acidosis, interstitial nephritis)



Serum / Plasma

Sodium	138 mmol/L
Potassium	5.5 mmol/L
Chloride	113 mmol/L
Bicarbonate	↓ 15 mmol/L
Urea	↑ 11.5 mmol/L
Creatinine	↑ 140 µmol/L
Lactate	↑ 3.2 mmol/L
Arterial pH	↓ 7.28
P_aCO_2	↓ 30 mm Hg
P_aO_2	↓ 70mm Hg

Mesenteric ischaemia and serum chemistry – metabolic (lactic) acidosis

2) 005B Metabolic alkalosis

a) Addition of alkali/base

- Milk-alkali syndrome

b) Loss of acid

- Gastrointestinal loss of acid (gastric loss in vomiting, pyloric stenosis)
- Renal loss of acid (e.g. diuretics)
- Associated with potassium loss (e.g. Conn syndrome – primary aldosteronism)



Serum / Plasma

Sodium	↓ 129 mmol/L
Potassium	↓ 2.9 mmol/L
Chloride	↓ 85 mmol/L
Bicarbonate	↑ 39 mmol/L
Creatinine	120 µmol/L
Arterial pH	↑ 7.49
P_aCO_2	41 mm Hg
P_aO_2	100mm Hg

Pyloric stenosis and serum chemistry – hypochloraemic hypokalaemic metabolic alkalosis

3) 005C Respiratory (gaseous) acidosis (due to decreased alveolar ventilation, usually with attendant hypoxia)

- a) Pulmonary causes of underventilation (e.g. chronic obstructive pulmonary disease (COPD), upper airway obstruction, atelectasis and sputum retention, pneumonia, interstitial lung disease, pulmonary fibrosis, etc.)
- b) Neuromuscular causes (e.g. wound pain, drugs, cerebral trauma and coma, encephalitis, bulbar palsy, myasthenia, muscle paralysis)

4) 005D Respiratory alkalosis (due to increased alveolar ventilation from overbreathing)

- a) Psychogenic overbreathing from anxiety state
- b) Miscellaneous (e.g. fever, drugs, salicylate, central nervous system (CNS) disorders)

5) 005E Mixed acid-base disorders

Key Objectives

- In relevant clinical situations, determine the possibility of an acid-base disorder by examining laboratory parameters of: $[H^+]$, P_{CO_2} , $[HCO_3^-]$, and anion gap.
- Diagnose and treat the causal condition in conjunction with appropriate additional measures to correct the abnormality and restore or maintain renal and respiratory functions.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Diagnose the precipitating cause of acidaemia/alkalaemia expeditiously.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret appropriate investigations for various patients with acidaemia/alkalaemia in order to identify the primary abnormality and the adequacy of the associated secondary 'compensation' (e.g. renal failure with acidotic breathing, pyloric stenosis with secondary renal effects, etc.).
- Conduct an effective plan of management for a patient with acidaemia/alkalaemia:
 - Outline general supportive measures.
 - Outline specific management for patients in each of the main groups.
 - Select patients who need specialised care and/or consultation.

006 Abnormal Serum Lipids

Overview

Abnormalities of serum lipid levels in Australian society due to dietary and other lifestyle factors are very common. Secondary causes are relatively uncommon. Abnormal lipid levels comprise one of the risk factors for arterial disease, particularly coronary artery disease (CAD).

Identification is by screening of well adults, those with other risk factors and patients with suspected or established CAD. Interpretation of results and their classification is complex depending on the lipid profile found and the presence of other risk factors. Levels can be modified by lifestyle changes and drug therapy, although the use of lipid lowering agents to reduce risk is expensive.

Causes

1) Hypercholesterolaemia (elevated low-density lipoprotein (LDL))

a) Primary causes

- Polygenic (most common)
- Familial hypercholesterolaemia (rare)

b) Secondary causes

- Hypothyroidism
- Obstructive liver disease
- Nephrotic syndrome
- Drugs (such as cyclosporine, thiazides)

2) Hypertriglyceridaemia

a) Primary causes

- Dietary
- Familial hypertriglyceridaemia

b) Secondary causes

- Obesity
- Diabetes mellitus
- Chronic renal failure
- Moderate ethanol use

3) Low high-density lipoprotein (HDL)

a) Obesity

b) Cigarette smoking

c) Inactivity

Key Objectives

- Identify persons at risk of CAD who would benefit from serum lipid reduction.
- Conduct an effective management plan for a patient with abnormal serum lipids, which includes behavioural change and possible drug therapy, according to their overall risk of CAD.

General/Specific Objectives

- Understand the principles of population screening.
- Identify patients with secondary causes of lipid abnormalities.
- Interpret and integrate lipid profile with other risk factors for CAD.
- Specify lipid levels (especially LDL cholesterol) to be attained by treatment, including a long term followup plan.
- Be aware of the range of therapeutic agents which lower serum lipids, their indications, costs and side-effects.

Overview

Thorough investigation can distinguish benign reversible liver disease requiring no treatment from potentially life-threatening conditions requiring immediate therapy. Drugs, alcohol and infection (hepatitis) are the commonest causes. Liver function tests may also assist in making the decision on whether some form of intervention is required.

Causes

1) Isolated hyperbilirubinaemia

a) Unconjugated or indirect

- Haemolysis and ineffective erythropoiesis
- Defects in transport into the hepatocytes or intracellular conjugation:
 - Gilbert disease
 - Neonates
 - Crigler-Najjar syndrome

b) Conjugated or direct – defect in transport out of the hepatocyte

- Rotor syndrome, Dubin-Johnson syndrome

2) Hepatocellular (may lead to cirrhosis)

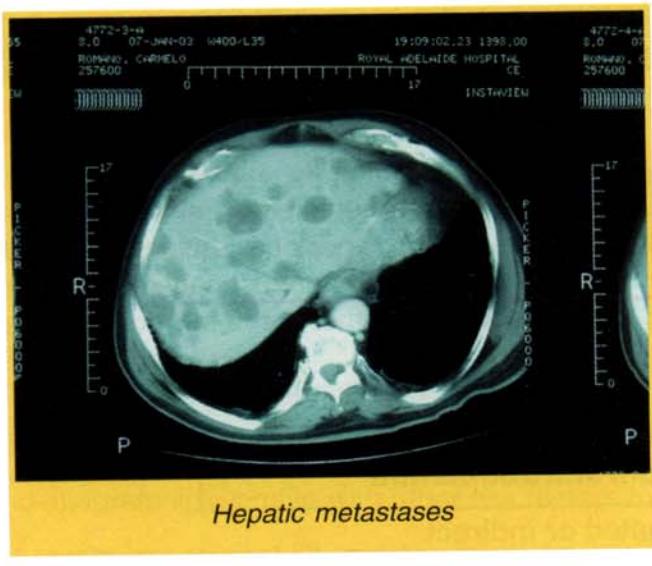
- a) Alcohol, drugs, toxins (paracetamol, isoniazid)
- b) Fatty liver and steatohepatitis
- c) Viral hepatitis
- d) Metabolic liver disease (haemochromatosis, Wilson disease, etc.)
- e) Autoimmune chronic hepatitis
- f) Shock or ischaemia
- g) Septicaemia

3) Cholestatic

a) Intrahepatic

- Drugs (oral contraceptives)
- Infiltrative (amyloid, malignant)
- Congestive (e.g. heart failure)
- Autoimmune (primary biliary cirrhosis, sclerosing cholangitis)
- Granulomatous disease

b) Extrahepatic (cholestasis from stone or neoplasm, stricture, atresia)



Key Objectives

- Discuss abnormal laboratory tests in the context of the clinical presentation, and select patients requiring medical or interventional management.
- Understand the effects of various toxins (e.g. drugs, alcohol, sepsis) on the liver.
- Understand the liver's role in storage, synthesis and metabolism.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate between the causal conditions for abnormal liver function tests.
 - Identify complications related to the presence of liver disease.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select diagnostic tests appropriate for the identification of acute and chronic liver diseases.
 - List the indications for abdominal ultrasound and ascitic fluid analysis.
 - List indications for liver biopsy.
- Conduct an effective plan of management for a patient with abnormal liver function tests:
 - Select patients in need of hospitalisation.
 - List indications for active and passive prophylaxis against infective hepatitis.
 - Select patients in need of specialised care.
 - Counsel and educate patients about primary and secondary prevention strategies for viral hepatitis (include public health measures).
 - Understand the role of monitoring liver function in terms of management of a patient's illness.

008 Abnormal Serum Potassium Concentration / Magnesium

008A Hypokalaemia

Overview

Potassium is the major intracellular cation. Of a total body potassium of 3,000 mmol in adults, only two percent is in extracellular fluid (60 mmol) at a concentration of around 5 mmol/L. Normal daily intake is also about 60 mmol. Most excretion is in the urine which is the source of most depletions, but abnormal gastrointestinal losses are also important. Hypokalaemia usually indicates potassium depletion; and is most often discovered on routine analysis of serum electrolytes or electrocardiogram (ECG) results. Symptoms usually develop much later when depletion is quite severe (muscle weakness, paralytic ileus, cardiac arrhythmias and sensitivity to digitalis). Hypokalaemia implies a serum level below 3.5 mmol/L. Potassium deficiency, metabolic alkalosis, hypocalcaemia and hypomagnesaemia are commonly associated.

Causes

1) Increased losses

a) Renal losses

- Diuretics
- Endocrine effects
 - Prolonged steroid therapy
 - Primary hyperaldosteronism (Conn syndrome)
 - Secondary hyperaldosteronism (e.g. renovascular disease)
 - Adrenal hyperplasia
 - Cushing syndrome
 - Ectopic adrenocorticotrophic hormone (ACTH)

b) Gastrointestinal losses

- Vomiting – vomiting and pyloric stenosis; secondary renal losses occur as well.
- Bowel obstructions and prolonged gastrointestinal aspirates
- Diarrhoea (villous adenoma of colorectum, laxative abuse, inflammatory bowel disease (IBD), enteric fistula)

2) Decreased intake (e.g. anorexia nervosa, malnutrition)

3) Redistribution (familial periodic paralysis)

Key Objective

- Assess intake and shift of potassium into cells, select renal loss as the category into which most problems fall, but remember gastrointestinal causes of loss.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate between renal and gastrointestinal losses as causative lesions.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Outline how urinary electrolytes assist in the elucidation of excess losses of potassium.
- Conduct an effective plan of management for a hypokalaemic patient based on sound principles:
 - Outline indications and guidelines for intravenous (IV) potassium therapy.

008 Abnormal Serum Potassium Concentration / Magnesium

008B Hyperkalaemia

Overview

Hyperkalaemia (serum $[K^+]$ greater than 5.5 mmol/L) may produce serious side-effects and may also be indicative of the presence of serious associated medical conditions. Dangerous hyperkalaemia (greater than 6 mmol/L) is rare if renal function is normal despite significant release of intracellular potassium into the extracellular phase in response to the stress of injury, acidosis, sepsis and any catabolic state. Hyperkalaemia is seen when renal failure and shock interfere with renal handling of potassium.

Causes

1) Reduced urinary excretion in renal failure and shock

- a) Decreased glomerular filtration rate (GFR) in shock, acute or chronic renal failure
- b) Decreased tubular secretion in adrenal insufficiency
- c) Effects of drugs on tubular function (angiotensin-converting enzyme (ACE) inhibitors, nonsteroidal anti-inflammatory drugs (NSAIDs))

2) Redistribution

- a) Red cell lysis
 - Rhabdomyolysis
 - Crush syndrome / Burns
 - Intravascular haemolysis
 - Tumour breakdown
- b) Metabolic acidosis with normal anion gap

Key Objectives

- Diagnose true dangerous hyperkalaemia (greater than 6.0 mmol/L), a potentially lethal condition for which emergency treatment is the first consideration, from pseudohyperkalaemia, due to cell lysis during collection, and screen for causal conditions.
- Recognise that dangerous effects are confined to the heart, and cause changes as in the electrocardiogram (ECG), arrhythmias and death.
- Recognise principles of emergency (temporary plus definitive) treatment of dangerous hyperkalaemia.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Distinguish between life-threatening hyperkalaemia and pseudohyperkalaemia utilising ECG.
 - Recognise signs of dangerous hyperkalaemia on ECG.
 - Distinguish between causes of hyperkalaemia by ruling out redistribution or intake problems quickly, and concentrating on the more common renal causes.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Identify patients with renal failure.
- Outline an effective emergency treatment plan for a dangerously hyperkalaemic patient:
 - Select patients in need of specialised care and dialysis.
 - Understand principles of haemodialysis and peritoneal dialysis.

008C Hypomagnesaemia

Overview

Although hypomagnesaemia occurs in only about 10% of hospitalised patients, the incidence rises to over 60% in severely ill patients. Hypomagnesaemia may be responsible for otherwise puzzling clinical features in patients with prolonged illness. Clinical features are mainly neuromuscular. Magnesium, like potassium, is a predominantly intracellular cation and is poorly absorbed orally, so replenishment is best by intravenous (IV) supplementation.

Causes

1) Gastrointestinal

- a) Marked decrease in dietary intake
- b) Diarrhoea, acute/chronic; malabsorption and steatorrhoea, short gut
- c) Acute pancreatitis

2) Renal loss

- a) Diuretics (loop, thiazide, hypercalcaemia)
- b) Volume expansion
- c) Tubular dysfunction (alcoholics, aminoglycosides, amphotericin, cisplatin, cyclosporin, acute tubular necrosis (ATN) in diuretic phase, primary)

Key Objectives

- Determine which patients are likely to be hypomagnesaemic since magnesium levels are not measured routinely.
- Evaluate patients with ventricular arrhythmias for possible hypomagnesaemia, especially during ischaemic events and if diuretics were prescribed.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Diagnose the cause of hypomagnesaemia.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - If no cause is clinically apparent, differentiate between gastrointestinal and renal causes by measuring urinary magnesium excretion.
- Conduct an effective plan of management for a hypomagnesaemic patient:
 - Recognise that cellular uptake of magnesium is slow, and repletion requires sustained correction.
 - Select potassium-sparing diuretics as an adjunct to management in patients with diuretic-induced hypomagnesaemia if diuretic therapy cannot be stopped.

009A Hyponatraemia

Overview

Serum sodium levels comprise the major determinant of extracellular fluid osmolality and are normally held constant within a range of 135–145 mmol/L by regulatory mechanisms acting via sensitive hypothalamic osmoreceptor feedback, utilising stimulation of antidiuretic hormone (ADH) by increased osmolality or vice versa. Causes of increased ADH secretion, apart from hypertonicity of body fluids, include volume depletion, stress, drugs and inappropriate sources of ADH from cerebral injury, tumours or burns. The syndrome of inappropriate ADH secretion (SIADH) is seen quite commonly in the setting of chest infections and other lung conditions and is an idiosyncratic reaction of a number of drugs, particularly thiazide diuretics.

Hyponatraemia implies only a fall in the concentration of serum sodium and may be associated with water gain, sodium depletion or often both.

Hyponatraemia (serum $[Na^+]$ less than 135 mmol/L) is often detected in asymptomatic patients because serum electrolytes are measured almost routinely. Minor abnormalities often are transient and need no treatment. In adults or children with hyponatraemia, the cause is usually iatrogenic, and the hyponatraemia dilutional rather than depletional. Normally any fall in body fluid osmolality due to water overload is countered by the excretion of excess water by the kidneys. Poor renal function with poor concentrating power diminishes the ability to cope with excess water, which is also a feature of the early period after major surgery.

Causes

1) Hypo-osmolar hyponatraemia

a) Water gain

- Iatrogenic** excess water administration (particularly after operations and in children)
- SIADH with decreased water excretion

b) Sodium depletion

- Renal or extrarenal **sodium and water loss with water replacement in excess of electrolytes** (enteric losses and fistulae, renal losses)
- Addison disease

c) Oedema states

2) Non-hypo-osmolar hyponatraemia – associated with hyperglycaemia or azotaemia

Key Objectives

- Recognition that hyponatraemia may mean water gain, sodium depletion or a mixture of both.
- Recognition that minor abnormalities are often transient and need no treatment.
- Identification of the main process is important because this will affect need for and choice of therapy, and rate of correction.
- Recognition that water retention is spread over the whole body, and marked intracellular oedema has its most severe effects on essential functions causing mental confusion and ultimately coma.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine whether an increase in water relative to sodium exists thereby expanding volume of cells or the change in sodium concentration is artefactual or caused by hyperglycaemia.
 - Differentiate between sodium depletion and water gain by assessment of volume status and/or the presence of an oedema state.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Interpret urinary electrolyte concentrations.
 - Interpret plasma and urinary osmolality.
- Conduct an effective plan of management for a patient with hyponatraemia:
 - Outline a therapeutic approach based on the underlying process.
 - Select the patients with hyponatraemia in need of specialised care or consultation.

N.B. When serum sodium concentration is measured by flame photometry or other methods requiring major dilution of plasma, hyperlipidaemia or hyperproteinaemia may cause pseudohyponatraemia (iso-osmotic). If sodium concentration is measured with a sodium selective electrode on undiluted plasma (most laboratories today), a true sodium concentration is obtained, and this type of 'pseudohyponatraemia' no longer exists.

009B Hypernatraemia

Overview

Hypernatraemia is often iatrogenic with solute administration in excess of the water required to excrete it. Thirst is a significant warning sign of hyperosmolar states in the conscious patient. Hypernatraemia is usually associated with inability to respond to thirst by drinking water. Hypernatraemia is thus most likely to be encountered at the extremes of life in the very young and in the very old, in comatose patients and in those depending on tube or parenteral intravenous (IV) feeding.

Causes

1) Sodium gain (relative solute excess)

- a) Excessively concentrated enteric or IV feeding solutions
- b) Primary hyperaldosteronism (Conn syndrome)

2) Water depletion (true dehydration)

- a) Water intake insufficient, inability of unconscious or incapacitated patient to respond to thirst
- b) Excess renal water loss – osmotic diuresis, diabetes insipidus
- c) Excess insensible water loss – fever, overbreathing, burns

Key Objectives

- Recognition that hypernatraemia is almost always caused by a combination of water loss with diminished ability to respond to thirst.
- Recognition of importance of careful monitoring of use of parenteral or enteral feeding solutions in incapacitated patients to avoid hyperosmolar hyperglycaemic hypernatraemic coma.
- Recognise that correction of the hypernatraemic state requires restoring the water deficit to relieve and to reverse the hyperosmolar state, and restoring normal urine output instead of continuing osmotic solute diuresis.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine the underlying cause of water loss and/or diminished thirst.
 - Determine the severity of the problem by assessment of patient's volume status.

- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Evaluate urinary osmolality results in order to differentiate between causes of water loss.
- Conduct an effective plan of management for a patient with hypernatraemia:
 - Outline a therapeutic approach based on the underlying process.
 - Discuss potential side-effects of rapid replacement of water losses.
 - Select the patients with hypernatraemia in need of specialised care or consultation.

Overview

Because abnormalities of white blood cells occur commonly in both asymptomatic as well as acutely ill patients, every clinician will need to evaluate patients for this common problem. Most important to understand are the causes and clinical implications of neutropenia and neutrophilia.

Causes

1) Leucopenia

a) Neutropenia

- Decreased marrow production
 - Drug-induced (alkylating agents, antimetabolites, antibiotics (sulphonamides, penicillins, cephalosporins), anti-thyroid drugs (carbimazole, propylthiouracil), anticonvulsant drugs (phenytoin, carbamazepine, sodium valproate), anti-inflammatory drugs (phenylbutazone, gold, diflunisal, penicillamine, naproxen), antidepressants (amitriptyline, dothiepin, mianserin), antimalarial drugs (maloprim, fansidar, chloroquine))
 - Nutritional deficiency (vitamin B₁₂, folate)
 - Sepsis
 - Viral (hepatitis, human immunodeficiency virus (HIV), influenza)
 - Bacterial (typhoid, tuberculosis (TB))
 - Hypersplenism
 - Immune: rheumatoid arthritis (Felty syndrome); systemic lupus erythematosus (SLE)
 - Nonimmune: severe portal hypertension
 - Marrow infiltration
 - Malignancy: haematologic (acute and chronic leukaemia); non-haematologic (carcinoma lung)
 - Cyclic neutropenia

b) Lymphocytopenia

- Drug-induced (glucocorticoids, cytotoxics)
- Infections (HIV, TB)
- Tumour-associated (lymphoma)
- Acute illness (sepsis, myocardial infarction (MI))
- Chronic illness (uraemia, congestive cardiac failure (CCF), SLE)
- Marrow infiltration (*vide supra*)

c) Combined neutropenia and lymphocytopenia

- Marrow infiltration (*vide supra*)
- Drug-induced (cytotoxics)
- Aplastic marrow (idiopathic, benzene, irradiation)

2) Leucocytosis

a) Neutrophilia

- Inflammation
 - Infective – bacterial
 - Noninfective
 - Tissue necrosis / Infarction / Trauma of burns
 - Autoimmune disorders: SLE/vasculitis
 - Gout
- Neoplasia
 - Myeloproliferative neoplasms, other malignancies
- Drugs (glucocorticoids, lithium)
- Acute stress or haemorrhage
- Post-splenectomy
- Leukaemoid reactions (white cell count (WCC) greater than $50.0 \times 10^9/L$ – normal range $4.0 - 11.0 \times 10^9/L$)
 - Severe sepsis
 - Acute (septicaemia)
 - Chronic (TB)
 - Malignancy
 - Severe haemolysis
 - (Chronic myeloid leukaemia is a myeloproliferative disorder **not** a leukaemoid reaction)

b) Lymphocytosis

- Infections (viral, TB, pertussis, other)
- Acute and chronic lymphocytic leukaemia
- Other (adrenal insufficiency)

Key Objective

- Interpret the clinical setting in which the leucocyte abnormality occurs as this will often suggest the correct diagnosis and direct further investigation.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Distinguish between chronic conditions requiring non-urgent evaluation and acute life-threatening illnesses requiring admission to hospital.
- Interpret critical clinical and laboratory findings important in formulating a differential diagnosis:
 - Interpret the differential leucocyte count.
 - List the indications for bone marrow aspiration and biopsy.
- Conduct an effective plan of management for a patient with white blood cell abnormalities:
 - Diagnose or exclude infection first as the cause of the leucocyte abnormality.
 - Select patients for specialised care, including those with a neutrophil count of less than $1.0 \times 10^9/L$.
 - Counsel and educate patients with chronic leucocyte abnormalities.

011A Paediatric Emergencies

Overview

Although paediatric emergencies such as the ones listed below are discussed with the appropriate condition, the care of the patient in the paediatric age group demands special skills.

Causes

1) Respiratory emergencies

(see #126 Wheezing/Respiratory Difficulty/Stridor)

- a) **Upper respiratory tract disorders** (see #126A Upper Respiratory Tract Disorders)
- b) **Lower respiratory tract disorders** (see #126B Lower Respiratory Tract Disorders)

2) Infectious emergencies

(see #040 Fever and Chills (Adult and Paediatric) and #102B Childhood Communicable Disease With or Without Skin Rash)

3) Cardiovascular emergencies

- a) **Arrhythmias** (see #072 Palpitations (Abnormal Electrocardiogram (ECG) / Arrhythmia))
- b) **Congestive heart failure** (see #032A With Diffuse Chest X-Ray Abnormality)

4) Fluid and electrolyte emergencies

- a) **Dehydration / Volume depletion** (see #098 Shock/Hypotension and #009B Hypernatraemia)
- b) **Hyperkalaemia** (see #008B Hyperkalaemia)

5) Neurological emergencies

- a) **Seizures** (see #095 Seizures (Epilepsy))
- b) **Febrile seizure**

6) Abdominal emergencies

- a) **Abdominal pain** (see #003E Anal Pain)
- b) **Abdominal distension** (see #001 Abdominal Distension/Ileus)

7) Trauma

(see #018 Burns and #113 Trauma/Accidents/Prevention)

8) Poisoning

(see #079 Poisoning)

9) Environmental emergencies (hypothermia / heat stroke)

(see #040D Hypothermia and #040E Hyperthermia)

Key Objectives

- Recognise and manage effectively infants and children with life-threatening paediatric emergencies.
- Describe the differences between paediatric and adult medicine and their effect on emergency management.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Elicit symptoms and signs in a focused fashion for the assessment of an infant/child in an urgent/emergent situation.
 - Perform physical examination and blood pressure (BP) measurement and determine whether the patient is in shock.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis.
- Conduct an effective plan of management for an infant/child in an urgent/emergent situation:
 - Recognise special features of paediatric airway management.
 - List sites for intravenous (IV) access in the paediatric population.
 - Select patients in need of referral to intensive care units.
 - Outline initial management in a paediatric patient with seizures including febrile seizures.
 - Outline initial management in a paediatric patient with acute sepsis.

011B Crying/Fussing Child

Overview

A young infant whose only symptom is crying/fussing, challenges the doctor to distinguish between the various causes, some of which can be serious.

Causes

1) Infections (systemic/focal)

2) Gastrointestinal/Intra-abdominal conditions

- a) Infection
- b) Inflammation
- c) Intussusception
- d) Constipation/Anal fissure
- e) Diarrhoea

3) Trauma (neglect / child abuse / fracture)

4) Psychologic / Functional / Hunger / Discomfort / Boredom / Irritability

Key Objective

- Differentiate paediatric emergencies, including intussusception, from conditions not requiring emergency treatment.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Elicit a history of patient's previous behaviour, oral intake of food and drink, vomiting, diarrhoea or constipation, and any medications received.
 - Assess the parent-child emotional interaction.

- Perform a full physical examination in order to identify the cause of the illness with a focus on searching for sites of infection, intra-abdominal conditions, increased intracranial pressure, cardiac and respiratory disorders.
- Differentiate serious from benign causes, and determine if a life-threatening situation exists.
- Interpret critical clinical and laboratory findings which are key in the processes of exclusion, differentiation and diagnosis:
 - Select investigations (when appropriate) to differentiate between acute and benign disease.
- Conduct an effective plan of management for a crying/fussing child:
 - Counsel caregivers of crying/fussing children without organic disease.
 - Select children who require followup for additional investigation and management.
 - Select patients in need of referral.

011C Hypotonia / Floppy Infant

Overview

Children with decreased resistance to passive movement differ from those with weakness and hyporeflexia. They require detailed, careful neurologic evaluation. Management programmes, often life-long, are multidisciplinary and involve patients, family and community.

Causes

1) Central causes

- a) 'Benign congenital hypotonia'
- b) Cerebral malformations (holoprosencephaly); neurodegenerative (leucodystrophy)
- c) Seizures, trauma (subarachnoid or subdural haemorrhage)
- d) Hydrocephalus / Increased intracranial pressure
- e) Infectious causes (e.g. encephalitis, abscess, meningitis)
- f) Neoplasms
- g) Hypoxic/Ischaemic encephalopathy
- h) Effects of toxins and drugs

2) Neural disease, peripheral

- a) Anterior horn cell (e.g. progressive spinal muscular atrophy, spinal cord infarction)
- b) Peripheral nerves / Polyneuropathies (trauma)
- c) Myoneural junction (myasthenia gravis, botulism)

3) Muscular disease

- a) Muscular dystrophy
- b) Myotonic dystrophy
- c) Congenital myopathies
- d) Inflammatory myopathies (dermatomyositis / polymyositis)

4) Metabolic/Electrolyte causes (hypokalaemia, hypoglycaemia, etc.)

5) Other genetic causes (trisomy 21, Prader-Willi syndrome, Niemann-Pick disease, Tay-Sachs disease)

Key Objectives

- Determine the presence of conditions amenable to rapid treatment (electrolyte imbalance, seizure, infection, intracranial bleeding, hydrocephalus).
- Differentiate infants with generalised hypotonia from those with weakness and hyporeflexia.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine birth history, age and rapidity of onset and progression of symptoms.
 - Determine whether lesion is localised or general, through appropriate observation of posture, together with neurological, muscle and joint examination.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select investigations to differentiate central from neuromuscular causes (e.g. computed tomography (CT) versus serum creatine kinase (CK), electromyography (EMG), muscle biopsy).
 - Determine which children require genetic studies.
- Conduct an effective plan of management for a floppy infant:
 - Determine whether respiratory status is adequate or intubation is required.
 - Counsel families with afflicted children about management, prognosis and genetic implications.
 - Develop a management plan that involves the family and community resources.
 - Select patients in need of specialised care.

Overview

Anaemia may be the sole manifestation of serious medical disease. Anaemia may be due to blood loss, decreased production or increased destruction of red blood cells. Simple tests may provide important information. Key to an understanding of anaemia is a full blood examination (FBE) (which includes a blood film). This will give an indication as to both the aetiology and the severity of the anaemia, and will guide the direction for further investigation.

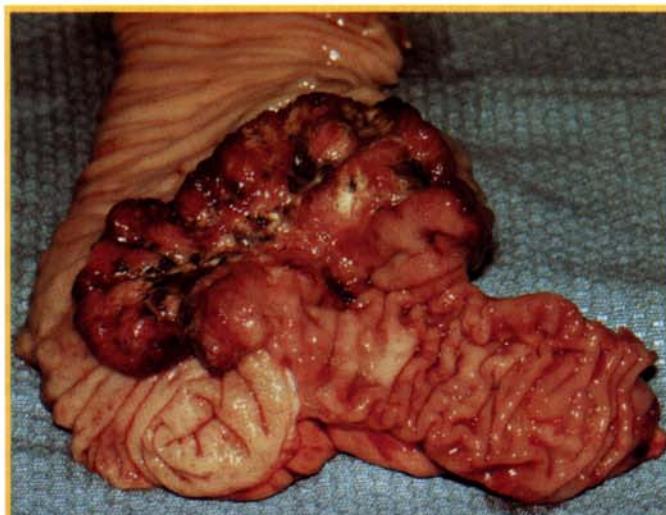
Causes

1) Normocytic

- a) Decreased production (bone marrow disease, reduced erythropoietin, drugs)
- b) Increased destruction
 - Red blood cell (RBC) abnormalities
 - Auto-antibodies
 - Drugs
- c) Blood loss (visible, occult)
- d) Apparent (dilutional anaemia, e.g. of pregnancy)

2) Microcytic (iron deficiency, chronic blood loss, haemoglobinopathies)

3) Macrocytic (folate, vitamin B₁₂ deficiency, chemotherapy, alcohol abuse)



Iron deficiency anaemia – carcinoma caecum

Key Objectives

- Be able to interpret a blood count and film.
- Understand that iron deficiency anaemia may indicate the presence of serious gastrointestinal disease.
- By considering the clinical context, determine if anaemia is present, since all three laboratory indices of anaemia are concentration measurements.
- Interpret the signs and symptoms of anaemia with the understanding that they are dependent on the rapidity with which anaemia developed.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine the presence of anaemia and differentiate between the various causes, according to the patient's age.
 - Select a causal classification of anaemias using red cell morphology.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Outline diagnostic plans for an adult presenting with an iron-deficiency anaemia.
- Conduct an effective plan of management for a patient with anaemia:
 - Outline treatment of iron deficiency anaemia.
 - Outline treatment of vitamin deficiency anaemias.
 - Select patients in need of referral for haematological consultation and care (e.g. haemolysis, bone marrow disease).
 - Conduct counselling and education of patients with anaemia caused by nutritional deficiencies and haemoglobinopathies.

Overview

Doctors may be confronted by developmental and behavioural problems of childhood and adolescence and required to liaise with other caregivers.

Causes

Learning disability – differential diagnosis includes:

- 1) Global cognitive problem (mental retardation or borderline IQ)**
- 2) Sensory impairment (auditory/visual)**
- 3) Family emotional disturbance: oppositional defiant / conduct disorder**
- 4) Anxiety/Mood disorders**
- 5) Developmental coordination disorder**
- 6) Dyslexia**
- 7) Attention deficit hyperactivity (hyperkinetic) disorder (ADHD)**
 - a) Predominantly inattentive
 - b) Hyperactive/Impulsive
 - c) Combined
- 8) Pervasive developmental disorder**
 - a) Autistic disorder
 - b) Rett disorder/syndrome
 - c) Childhood disintegrative disorder
 - d) Asperger disorder
- 9) Communication and language**
- 10) Child abuse or neglect**
- 11) Chronic medical disease**
- 12) Tic disorder**
- 13) Drug or alcohol dependence**

Key Objectives

- Make a clinical assessment of a child's developmental level.
- List the criteria of ADHD.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine whether environmental factors in school or family may be contributing to school failure.
 - Determine whether there is a family history for attention deficit or any of the comorbid conditions.
 - Determine whether there is evidence of developmental delay, genetic syndromes, encephalopathies, or poisoning (e.g. alcohol, lead).
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select patients who require further investigation or psychological testing.
- Conduct an effective plan of management for a patient with school failure:
 - Select patients in need of specialised care.
 - Along with other caregivers, outline a management plan which includes (when appropriate):
 - * Parent, child and teacher education and other educational interventions.
 - * Structured educational and recreational activities.
 - * Behavioural management strategies.
 - * Psychological counselling.
 - * Medication.
 - Discuss the use of medication (amphetamine derivatives) in the treatment of ADHD.

014 Behaviour Disorder

(See also #026 Development Disorder / Developmental Delay)

Overview

Clinicians are usually the initial caregivers to be confronted by the developmental and behavioural problems of childhood and adolescence, which can lead to impaired social, academic and occupational functioning. The behaviour disorders are more common in males, eating disorders are more common in females. The early diagnosis of autism is particularly difficult.

Causes

- 1) Attention deficit hyperactivity (hyperkinetic) disorder (ADHD)**
- 2) Oppositional defiant / Conduct disorder**
- 3) Eating disorders (anorexia nervosa / bulimia, pica, rumination disorder)**
- 4) Sleep disorders (nightmares, sleep terror disorder, sleepwalking, enuresis)**
- 5) Tic disorders (Tourette syndrome, motor or vocal tic disorders)**
- 6) Autism**

Key Objective

- Determine whether the patient has a behaviour disorder or some other underlying medical condition, mood disorder or a comorbid developmental disability.

General/Specific Objectives

- Through efficient, focused data gathering, which includes assessment of family history, context and culture:
 - Determine whether the patient requires consultation with a psychiatrist/psychologist for oppositional defiant or conduct disorder.
 - Determine whether criteria for anorexia/bulimia are present (on history and physical examination).
 - Determine whether a patient with a tic disorder requires referral to a paediatrician, neurologist, or child psychologist.
 - Determine whether a patient with a sleep disorder requires further investigation or specialist referral.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select patients requiring further investigation.
- Conduct an effective plan of management for a patient with behaviour disorder:
 - Discuss indications for hospital admission in a patient with an eating disorder.
 - In a patient with an eating disorder, outline a management plan including (when appropriate) psychotherapy (individual and family), behaviour modification techniques, nutritional therapy, and medication.

Overview

A bleeding tendency can manifest with cutaneous and/or systemic features. It may signify a drug-induced disorder, a problem of haemostasis or a vessel abnormality. Taking a detailed history (including a family history) is essential.

Causes

1) Platelet problem

a) Decreased number

- Decreased production (e.g. leukaemia)
- Increased destruction
- Abnormal sequestration

b) Abnormal function

- Congenital
- Acquired
 - Drugs (aspirin)
 - Renal disease

2) Coagulation factor problem

a) Congenital

- Factor VIII deficiency
- von Willebrand disease
- Factor IX deficiency

b) Acquired

- Liver disease
- Anticoagulants
- Disseminated intravascular coagulation (DIC)
- Vitamin K deficiency
- Inhibitors
- Drugs (heparin-induced thrombocytopenia)
- Massive blood transfusion

3) Vessel problem

a) Congenital (collagen disorders)

b) Acquired (steroids, vasculitis)

Key Objectives

- Understand the role of a clinical history in determining the cause of a bleeding disorder.
- Be aware of the appropriate clinical signs and tests that will help to determine the underlying cause of a bleeding disorder.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate between platelet problems, coagulation factor problems, and vessel problems.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Select investigative tests appropriate for platelet, coagulation, and vessel problems.
 - Contrast the results and their interpretation.
 - Select 'at risk' families for investigation of potentially affected children.
- Conduct an effective plan of management for a patient with bleeding tendency or bruising:
 - Select platelet transfusions, vitamin K, and plasma derivatives in the management of patients with bleeding disorders according to the diagnosis made.
 - Formulate a management plan for the reversal of the anticoagulant effect of heparin, warfarin or aspirin.
 - Select patients in need of specialised care.



Retroperitoneal haemorrhage following warfarin

016 Bleeding with Defaecation / Acute Lower Gastrointestinal Bleeding / Melaena / Occult Blood in Stool / Prevention of Cancer

Overview

Occult gastrointestinal bleeding may be due to serious gastrointestinal disease (carcinoma of the colon or stomach). Screening the stool for occult blood in high-risk groups may increase diagnostic yield.

Bright red blood unmixed with the stool noticed at the time of defaecation is usually due to a benign anorectal cause, but other associated serious pathology must be excluded.

Acute lower gastrointestinal bleeding with fresh blood and clots independent of defaecation is usually colonic and associated with colonic diverticula. Localisation of the bleeding source can be difficult.

Melaena (a black tarry stool), with or without haematemesis, almost invariably signifies significant upper gastrointestinal haemorrhage.

Causes

1) Upper gastrointestinal bleeding with melaena

(see #048 Haematemesis/Melaena)

2) Lower gastrointestinal bleeding

- a) Colorectal cancer/polyp
- b) Anorectal disease (haemorrhoids, anal fissure, acute or chronic)
- c) Colonic diverticulosis
- d) Angiodysplasia
- e) Enterocolitis (ischaemic, infectious, inflammatory bowel disease (IBD), nonsteroidal anti-inflammatory drugs (NSAIDs))
- f) Other (small bowel neoplasms, Meckel diverticulum)

3) Occult blood in stool



Key Objective

- List the three key steps in the management of the separate presentations of lower gastrointestinal bleeding as: resuscitation and assessment; localisation; and diagnosis and treatment.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Define the relationship between bleeding and defaecation.
 - Undertake an appropriate examination to determine the cause of the bleeding.
 - List and diagnose the most likely cause of blood in the stool.
 - Identify patients requiring urgent assessment and treatment.
 - List and diagnose the presence of associated medical conditions predisposing to the development of colorectal cancer.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - List advantages and disadvantages for anoscopy, rectoscopy, sigmoidoscopy, colonoscopy versus barium studies, radionuclide imaging, and angiography in patients with blood in the stool and the appropriate time to perform these.
 - Select asymptomatic patients in need of screening for colorectal cancer.
 - Outline the diagnostic value and limitations of contrasting haematochezia (fresh blood in stool) and melaena.
- Conduct an effective plan of management for a patient with blood in stool:
 - Select patients in need of immediate therapy.
 - Contrast diagnostic and management plans for patients with persisting acute lower gastrointestinal haemorrhage with plans for evaluation of intermittent passage of bright blood unmixed with the stool.
 - Evaluate patients in a cost-effective manner.
 - Outline the assets and limitations of screening using faecal occult blood testing.
 - Select patients in need of specialised care.

017A Male (Gynaecomastia)

Overview

Physiologic gynaecomastia is common in the newborn, in adolescence, and in males over 50 years of age. In pathologic gynaecomastia, a definite aetiology is found in the minority; but a careful drug history is important to detect a treatable cause, as is clinical screening for occult malignancy. An underlying feature is increased oestrogen to androgen ratio.

Causes

1) Physiologic gynaecomastia

- a) Newborn
- b) Adolescence
- c) Ageing (50–80 years; decreased testosterone or increased binding globulin)

2) Pathologic gynaecomastia

a) Deficient production or action of testosterone

- Anorchia
- Defects in testosterone synthesis
- Orchitis
- Renal failure

b) Increased oestrogen production

- Testicular tumours
- Other tumours producing human chorionic gonadotropin (hCG)
- Klinefelter syndrome
- Hyperthyroidism
- Liver disease
- Obesity
- Malnutrition/Starvation

c) Drugs

- Oestrogens/Oestrogen-like (oral contraceptive pill (OCP), digitalis)
- Anabolic steroids in body-builders
- Inhibitors of testosterone synthesis or action (spironolactone, cimetidine, flutamide)
- Other drugs (methyldopa, captopril, tricyclics)

d) Idiopathic

Key Objectives

- Differentiate between gynaecomastia and breast cancer.
- Differentiate between gynaecomastia and pseudogynaecomastia (fat deposition without glandular proliferation).

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate patients with gynaecomastia due to physiologic or pathologic causes.
- Identify patients who require further investigation.
- Interpret the critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret laboratory tests in the investigation of gynaecomastia.
 - Select and interpret imaging tests and cytology/histology in the investigation of gynaecomastia.
- Conduct an effective plan of management for a patient with gynaecomastia:
 - Diagnose patients with physiologic gynaecomastia who require no specific therapy.
 - Diagnose patients with drug-induced gynaecomastia who would benefit from withdrawal of the drug.
 - Identify patients requiring surgery.



Gynaecomastia

017B Female (Breast Lump / Prevention of Cancer / Screening)

Overview

Breast cancer is the most common cancer in women. One in 13 Australian women will develop breast cancer in their lifetime. Screening women over 50 years and other high-risk groups by regular two-yearly mammography improves survival and identifies small preclinical lesions. A small but significant proportion of patients have familial cancer in whom genetic screening and counselling may be helpful.

Causes

1) Breast carcinoma (the most important, although not the most common cause of a breast lump)

a) Non-invasive

- Ductal carcinoma-*in-situ* (DCIS)
- Lobular carcinoma-*in-situ* (LCIS)

b) Invasive

- Invasive ductal carcinoma
- Invasive lobular carcinoma
- Others (tubular, medullary, papillary, mucinous)

2) Diffuse nodularity – fibrocystic change

3) Discrete benign breast lumps ('dominant lumps')

- a) Localised fibrocystic change
- b) Gross cysts
- c) Galactoceles
- d) Fibroadenomas
- e) Traumatic fat necrosis
- f) Mammary duct ectasia

4) Breast infections

- a) Associated with lactation – lactational mastitis / breast abscess
- b) Not associated with lactation – mammary duct ectasia, subareolar abscess, mamillary fistula

5) Rarer causes – phyllodes tumour (usually benign – and only locally invasive, occasionally true sarcoma)

Key Objectives

- Ability to perform a standardised clinical breast examination, ensuring correct patient comfort and positioning, and appropriate technique.
- Ability to distinguish normal from abnormal and suspicious findings.
- Understanding risk factors for development of breast cancer in women.
- Understanding the investigations for mammography and breast ultrasound and the appropriateness of each for different clinical situations and age groups.
- Understanding the principles of management of breast cancer by surgery and adjuvant means.

General/Specific Objectives

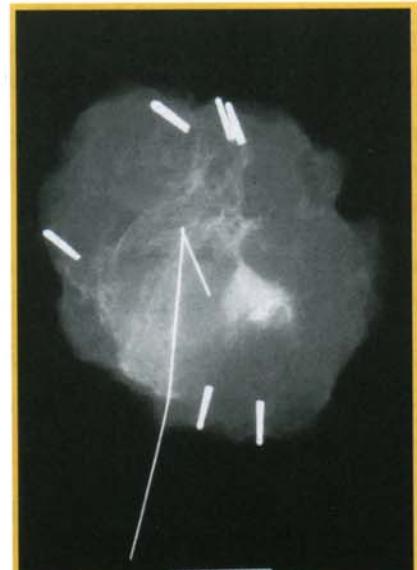
- Through efficient, focused data gathering:
 - Determine which women are at high risk for breast cancer.
- Interpret critical clinical and laboratory findings, which were key in the processes of exclusion, differentiation and diagnosis.
- Identify groups based on age or other pre-existing risk factors for regular screening mammography; outline benefits and drawbacks of screening programmes.
- Identify families in whom genetic screening and counselling may be considered; outline the benefits and drawbacks of genetic screening.
- Counsel and educate patients on the role of breast self-examination.
- Conduct an effective diagnostic/management plan for a patient presenting with a breast lump:
 - Outline an algorithm for diagnosis of a patient presenting with a breast lump. Which patients do NOT require surgery?
 - Outline the indications for percutaneous fine needle aspiration cytology (FNAC) and core biopsy in patients with breast pathology.
 - Outline the indications for surgery, radiotherapy, hormonal anti-hormonal therapy, and chemotherapy in women with breast cancer.
 - Outline the indications for breast-conserving surgery in women with breast carcinoma.
 - Counsel women with risk factors for the development of breast cancer on the utility of screening.



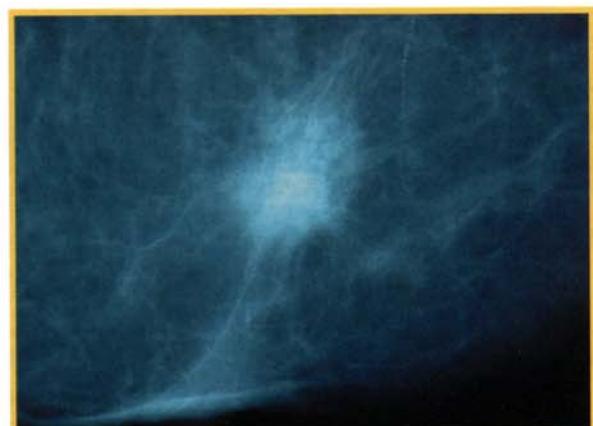
Breast cyst – mammogram and ultrasound



Screening mammogram



Screen-detected cancer excision



Mammogram – carcinoma of breast

017C Nipple Discharge / Galactorrhoea

Overview

Nipple discharge is a common symptom of concern to the patient. Spontaneous discharges are of more significance than those evoked only by squeezing, as are bloody discharges from a single duct. In the absence of an accompanying lump, the cause is almost always benign. Although milky breast secretions may be noticeable (and are normal) in 25% of previously pregnant women, spontaneous persistent galactorrhoea may reflect underlying breast or endocrine disease and requires investigation.

Causes

1) Nipple discharge

- a) Bloody: breast neoplasm, usually benign duct papilloma
- b) Serous, green, yellow-brown: usually benign fibrocystic change
- c) Toothpaste, worms: mammary duct ectasia

2) True galactorrhoea (fat droplets present)

a) Autonomous prolactin production

- Pituitary tumours (micro- or macro-adenoma)
- Ectopic production of prolactin (bronchogenic or renal cell cancer)

b) Enhanced prolactin release

- Hypothyroidism
- Sucking reflex

c) Failure to inhibit release of prolactin

- Pituitary stalk section or compression by mass lesion
- Drugs (phenothiazines, methyldopa, opiates)

d) Idiopathic (most common cause)

Key Objectives

- Identify patients with nipple discharge requiring surgery.
- Differentiate between nipple discharge and galactorrhoea.
- Differentiate physiological from pathological galactorrhoea.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Identify patients with spontaneous or evoked, unilateral or bilateral, single or multiple duct discharges and different types of discharge.
 - For galactorrhoea, determine which patients have menstrual irregularities or visual field defects since they are likely to have an underlying disease.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret laboratory tests and diagnostic imaging in patients with nipple discharge/galactorrhoea.
- Outline an effective plan of diagnosis and management for a patient with nipple discharge:
 - Determine which patients are likely to have a breast neoplasm.
 - Outline the role of surgery in patients with nipple discharge
 - List the medications which can cause galactorrhoea.
 - Outline the role of bromocriptine and other dopamine agonists in the management of patients with hyperprolactinaemia and galactorrhoea.
 - Counsel and educate patients with chronic galactorrhoea how the galactorrhoea may be minimised.

017D Breast Pain (Mastalgia)

Overview

Breast pain is a very common symptom in women. It is rarely caused by malignant disease, and pain severity varies widely. Mastalgia is most common between the ages of 30–50 years; it is unusual after the menopause, apart from those patients on hormone replacement therapy (HRT). Pain may be cyclical or non-cyclical, bilateral or unilateral. Pain is a feature of acute infections (mastitis / breast abscess) complicating lactation.

Causes

- 1) Cyclical mastalgia and nodularity (two-thirds)**
- 2) Non-cyclical mastalgia**
- 3) Focal mastitis/abscess**
- 4) Extramammary causes (costochondritis / Tietze syndrome, musculoligamentous strain, etc.)**



Left breast abscess

Key Objectives

- Exclude focal treatable lesions (inflammations, infections, neoplasms).
- Arrange treatment according to severity and with appropriate reassurance as to benign aetiology.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Identify focal or diffuse infective mastitis and treat appropriately with antibiotics, expression or drainage.
- Interpret critical clinical and laboratory findings which were key in the process of exclusion, differentiation and diagnosis.
 - Select appropriate investigations in a patient with mastalgia.
- Conduct an appropriate step-wise plan of management for a patient with mastalgia.

017E Breast – Skin Changes

Overview

Skin changes involving the breast, nipple and areola may indicate tethering or invasion from an underlying carcinoma. Physical examination should be meticulous to identify suspicious features.

Causes

1) Nipple and areola

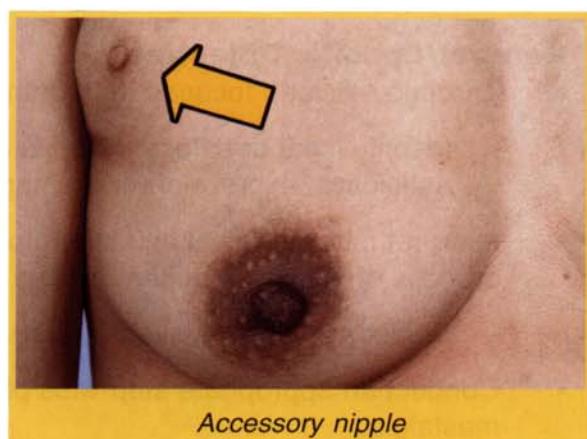
- a) Paget disease – underlying duct carcinoma
- b) Simple dermatitis/eczema
- c) Benign squamous papilloma of nipple, accessory nipple(s)
- d) Enlarged/Inflamed sudoriferous gland (Montgomery follicle)
- e) Retraction – significant if recent and fixed (underlying carcinoma)

2) Skin of breast

- a) Skin dimple and tethering over carcinoma (may only be evident on raising arms)
- b) '*Peau d'orange*' – due to dermal oedema (neoplastic or inflammatory)
- c) Subcutaneous 'string' from lymphangitis (Mondor disease)
- d) Intertrigo (common beneath pendulous large breasts)
- e) Mamillary sinus/fistula (secondary to mammary duct ectasia)
- f) 'Pseudolipoma' – (prominence of compressed fat overlying a deeper carcinoma)



Paget disease of nipple



Accessory nipple



Cancerous skin dimpling



Peau d'orange

Key Objectives

- Ability to identify skin, nipple and areola abnormalities on physical examination.
- Ability to identify suspicious lesions requiring further investigation.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Identify abnormalities of skin and nipple requiring further assessment.
- Interpret critical clinical and laboratory findings which were key in the process of exclusion, differentiation and diagnosis.
 - Outline appropriate diagnostic and management plans for a patient presenting with nipple eczema.
 - Conduct appropriate management plans for a patient with a mamillary fistula.

(See also #113C Burn Injuries)

Overview

Burns range from minor cutaneous wounds to massive life-threatening traumas, and remain a frequent cause of accidental death, and of gross burn morbidity. Many domestic and industrial accidents are preventable. Public education concerning risks and their avoidance is of major importance.

Causes

- 1) Scalds (hot water spills are commonly partial thickness, molten metal spills cause full thickness localised burns)**
- 2) Flame burns (commonly full thickness)**
- 3) Burns from radiant heat and hot objects**
- 4) Electrical burns (high amperage and voltage electrical burns add risks of electrocution)**
- 5) Chemical burns (cause additional damage by continuing contact)**
- 6) Requiring special care**
 - a) Partial thickness (second degree) and full thickness (third degree) greater than 10% body surface area (BSA) in patients aged less than 10 and more than 50 years or greater than 20% any age
 - b) Second and third degree greater than 15% BSA require intravenous (IV) replacement; greater than 20% BSA require urinary catheter
 - c) Second and third degree on face, hands, feet, genitalia, perineum, major joints
 - d) Third degree greater than 5% BSA
 - e) Electrical burns (including lightning) and chemical burns
 - f) Circumferential burns
 - g) Burns plus other serious illness

Key Objectives

- Perform assessment and initial treatment of burn patients according to emergency management of severe trauma (EMST) protocol: primary survey, secondary survey, etc.
- Diagnose burns according to:
 - Percentage BSA involved ('rule of nines', modified in children).
 - Depth of skin injury.
 - Partial thickness burns (first and second degree) – erythema, blistering, moist exudates, soft, painful to pinprick, circulation present.
 - Full thickness burns (third and fourth degree) – dull white, opaque, brown and charred, visible thrombosed veins, dry, firm, painless to pinprick, no capillary response.
- Outline effective management plans for:
 - The burned patient.
 - The burn wound.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine the BSA affected first, since depth is difficult to determine initially.
 - After 24 hours, determine depth of skin injury (first degree to fourth degree).
 - Determine whether there are other associated clinical problems or other trauma.
 - Determine patient's tetanus immunisation status.
 - Determine whether inhalation injury has caused respiratory distress.



Superficial scald burns

- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Determine whether carbon monoxide poisoning has occurred by measuring carboxyhaemoglobin.
- Conduct an effective plan of management for a patient with severe burns:
 - Outline initial management in a burn patient who will require referral including stopping further burn injury, covering of burn area, and resuscitation with oxygen, IV fluids, and physiologic monitoring.
 - Outline initial topical antibacterial treatment.
 - Discuss mechanism of injury of electrical burns and need for cardiac and renal monitoring.
 - Select patients in need of specialised care.
- Outline an appropriate initial plan of IV fluid replacement (e.g. %BSA x kg weight x 2 ml fluid in first 24 hours – one-third first 4 hours, one-third next 8 hours, one-third next 12 hours).



Full thickness burns – legs



Superficial burns – face

Overview

Most cases of cardiac arrest occur secondary to a cardiac arrhythmia. The ability to perform and manage cardio-pulmonary resuscitation effectively is a pre-requisite for all medical graduates.

Causes

1) Tachyarrhythmias (marked)

- a) Ventricular fibrillation/tachycardia
- b) Atrial fibrillation/flutter

2) Bradyarrhythmias / Asystole

- a) Sinus bradycardia / Arrest / Sick sinus syndrome
- b) Third degree block (slow/absent escape rhythm)

3) Acute vascular occlusion

- a) Myocardial infarction (MI)
- b) Obstruction of cardiac filling
 - Pulmonary embolus (massive)
 - Acute cardiac tamponade
 - Tension pneumothorax
- c) Mechanical heart valve blockage

4) Cardiac/Vascular ruptures

- a) Type I dissecting aortic aneurysm
- b) Ventricular rupture
- c) Mitral papillary muscle rupture with torrential mitral regurgitation

5) Vasodepressor collapse

- a) Neurocardiogenic collapse
- b) Hypersensitive carotid sinus syndrome
- c) Marked orthostatic hypotension

Key Objective

- Be confident and competent in your ability to manage a cardio-pulmonary arrest.

General/Specific Objectives

- Conduct an effective plan of management for a patient with cardiac arrest / respiratory arrest:
 - Evaluate the status of the airway and provide respiratory support as indicated.
 - Demonstrate the techniques of cardiopulmonary resuscitation (CPR) according to the age of the patient.
- Through efficient, focused data gathering:
 - Identify and interpret quickly the signs of impending and actual cardiac and/or respiratory arrest.
 - Differentiate between the possible causes of the cardiac and/or respiratory arrest.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret appropriate investigations for patients presenting with cardiac and/or respiratory arrest, including electrocardiography, chest X-ray, serum electrolytes, and blood gases.
 - If the resuscitation attempt was not successful, communicate, with sensitivity, the news of death to family members and discuss the possibility of an autopsy if indicated; if resuscitation is successful, communicate with sensitivity, the news to the family and answer all pertinent questions.

Overview

Chest pain may be central or peripheral. Central chest pain is a common presentation of cardiac disease, but it may also be due to disease of the lungs, gastrointestinal tract or a musculoskeletal disorder. Coronary artery disease (CAD) is a potential life-threatening disease. Doctors must recognise the manifestations of CAD and the key characteristics that help to distinguish cardiac pain from other causes of chest pain.

Causes

1) Ischaemic heart disease

- a) Acute myocardial infarction (MI)
- b) Angina pectoris (stable, unstable, microvascular, coronary spasm)

2) Mitral valve prolapse

3) Aortic dissection

- a) Hypertensive
- b) Cystic medial necrosis, Marfan syndrome, Ehlers-Danlos syndrome
- c) Connective tissue disease
- d) Syphilis

4) Pericarditis

- a) Infective
 - Viral (Bornholm disease)
 - Bacterial
- b) Noninfective
 - Post-myocardial infarction
 - Post-coronary artery bypass graft (CABG)
 - Uraemic
 - Connective tissue disease

5) Pulmonary causes (embolism, pneumonia, pleuritis, pneumothorax, etc.)

6) Chest wall origin

- a) Costochondritis (Tietze syndrome)
- b) Herpes zoster

7) Psychogenic

- a) Anxiety, hyperventilation syndrome
- b) Cardiac neurosis

8) Neoplasia (lung carcinoma, mediastinal malignancy)

Key Objectives

- Know the key characteristics that help to distinguish cardiac and non-cardiac sources of chest pain.
- Differentiate between MI and other forms of CAD early, in order to take advantage of potential life-saving therapy.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate cardiac pain from other types of visceral pain.
 - Differentiate MI from unstable and stable angina.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Select and interpret electrocardiograms (ECGs) and cardiac enzymes, and discuss newer biochemical markers (such as troponin).
 - Select and interpret diagnostic imaging of the chest.
- Conduct an effective plan of management for a patient with chest pain:
 - Outline initial management of stable and unstable angina, acute MI, and other causes of chest discomfort.
 - List indications and contraindications of thrombolytic therapy and list potential complications.
 - Select patients in need of specialised care and/or consultation.
 - Counsel patients with chest pain caused by life-threatening conditions and counsel their families.
 - Identify the coronary risk factors and define a plan of management for these where appropriate.
 - State the long term management of patients after MI, including secondary prevention strategies.
 - Select cost-effective investigative and therapeutic modalities.
 - Discuss primary and secondary preventive strategy education for patients with ischaemic heart disease.

Overview

Patients with altered level of consciousness account for five percent of hospital admissions. Causes range from those which are rapidly treatable and recoverable, to those causing severe morbidity and mortality. Management of prolonged coma requires expert and intensive nursing and medical care and monitoring for changing levels of consciousness.

Causes

1) Metabolic encephalopathy

- a) Drugs or toxins (e.g. alcohol)
- b) Electrolyte abnormalities (hyponatraemia/hypernatraemia, hypercalcaemia, hypoglycaemia)
- c) Liver or renal failure
- d) Hypertensive encephalopathy
- e) Hypoxaemia/Hypercapnia
- f) Sepsis (systemic)

2) Structural brain damage

- a) Hemispheric (haemorrhage, ischaemia/infarction, neoplastic, traumatic)
- b) Brainstem (haemorrhage, ischaemia/infarction, neoplastic, traumatic)

3) Infectious

- a) Central nervous system (CNS) (meningitis, encephalitis)
- b) Non-CNS (sepsis)

4) Miscellaneous

- a) Seizure (post-ictus)
- b) Myxoedema

5) Malingering

Key Objective

- Diagnosis and management of coma relies on the knowledge of the potential causes, an interpretation of simple clinical signs and the efficient use of diagnostic tests.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Determine the most likely cause for, and seriousness, of coma by means of physical examination leading to rational investigation.
 - Conduct a clinical assessment of the level of consciousness.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret laboratory investigations for patients suspected of metabolic encephalopathy.
 - Select diagnostic imaging appropriate for comatose patient.
- Conduct an effective plan of management for a patient with coma/impaired consciousness:
 - Define level of consciousness utilising the Glasgow Coma Scale.
 - Select patients in need of immediate therapy and perform initial treatment.
 - Select patients in need of specialised care.
 - Outline potential issues of importance in the ethical management of the incompetent patient, including those of consent for treatment and advanced directives.
 - Conduct assessment for suspected brain death prior to referring patient to neurological specialist for the definitive diagnosis of brain death.

Overview

Delirium is a common and serious problem, particularly in the elderly, the hospitalised and postoperative patients. It represents a disturbance of consciousness and cognitive impairment with reduced ability to focus, sustain, or shift attention (The Diagnostic and Statistical Manual 4 – Text Revision (DSM-IV-TR)). This disturbance tends to develop over a short period of time (hours to days) and tends to fluctuate during the course of the day. It is often associated with a disturbance of both the sleep-wake cycle and psychomotor behaviour. It may be superimposed on a dementing process or it may have multiple contributing factors. A clear understanding of the differential diagnosis enables rapid and appropriate treatment.

Causes

1) Systemic

a) Intoxication/Withdrawal

- Drugs (opiates, psychotropics, anticholinergics, corticosteroids, cannabis, alcohol, amphetamines, hallucinogens)
- Withdrawal (alcohol, opiates, psychotropics)
- Poisoning / Toxins / Heavy metals

b) Metabolic

- Hypoxaemia
- Electrolyte disturbances (hyponatraemia/hypernatraemia, hypercalcaemia)
- Hypoglycaemia
- Organ failure (uraemia, hepatic encephalopathy, hypoxaemia, hypercarbia, heart failure)
- Hypertensive encephalopathy
- Hypoalbuminaemia
- Porphyria
- Thiamine deficiency

c) Miscellaneous

- Systemic sepsis, pneumonia, urinary tract infections (UTIs), encephalitis, acquired immune deficiency syndrome (AIDS)
- Postoperative states (residual anaesthetics, stress, sleep deprivation, cataract surgery, fat embolism, anaemia)
- Endocrinopathies (thyroid, adrenal)
- Burns/Electrocution
- Hyperthermia

2) Local (central nervous system (CNS))

- a) CNS infections**
- b) Acute vascular events (stroke, migraine, vasculitis, carotid stenosis)**
- c) Neoplasm and paraneoplastic processes**
- d) Epilepsy**
- e) Post-electroconvulsive therapy**
- f) Post-head injury**

Key Objective

- Differentiate delirium due to general medical conditions from dementia, drug intoxication or withdrawal, psychotic disorders, personality disorders or malingering and factitious disorder.

General/Specific Objectives

- Through efficient, focused data gathering (which will frequently involve interviewing other informants):
 - Determine which patients are at risk for the development of delirium.
 - Diagnose the underlying causes for delirium.
 - Contrast delirium and dementia (a potent risk factor for delirium); categorise a sudden change in behaviour in a patient with dementia as possible delirium superimposed on dementia.
- Interpret critical clinical and laboratory and mental state findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select and interpret laboratory investigations in a patient with delirium.
 - List the indications for radiological imaging of the brain in a patient with delirium.
- Conduct an effective plan of management for a patient with confusion/delirium:
 - Outline the initial emergency management of patients with delirium including protection of the patient from self-inflicted harm, harm to others and methods to reduce disorientation and anxiety.
 - Describe the specific management of patients with delirium due to hepatic encephalopathy, metabolic abnormalities and drugs.
 - Select patients in need of specialised care.
 - Inform and support relatives.

Overview

Cyanosis is the physical sign of hypoxaemia, but at times is difficult to detect (cyanosis must be sought carefully, under proper lighting conditions). Hypoxaemia (low partial pressure of oxygen in blood), when detected, may be reversible with oxygen therapy after which the underlying cause requires diagnosis and management.

Causes

1) Central cyanosis

a) Lung disease

- Upper airway obstruction
- Pulmonary embolism
- Interstitial
 - Infectious
 - Inorganic dust (silicosis, asbestosis, coal, metals, etc.)
 - Associated with other diseases (sarcoid, vasculitis, etc.)
 - Chronic pulmonary oedema
 - Idiopathic pulmonary fibrosis / fibrosing alveolitis
 - Lymphangitic carcinomatosis
- Chronic obstructive airways disease

b) Cyanotic heart disease

- Eisenmenger syndrome (pulmonary hypertension with right-to-left shunt)
- Fallot tetralogy (ventricular septal defect (VSD); right ventricular outflow obstruction; overriding aorta; right ventricular hypertrophy)
- Transposition of great vessels
- Total anomalous pulmonary venous drainage, truncus, single ventricle

2) Peripheral cyanosis

a) Low cardiac output

b) Local flow diminished (arterial/venous obstruction)

3) Localised cyanosis

Key Objectives

- Define cyanosis, hypoxaemia, and hypoxia (insufficient levels of oxygen in tissues to maintain cell function).
- Contrast pathophysiology of central and peripheral cyanosis.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Differentiate central cyanosis from peripheral and localised cyanosis.
 - Contrast respiratory causes and cyanotic congenital heart disease.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis.
- Conduct an effective plan of management for a patient with cyanosis/hypoxaemia/hypoxia:
 - Outline an initial plan of management which includes treatment of the underlying condition along with oxygen administration.
 - List the adverse effects of oxygen treatment.
 - List useful outcome criteria for a trial of long term use of oxygen in patients with chronic hypoxaemia.

023A Cyanosis / Hypoxia / Apnoea in Children

Overview

Evaluation of cyanosis and hypoxia in children depends heavily on the age of the child. Cyanosis is an ominous finding, especially in the older child, and differentiation between peripheral and central is essential in order to mount appropriate management.

Causes of cyanosis or hypoxaemia

1) Neonatal

a) Central

- Cyanotic congenital heart disease
 - Increased pulmonary blood flow (transposition, truncus arteriosus, total anomalous pulmonary venous return, hypoplastic left heart)
 - Obstruction to pulmonary blood flow (tricuspid, pulmonary atresia)
- Respiratory insufficiency
 - Pulmonary (respiratory distress syndrome, sepsis, aspiration, diaphragmatic hernia)
 - Central nervous system (CNS) (maternal sedative, asphyxia, intracranial haemorrhage, hypoglycaemia)

b) Peripheral vascular ('physiologic acrocyanosis', sepsis, cardiogenic/septic shock, thrombosis, vasomotor instability)

2) Infant and child

a) Central

- Decreased oxygenation of haemoglobin
 - Respiratory (pneumonia, cystic fibrosis, embolus, aspiration / foreign body, CNS depression)
 - Cardiac disease – cyanotic congenital heart disease, severe congestive cardiac failure (CCF) from any cause
- Abnormalities of haemoglobin (methaemoglobinaemia)

b) Peripheral

- Vascular problem (Raynaud disease, sepsis)
- Obstruction (superior vena cava syndrome, deep venous thrombosis (DVT))
- Hyperviscosity (polycythaemia)

Key Objectives

- Differentiate between peripheral and central cyanosis since generalised cyanosis is more consistent with primary heart disease or respiratory insufficiency.
- Appreciate that if the process causing peripheral cyanosis is severe enough (e.g. sepsis), generalised cyanosis may occur.

General/Specific Objectives

- Through efficient, focused data gathering:
 - Elicit maternal history of illness or sepsis in pregnancy, gestational age, delivery complications, presence of meconium, suction of infant, Apgar score, family history of congenital heart disease.
 - Determine the vital signs, age of infant (ductus arteriosus usually closes by third day), whether the infant is alert and active, if the infant is able to feed, and the presence of respiratory distress (tachypnoea, grunting, costal margin flaring or retraction).
 - Perform examination of the newborn for evidence of respiratory distress, congestive heart failure or shock, signs of CNS depression, whether the cyanosis is central or peripheral.
 - Elicit history in the older child of acute versus chronic or recurrent cyanosis, history of lung disease or heart disease, history of foreign body or aspiration, fever, upper respiratory symptoms, exposure to medications, dyes, chemicals.
 - In the older child, focus examination first on respiratory distress and obtundation of neurologic disease; determine whether hypotension or bradycardia is present (ominous signs).
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation and diagnosis:
 - Select laboratory investigations including diagnostic imaging, electrocardiogram (ECG), and blood tests.
 - Explain the interpretation of hyperoxia test (arterial blood gas from a site distal to the ductus on room air and 100% oxygen).
- Conduct an effective plan of management for a patient with cyanosis / hypoxia / apnoea in children:
 - Outline initial management including cardio-respiratory monitoring.
 - Explain the benefit of 'knee-chest' position in a child with cyanosis and tetralogy of Fallot.
 - Select patients in need of specialised care.

Overview

Dementia is an acquired, progressive impairment of cognitive function characterised by memory impairment, accompanied by other intellectual and personality changes, in the setting of full consciousness. Dementia is a common problem, with most cases being irreversible, the commonest cause being Alzheimer disease (more than 50% of cases). Progress may be temporarily arrested or modified with specific treatments, and potentially treatable causes must be sought.

Causes

- 1) Primary dementias (Alzheimer disease, Lewy body dementia, Niemann-Pick disease, fronto-temporal dementia)**
- 2) Vascular**
 - a) Multi-infarct
 - b) Vasculitis / Autoimmune diseases
 - c) Focal subcortical strokes
- 3) Toxic**
 - a) Alcohol, drugs and narcotics
 - b) Heavy metals / Dialysis dementia
 - c) Organic toxins
 - d) Carbon monoxide
- 4) Brain trauma (head injury, boxing, hypoxia)**
- 5) Chronic infections (HIV, syphilis, Creutzfeldt-Jakob disease, herpes simplex, malaria)**
- 6) Mass lesions and/or neoplasms**
 - a) Primary and secondary tumours, carcinomatous meningitis, paraneoplastic encephalitis
 - b) Chronic subdural haematoma
 - c) Normal pressure hydrocephalus
- 7) Movement disorders**
 - a) Parkinson disease
 - b) Huntington chorea

8) Endocrine, metabolic, and vitamin deficiency

- a) Hypothyroidism/Hyperthyroidism
- b) Hypoparathyroidism/Hyperparathyroidism (HPT)
- c) Hypoglycaemia/Hyperglycaemia (chronic)
- d) Hypopituitarism
- e) Pyridoxine, vitamin B₁₂ and thiamin deficiency

9) Depressive pseudodementia

Key Objective

- Assess and identify treatable and reversible causes of cognitive dysfunction, including the early stages of Alzheimer-type dementia.

General/Specific Objectives

- Through efficient, focused data gathering involving other informants as well as the patient:
 - Establish the history of onset and progression of symptoms and current level of functioning including daily living activities.
 - Conduct a baseline mental status assessment, including a Folstein Mini-Mental State Examination (MMSE) and tests of frontal lobe functioning.
 - Using the MMSE, differentiate depression or delirium from dementia.
 - Perform a comprehensive physical examination including neurological, cardiovascular and endocrine systems and sensory impairments.
 - Categorise possible causes of dementia, and perform screening investigations.
- Interpret critical clinical and laboratory findings which were key in the processes of exclusion, differentiation, and diagnosis:
 - Identify and treat correctable conditions.
 - Know the indications for centrally acting cholinergic agents.
 - Determine the need for antidepressant or antipsychotic medication, or psychiatric referral.
 - Assess and determine the role of the family or primary carer in the support of the patient. Break the news sensitively.
 - Determine whether occupational and social or other therapy referral and assessment is needed after considering the availability of community resources.