

CLUES TO DIAGNOSES
IN
EMERGENCY DEPARTMENTS
(EDs)

Aziz Aboo

April 2008

CONTENTS

Introduction	3
1. The Focussed General Examination	4
2. Acute Chest Pain	10
3. Acute Shortness of Breath	12
4. Heart Failure	15
5. Stridor	17
6. Abdominal Disorders	18
7. Headache	21
8. The Dizzy Patient (and Stroke)	22
9. Blackouts, Strange Behaviour and Related Disorders	25
10. Musculo-Skeletal Disorders	30
11. Loss of Weight	34
12. Factors causing acute and subacute deterioration of chronic medical disorders	35
13. Managing bleeding/prolonged INR in patients on Warfarin	37
14. Poisoning and Overdose	39
15. Interpretation of some common investigations in EDs	42

INTRODUCTION

Overcrowding in public hospital Emergency Departments (EDs) adversely affects the optimal management of emergencies and the training of emergency physicians. Much of this overcrowding is due to the chronically ill (HIV, TB, diabetes, hypertension), diagnostic and therapeutic problems which should be seen at outpatient departments and the mentally disturbed as a result of substance abuse (in urban areas). These patients are often seen by junior medical staff with little senior support. This booklet is an attempt to assist junior medical staff, medical students and nurse practitioners in EDs to identify serious problems, plan investigations and refer those who could be treated at OPDs. Some “emergencies” are discussed here but there is little emphasis on treatment as many protocols and guidelines by experts are well circulated in EDs. I might add that when following protocols health practitioners should always take into account certain patient factors eg. a morbidly obese diabetic (NIDDM) is better off with a dietitian (for health and financial reasons) than with a medical practitioner or managing hypertension in an alcoholic patient who is not prepared to have his/her addiction treated will be a futile exercise. My main focus will be on the group of patients described above.

I have concentrated on common disorders and emphasised signs and symptoms often overlooked by junior doctors in ED's. We still see far too many patients with disseminated cancers and advanced tuberculosis whose chief complaints were overlooked on previous presentations at primary care levels, OPDs and EDs.

Patients with chronic medical disorders are the most frequent attenders in EDs and many do not comprehend that they suffer from a lifelong disorder hence the non-compliance. A few minutes emphasising the importance of pharmacological and non-pharmacological measures and explaining their disorder in a language they understand will hopefully produce the desirable result.

I am grateful to my colleagues (nursing staff, consultants, registrars and medical officers in various disciplines) for their input and assistance at Groote Schuur Hospital Emergency Unit.

A. Aboo

*CHAPTER ONE***THE FOCUSSED GENERAL EXAMINATION**

Avoid terms like JACCOLDS etc. Rather focus your general examination related to the primary presentation for eg. in a patient with central chest pain look for signs of risk and aggravating factors: - tendon xanthomata, signs of metabolic syndrome and diabetes, anaemia etc.

HEAD AND NECK

A.) Truncal obesity, acanthosis and skin tags on the neck suggest the metabolic syndrome.

In many of these patients careful palpation will also reveal not fat pads but bilateral parotid enlargement. The commonly held belief that parotid enlargement in this setting is due to alcohol abuse is not true as it also occurs in teetotalers. The triad of acanthosis, skin tags and parotid enlargement we shall call the “fast food facies”.

Bilateral parotid enlargement does occur in alcoholism but is then in a setting of malnutrition, as in HIV. Acute salivary gland enlargement is sometimes seen in an allergic reaction to radiocontrast material.

Bilateral proptosis in this setting suggests established hypertension.

Proptosis is also seen:

1. Graves Disease (past or present)
2. Advanced Chronic Renal Failure
3. Chronic Obstructive Pulmonary Disease
4. Familial

B.) Pallor

1.)The commonest cause of anaemia in adults is iron deficiency due to gastrointestinal or uterine blood loss (dietary deficiency and postgastrectomy syndromes are rare causes)

Non steroidal use is the leading cause in all age groups (in urban regions).

In elderly patients, after excluding malignant and diverticular disease, consider angiodysplasia (small and large bowel)and hereditary haemorrhagic telangiectasia (observe the lesion on the tongue and lips). Other causes of anaemia will become apparent on the full blood count which is discussed elsewhere.

C.) Jaundice

Look for other signs of liver disease and liver failure.

In elderly patients always search for a palpable gall bladder.

In extrahepatic obstruction the urine will contain bilirubin and NO urobilin.

In hepatic causes both pigments will be present.

In haemolytic jaundice NO Bilirubin will be detected.

A flapping tremor also occurs in renal failure, type 2 respiratory failure and severe heart failure. A unilateral flap is a focal sign usually due to a lacunar infarct.

D.) A pale and slightly oedematous face suggests poorly controlled, long standing diabetes; chronic renal failure or hypothyroidism.

Diabetic facies often have a very smooth skin texture. I suspect it is due to a vasculopathy.

In this setting proteinuria is always present with or without signs of a peripheral neuropathy.

Road map facies (over wrinkling) is seen in the elderly, those overexposed to the sun and wind AND in heavy smokers.

E.) Angular stomatitis suggests oral thrush, ill fitting dentures – especially in the elderly – or Vitamin B deficiency seen in alcoholics from a poor social background. In rural areas extreme poverty without alcohol abuse may also cause vitamin deficiencies.

F.) Pigmented and hairy facies occur after long term phenytoin use or porphyria.

Some men who regularly smoke marijuana (with mandrax) also known as a 'slowboat' also develop pigmented, hairy facies.

G.)Lymph node enlargement

One certain way of reducing the severity and spread of TB is making an early diagnosis. The patients who end up in ED's have been to primary care physicians on more than one occasion without the diagnosis being considered. HIV positive patients are frequently sputum negative and a careful search for lymph nodes which can be aspirated will confirm the diagnosis.

The commonest cause of a swelling in the neck is an enlarged lymph node. There are no lymph nodes in the scalp therefore a swelling in this area is not a lymph node.

The occipital nodes (between the mastoid process and back of the neck) enlarge due to scalp sepsis or viral infections.

Divide the neck into 3 regions :

1. **Lower third** (supraclavicular glands) – these glands enlarge due to disease in the thorax or abdomen.

With patient sitting, head slightly turned to the right and partially flexed palpate the right supraclavicular fossa. Repeat on the left.

The bony hard mass felt deep below the midpoint of the clavicle is the first rib. The scalene node when it enlarges is felt on this rib.

The right scalene node drains the whole right lung and the left lower lobe.

The left scalene node drains the left upper lobe.

Tuberculosis and lung cancer are the causes of scalene node enlargement.

The **Virchow-Troisier (VT)** node is best palpated with the patient supine, head turned to the left, thumb in the suprasternal notch and encircle the left sternocleidomastoid with the middle finger- the gland will be palpable between thumb and finger.

Sometimes the gland is visible between the two heads of the sternomastoid muscle when seated and the neck extended.

This palpable gland is due to malignant disease of stomach, pancreas and oesophagus.

We have also seen many HIV patients with CD4 of less than 200 and abdominal TB lymphadenitis (presentation is that of severe upper abdominal pain and marked tenderness) with a VT node and no other cervical lymph nodes, aspiration of which confirmed the diagnosis of TB. The chest X-ray in these patients is usually normal. Patients with TB lymphadenitis of the chest often have multiple supraclavicular nodes including the chain of nodes close to the internal jugular veins from sternum to the angle of the jaw.

2. Middle third – a node in this region is usually due to a thyroid lesion.

3. Upper third – these lymph nodes occur as a result of disease of the head and upper neck – floor of mouth, tongue, sinuses, nasopharynx, tonsils etc.

The midline submental node occurs most frequently in patients with HIV/AIDS and oral thrush.

Disseminated TB and lymphoma can involve all 3 groups.

THE UPPER LIMBS

1.) Digital clubbing plus central cyanosis occurs in:

- Chronic Obstructive Pulmonary Disease AND either carcinoma or bronchiectasis or extensive post TB bronchiectasis.
- Pulmonary Fibrosis eg. Fibrosing alveolitis (less common).
- Congenital heart disease (rare)

2.) Nicotine stains occur on the thumb, index or middle fingers.

Yellow pigmentation of one palm is suggestive of mandrax and marijuana smoked through a glass bottle neck.

3.) Hyperextending the metacarpophalangeal joints with your hand results in darkening of the palmar creases. Any person who has lost more than 3 -4 units of blood acutely will not display this sign. In chronic anaemia darkening of the creases occurs, no matter the severity.

4.) Many patients with thyrotoxicosis develop some degree of **onycholysis** in one or more finger nails. If there is no evidence of psoriasis or regular immersion in water consider this diagnosis.

Fungal infections of many nails suggests poorly controlled diabetes or HIV/AIDS.

5.) Bilateral flexion deformities of the little fingers only is either congenital or due to poorly controlled, long standing diabetes (the 'prayer' sign). These patients will almost always have bilateral wasting of the small muscles of the hands (peripheral neuropathy) and proteinuria. Note that motor neuron disease is the least common cause of wasting of the small muscles of the hands. Unilateral wasting is either due to ulnar nerve or cervical spine pathology.

6.) On the clenched fist palpate each extensor tendon from the knuckle to the PIP joint for tendon xanthomata.

7.) A bluish purplish pigmentation of the proximal nail beds (most pronounced in the thumbs) is sometimes seen in HIV/AIDS with very low CD4 counts. In some patients the whole nail bed is pigmented. Bluish pigmentation also occurs in Systemic Lupus and Anthracycline therapy.

Melanin pigmented nail beds are more often physiological than due to Addisons disease.

8.) A macular papular eruption on the palms and soles occurs in:

- Erythema multiforme (rash elsewhere with target lesions)
- Tick bite fever (headache, generalised rash)
- secondary syphilis (elsewhere a papular squamous eruption and lesions in the mouth)
- meningococcaemia (more common on lower limbs)

9.) Epitrochlear nodes are best felt (with the elbow partially flexed) above the medial epicondyle in the sulcus between the triceps and biceps muscles.

10.) The Temperature

The lowest reading on a standard thermometer is 35°C – therefore never accept a temperature of 35°C as the actual reading. Request the T' on a low-reading thermometer – you are dealing with hypothermia.

With every 1°C rise in temperature the heart rate accelerates by $\pm 15/\text{min}$.

An increase of less than 10 means a relative bradycardia, the causes of which are:

- Viral infections and typhoid fever
- Lymphoma
- Drug fever (usually on antibiotics for more than 5-7 days)
- Factitious fever- here the pulse rate will be normal.

THE LOWER LIMBS

1.) Oedema in the lower limb that indents with minimal pressure and fills out readily is usually due to hypoalbuminemia.

Heart failure indents with more pressure (and often tender) and takes a little longer to fill out whilst lymphoedema does not or hardly indents.

Cellulitis of lower limbs occur most commonly in diabetes, peripheral vascular disease, chronic oedema, previous scars and often not looked for - tinea pedis, in patients with diabetes, which if left untreated will result in recurrences.

2.) In suspected deep vein thrombosis tenderness over the femoral vein in the inguinal region (medial to femoral pulse) is diagnostic of a femoral vein thrombosis.

Painful swelling of the calf with haemosiderin pigmentation below the maleoli suggests a calf muscle tear and haemorrhage.

A swollen painful calf with a tense cystic swelling in the popliteal fossa is suggestive of a ruptured Baker's cyst.

3.) Medial to the femoral vein in the inguinal region lies a gland which enlarges with lesions of the perineum, anus, vulva and penis. Septic lesions of the buttocks results in enlargement of the lateral inguinal glands.

4.) An inflamed and painful knee may be due to a septic bursitis (patient can flex the knee) or a septic arthritis (cannot flex the knee).

5.) Note that if a patient complains of pain in a leg and you find no signs of inflammation or swelling or arthritis or vascular insufficiency you may be dealing with referred pain (lumbar disc or metastatic disease) - do a neurological examination of the lower limb.

6.) In patients with coronary heart disease palpate the Achilles tendons for xanthomata.

*CHAPTER TWO***ACUTE CHEST PAIN**

Of epidemic proportions in urban areas. Attempt at all times to try to confirm the diagnosis of Coronary Heart Disease (CHD) and not to exclude it.

Do not attribute chest wall tenderness as the cause of chest pain in middle aged and elderly patients as they will always say yes in the hope that you will pay more attention.

Know the risk factors :

- elderly
- diabetes
- hypertension
- hyperlipidaemia
- smoking
- sedentary lifestyle.

A history of regular chest pain at certain exercise levels, lasts for 5-8minutes and hardly ever occurs at rest favours CHD.

Chest pain in a patient with known CHD is due to ischaemia until otherwise proven.

CHD is no longer confined only to the middle and richer classes. Note the increasing incidence in the urban poor due to regular fast food consumption.

In practice you will face the following scenarios:

1. **Chest Pain with ECG changes:** administer aspirin. With ST elevation administer thrombolytic agent if not contraindicated and refer to an ED urgently.
2. **Chest pain with risk factors and a normal ECG:** refer to ED's for observation, repeat ECG and enzymes. Administer aspirin before referral.
3. **Typical chest pain with no risk factors and a normal ECG:** Observe and repeat ECG in a few hours. If still normal and the pain has settled refer to an OPD for an Exercise Stress Test.
4. **A normal ECG during an episode of chest pain:** consider other diagnoses eg. oesophageal spasm.

5. **Atypical chest pain with no risk factors and a normal ECG:** reassure
6. **Chest pain and aortic regurgitation suggests a dissection.**
7. **When in doubt always refer urgently.** Note that sometimes (especially females) pain occurs over the mid or lower scapular regions.

I am often asked when a Trop T should be done and its value in the assessment of chest pain. Clearly it should **NOT** be done if there are **NEW** ECG changes (ST \uparrow , ST \downarrow , and T wave inversion) as it does not add to the diagnosis.

The Trop T is most valuable if done 4 or more hours after the onset of typical chest pain in a patient with a normal ECG or an unchanged abnormal ECG recorded on a previous episode. If the Trop T is negative and the patient has significant risk factors it is worth repeating the Trop T at 12 hours.

Newer methods of making an earlier diagnosis of acute coronary syndrome will no doubt become available over the next few years.

Note that gastroesophageal reflux with spasm will result in central chest pain with exactly the same referral of pain as in Angina. Take a minute to minute account of the episode. You will note the raised BMI, that he/she is able to walk around during the episode, consumes large amounts of food or drink or both before retiring and that the pain occurs more often at night or after meals, that the pain lasts for hours and the ECG will either be normal or display minor T-wave changes.

A chest X-Ray to exclude dissection or other comorbid disorders is advised.

*CHAPTER THREE***ACUTE SHORTNESS OF BREATH**

Acute Severe Asthma and Acute Cardiogenic Pulmonary Oedema, if not diagnosed promptly and attended to urgently will result in rapid deterioration.

1. Acute Severe Asthma

Failure to recognize the signs of severity will result in increased morbidity and mortality.

Signs of severity- what do you

- **see** – a patient struggling to breathe, anxious, sweating and cyanosed, struggling to speak.
- **hear** - very loud expiratory wheeze or hardly any breath sounds. Cannot finish a sentence. Reduced breath sounds in one lung suggests a pneumothorax.
- **feel** – tachycardia, pulsus paradoxus.

Sometimes it may be difficult to distinguish between asthma and an exacerbation of COPD. Polycythemia and a positive Hoover's sign (indrawing of the lower ribs on inspiration) favour COPD.

The following features favour asthma:

1. family history of asthma
2. non-smoker
3. early morning attacks
4. commenced in childhood or early adult life

2. Acute Cardiogenic Pulmonary Oedema

Occurs most commonly in the early hours of the morning.

The patient is rushed in:

- extremely short of breath
- sitting upright
- the chest is noisy
- the pulse is rapid
- the BP usually is high (this does not mean the patient is hypertensive – and if the BP is low the prognosis is poor and may require inotropes)
- the extremities are COLD and often WET.

Massive pulmonary embolism may present with dyspnoea and cold extremities but the BP is usually low, the chest is clear and P2 will be accentuated.

Acute left ventricular dysfunction results in a release of catecholamines and angiotensin which leads to tachycardia, elevation of BP and intense vasoconstriction of the skin and splanchnic circulation with almost 80% of the blood volume in this circulation ejected into the pulmonary circulation.

The poor response to diuretics in the early stage of pulmonary oedema is due to this marked reduction in renal blood flow.

You do not need a chest X-Ray to make the diagnosis.

Administer oxygen, 2 tablets of sublingual nitrate and set up an IV line(no saline solution) and refer urgently. You may administer 2 to 5 mg of IV morphine and a large dose of furosemide.

At hospital continue with the above therapy and if the BP is still high administer a small dose of a crushed short-acting Ace Inhibitor (Captopril 6.25mg) sublingually.

Commence IV nitrates if no improvement occurs.

Commence inotropes (low BP) and consider intubation and ventilatory support.

When the extremities are warmer administer another dose of IV furosemide.

The commonest precipitating factors are acute coronary ischaemia, uncontrolled hypertension, tachy-arrhythmias and IV fluid therapy in hospital to patients with compromised cardiac and/or renal function.

Doctors on commercial flights who are asked to attend to a fellow passenger in pulmonary oedema know that the stethoscope is of no value (noise factor) and that the diagnosis is readily made by feeling the COLD extremities. The administration of oxygen and nitrates (every second passenger carries some form of nitrates) results in rapid recovery.

Note:

Neurogenic Pulmonary Oedema is rare and occurs after a grand mal seizure or a large cerebral haemorrhage, and the diagnosis is only made radiologically because none of the other signs are usually present – specifically the extremities are NOT cold and wet. There is a better response to diuretics than in cardiogenic pulmonary oedema.

3. Chronic Obstructive Pulmonary Disease (COPD)

Acute episodes may be indistinguishable from pulmonary oedema but the extremities are

WARM. Pulmonary oedema may occur in patients with COPD (due to acute coronary ischaemia) in which case the extremities will be COLD.

Patients with COPD also develop episodes of Paroxysmal Nocturnal Dyspnoea (PND) but the mechanism is different.

During sleep secretions that are not expectorated cause ventilation perfusion defects and hypoxia.

The patient wakes up acutely dyspnoeic with coughing which settles only after expectorating sputum.

When these episodes occur in a hospital ward the mis-diagnosis of pulmonary oedema is often made because of the PND and basal crackles. Careful examination will reveal the warm extremities and that the basal crackles are coarse and heard early in inspiration, a normal sign in COPD.

4. Also consider pneumonia, pneumothorax and in HIV/AIDS – PCP or advanced TB.

CHAPTER FOUR

HEART FAILURE

Once you have diagnosed heart failure you need to establish the aetiology and look for complications eg. atrial fibrillation, I.E.

In adults hypertension is the commonest cause of heart failure. If there is no history or evidence of hypertension the following approach to establish the aetiology is useful:

Enquire about alcohol intake in all. Note that in the urban working classes ischaemic heart disease is on the increase (regular fast food intake). Unexplained heart failure with absent ankle reflexes (in the absence of diabetes) is highly suggestive of Alcoholic Cardiomyopathy.

1. Age and Socio-economic status

Under 40 and poorer social classes

- Rheumatic Heart Disease
- Idiopathic Dilated Cardiomyopathy (includes post partum CMO)
- Congenital Heart Disease (rare)

Over 40 and poorer social classes

- Dilated Cardiomyopathy
- Ongoing Rheumatic Heart Disease
- Ischaemic Heart Disease
- Disease of the elderly (see later)

Under 40 and middle class

- Ischaemic Heart Disease
- Congenital Heart Disease (rare)
- Rheumatic Heart Disease (rare)

Over 40 and middle class

- Ischaemic Heart Disease
- Disease of the elderly

Heart Failure in the Elderly

1. Ischaemic Heart Disease
2. Hypertensive Heart Disease
3. Aortic Stenosis (AS)

In the elderly AS differs from younger patients in that the :

- apex beat is not typically sustained
- the pulse is not anacrotic
- the murmur is often high-pitched and is best heard just below the right clavicle and as in the young the second heart sound is soft or inaudible.

Recent onset of hypertension in the elderly

Is usually due to :

1. Chronic Renal Failure
2. Diabetes
3. Renal Artery Stenosis.

In elderly patients with no renal impairment, ischaemic heart disease or cerebrovascular disease treatment should only be initiated after a number of readings. The aim should be cautious reduction as complications of treatment often outweigh the benefits.

Management of known Hypertensive patients at the Primary Care Centres

Non compliance with drug therapy and diet is by far the commonest cause of poorly controlled BP when seen at regular follow-up at the health centres resulting in unnecessary referrals to hospitals.

If the patient does not have malignant hypertension (papilloedema, retinal haemorrhages) or hypertensive encephalopathy (confusion/convulsions/vomiting) or Angina or acute heart failure (pulmonary oedema) administer stat doses of their regular medication, send them to the pharmacy to collect their regular meds and see them a few hours later. If still unstable you may then refer to EDs.

Headache is often wrongfully attributed to hypertension. Only malignant hypertension or a hypertensive bleed causes a headache.

*CHAPTER FIVE***STRIDOR**

Note the associated symptoms to localise the site of disease.

1. Dysphagia – at the epiglottis (inflammation, tumours, foreign body)
2. Hoarseness -at the larynx (tumour, inflammation)
3. Dyspnoea – trachea (inflammation, haematoma, tumour, foreign body)

Angio-oedema due to ACE Inhibitors is not uncommon and frequently presents with stridor and oedema. ACE-Inhibitor induced angio-oedema may occur after many years of use.

Sometimes patients with carcinoma of the mid-oesophagus present with stridor and hoarseness due to recurrent aspiration and laryngeal oedema.

The delay in the diagnosis of this cancer is distressing. Most of the patients have lived or are living in rural areas. Failure to enquire about dysphagia is the primary reason.

Ask the patient to drink a glass of water as rapidly as possible and you will note the pain and discomfort on the face and swallowing only in sips. Coughing after the drink suggests a tracheo -oesophageal fistula. Consider this diagnosis every time you see a middle-aged or elderly patient from a rural area (or who grew up in a rural area) whose presenting symptoms are vomiting and weight loss.

CHAPTER SIX

ABDOMINAL DISORDERS

Gastritis is a diagnosis that should NOT be made in EDs. You will be proven wrong most of the time. Consider PUD, Reflux, Biliary Colic or Pancreatic disorders.

1. Acute Diarrhoea

Assess hydration:

Tachycardia and a postural drop of BP>15 confirms dehydration.

Small bowel diarrhoea:

epigastric and periumbilical pain, large volumes of watery diarrhoea, no blood or mucous – requires fluids only – no antibiotics

Large bowel diarrhoea:

Suprapubic pain, small frequent stools, blood and mucous. Shigella is the commonest.

Require fluids and antibiotics Consider amoebiasis in endemic areas. Do not use antidiarrhoeal agents (unless it interferes with function eg. writing exams, no toilet facilities)

Large bowel diarrhoea is frequently misdiagnosed as a pelvic infection in females because of suprapubic pain.

Patients with diabetes frequently attend EDs with either vomiting and/or diarrhoea and this is often wrongfully attributed to autonomic dysfunction. If the ankle reflexes are present consider the usual causes for diarrhoea/vomiting. Note that vomiting is a common presenting symptom of ketoacidosis.

2. Bleeding

Overt upper and lower GIT bleeding (haematemesis and meleana) require urgent resuscitation and gastroscopy.

Frank blood per rectum should be referred to a surgeon.

If the patient claims to have vomited blood and the vitals are normal and faecal occult blood is negative pass a NG tube to confirm the bleed. Malingerers will decline this procedure.

Occult bleeding with positive faecal occult blood.

In all age groups upper gastrointestinal bleeding is the commonest cause and NSAIDS use is the most frequent offender.

In urban SA a large section of the adult population is addicted to a powder containing caffeine, aspirin and paracetamol and are frequent visitors to ED's with either overt or occult bleeding and dyspeptic symptoms. Most of these patients deny this 'habit' until a family member confirms the addiction.

Diverticulosis and **colon cancer** are common lower bowel causes of bleeding.

In the elderly **angiodysplasia** of small and large bowel should be considered after excluding all of the above.

3. Acute Iliac Fossa Pain

Diverticulitis(also known as left-sided appendicitis) is the most likely cause of Left Iliac Fossa (LIF) pain in the elderly.

Acute appendicitis is the most likely cause in all patients with Right Iliac Fossa (RIF) pain.

Also consider the following:

- Urinary Tract Infections (in females)
- Ureteric Calculus (in males)
- Colonic Malignancies(rare)
- Psoas Abscess (rare)
- Ileocaecal TB and Crohns are rare causes of RIF (consider if risk factors for TB exist)

In females if adnexial tenderness is more pronounced than abdominal tenderness you are probably dealing with a gynaecological problem.

When in doubt observe and obtain a surgical or gynaecological opinion.

4. Renal Colic

More than **90% of renal calculi** occur in **males**. (95% of UTI's occur in females).

Younger males will have calcium stones and older males uric acid stones.

Regular fast food consumption has resulted in an increasing incidence of this disorder in young males in urban SA. Note that 5-10% of patients do not have haematuria.

All patients should receive IV fluids and analgesia and the majority will pass the stone.

Arrange a follow up at urology OPD. Urgent referral to a urologist is required when:

- the patient is known to have a single kidney
- has a fever
- there is ongoing pain
- there is renal impairment

Spiral CT is the investigation of choice. If not available do an ultrasound. An IVP may be done if renal function is not impaired.

5. Acute Testicular Swelling

In young patients consider STD or torsion.

No sexual contact and a clear urine favours torsion. Sexual activity does not exclude torsion.

With the patient standing you will note that with infection the affected testes hangs lower and the reverse is true for torsion.

In older patients with prostatomegaly an acute testicular swelling is more likely to be a UTI rather than an STD.

6. Haematuria

- With clots – emergency referral for bladder washout and cystoscopy etc.
- Frank Haematuria – maybe an infection or tumour or calculus. Refer to urology over the next few days and increase fluid intake. If it is due to a new catheter encourage fluid intake and review. If on anticoagulants correct the INR and continue with investigations.
- Microscopic haematuria – If there is associated hypertension or proteinuria or renal impairment the problem is nephrological and refer to a MOPD or nephrology clinic.

If none of the above arrange an OP IV pyelogram (if creatinine not elevated) or a renal ultrasound and refer to urology OP.

7. Urinary Retention

Chronic urinary retention (usually due to prostatic hypertrophy) is painless and non-tender.

Acute urinary retention is painful and tender.

In obese patients it may be difficult to diagnose retention as the percussion note may not be helpful. Best method is to place your stethoscope just above the pubic symphysis and scratch with your fingernail from the epigastrium downwards. As soon as you reach to bladder height the sound will change and increase in intensity.

Similarly in obese patients with hepatomegaly place your stethoscope firmly in the epigastrium and scratch from the right iliac fossa upwards.

Physicians of yesteryear used the same method to diagnose fractures of the Neck of Femur (NOF). Place the stethoscope on the pubic symphysis and lightly tap each patella in turn.

The sound is much softer on the side of the fracture (also if the patient has a Total Hip Replacement or dislocation).

The diagnosis of Pagets disease of the skull is made by auscultating the forehead for bronchial breath sounds!

CHAPTER SEVEN

HEADACHE

The commonest cause of severe headache in EDs is due to migraine (including cluster headaches).

1.) First onset with fever consider:

a.) extracranial infections eg. Type A Hepatitis, influenza, tickbite fever or any bacteremia eg. urinary tract infection. Acute sinusitis causes pain over the affected sinus with tenderness and a purulent discharge from one or both nostrils.

b.) intracranial infections eg. Meningococcal meningitis – look out for petechial rash on the lower limbs.

Pneumococcal meningitis occurs secondary to otitis media, sinusitis, pneumonia, head injury, alcoholism and post-splenectomy.

NB. After the LP allow patient to lie PRONE for 2 hours to prevent post LP headache.

2.) First onset with no fever consider subarachnoid haemorrhage or a stroke syndrome.

Note that cerebral tumours present with focal signs or convulsions or signs of raised intracranial pressure rather than headache as the presenting symptom.

If the patient is HIV positive consider Cryptococcal or TB meningitis (toxoplasmosis presents with convulsions or focal signs rather than headache).

RECURRENT HEADACHES

The commonest cause of headache in EDs:

Migraine – often unilateral, retro-orbital or suboccipital with photophobia, nausea, vomiting or phonophobia.

Cluster headaches are very severe, predominantly in males, smokers, occur usually at night, associated with lacrimation, rhinorrhea, miosis or ptosis.

They last for up to 2 hours in clusters for weeks.

Certain drugs also cause headaches:

Nitrate, calcium antagonists, “the pill”.

Tension headaches occur over the vertex with no associated symptoms or signs. The patient is often anxious or depressed.

*CHAPTER EIGHT***THE DIZZY PATIENT (AND STROKE)**

Establish if there is true vertigo -

1. sensation of walls, floor or ceiling movement.
2. Unsteady gait with or without falls
3. associated nystagmus

Many patients use the term 'dizzy' to imply nausea or that they are 'sick'. Anaemia, hypotension, a side effect of many drugs and psychogenic causes should be considered.

Vertigo is either peripheral or central .

Peripheral

The commonest causes are:

- 1.) Benign positional vertigo (related to movement of the head). The Hall – Pyke manouvre will confirm the diagnosis.
- 2.) Vestibular neuronitis – explosive in onset, sweating, vomiting and nystagmus. No deafness or tinnitus. No cerebellar signs. May last for weeks. Refer to ENT or confine to bed for a few days with anti-vertigo preparations.

Central

Vertebro-basilar disease in the elderly and those with risk factors for stroke.

You may hear a bruit in the supraclavicular fossa.

Measure the BP in both arms. A difference of more than 15mmHg in either arm suggests aortic arch atherosclerosis and hence possible subclavian and vertebral artery involvement.

Takayashu Arteritis and Aortic Dissection are other causes of this difference in BP.

With Vertebro- Basilar disease the patient will be :

Dizzy plus one or more of the following -

Diplopia

Dysarthria

Dysphagia

Dysmetria (cerebellar signs)

Double homogenous hemianopia (blindness)

Drop attacks

Demiplegia (hemiplegia)

Demianaesthesia (hemi)

Migraine, cerebello-pontine angle tumours (far less common) and multiple sclerosis (rare) are other central causes of vertigo.

Cerebellar Disease presents with any or all of the following group of signs/symptoms:

1. General – Headache (acute insult), dizziness, scanning speech, nystagmus and a rare but diagnostic symptom of acute cerebellar disease (stroke or head injury) – vomiting on sitting up from the supine position.
2. Limb ataxia (finger- nose, heel-shin tests)
3. Truncal ataxia (sitting or standing)
4. Gait ataxia with or without falls.

STROKE

Note the following conditions that should always be considered in the differential diagnosis of stroke:

1. Hypoglycaemia
2. Todds paralysis
3. Subdural Haemorrhage
4. Cerebral metastases (lung cancer). Note that in 10% of patients with lung cancer the first presentation is neurological (focal signs, convulsions).
5. In HIV patients intracranial infections may present with acute focal signs.

Hysterical paralysis should only be diagnosed by more experienced clinicians or those that have adequate malpractice cover.

Hypertension and Cerebral Atherosclerosis are the commonest causes of stroke.

Less common causes of stroke are due to :

Haematological (polycythaemia, thrombophilia)

Embolic

Migraine

Inflammatory – arteritis (Syphilis, TB, Lupus etc)

A common error made in EDs is to diagnose a mild stroke when in fact you are dealing with

a stroke in evolution. A stroke is only completed if the deficit is static for at least 36-48 hours.

If a patient with stroke is seen within an hour or two of onset consider thrombolytic therapy if facilities are available.

A Carotid bruit with a diastolic component is a certain diagnosis of significant internal carotid stenosis. (The same applies to renal artery bruits).

*CHAPTER NINE:***BLACKOUTS, STRANGE BEHAVIOUR AND RELATED DISORDERS****BLACKOUTS**

The diagnosis is either a seizure or syncope (fit or faint).

Interviewing a witness is crucial.

Factors favouring a SEIZURE:

1. A past or current history of epilepsy, head trauma or surgery, stroke, substance abuse, diabetes, HIV/AIDS, carcinoma of lung or breast.
2. During or immediately after the episode -
 - Tonic, clonic movements while supine (note that patients with syncope, if kept in a sitting position, will also develop tonic/clonic movements)
 - Urinary incontinence
 - Bleeding per os due to laceration of the lateral border of the tongue (lacerations of the tip of the tongue are seen in hysterical seizures).
 - Headache and confusion after the episode.
3. Examination may reveal
 - old depressed fracture of the skull
 - old burn scars
 - leonine facies (repeat injuries to forehead)
 - hairy facies and gum hypertrophy (phenytoin)
 - signs of liver or renal disease or HIV/AIDS
 - focal signs or signs of raised intracranial pressure
4. Investigations
 - Exclude hypoglycaemia
 - If still in doubt - a Creatinine Kinase that is markedly elevated immediately after the episode confirms a seizure.
 - An EEG immediately after the episode may be of value.
 - Hysterical seizures present with blepharospasm and flickering eyelashes (in true coma you can easily open the eyelids with slow closure to the shut position), no

spasm of conjugate gaze and downgoing plantars.

- A hysterical seizure may be difficult to distinguish from temporal lobe epilepsy -and in some cases only an EEG may assist with the diagnosis.

Status Epilepsy

Treated with Benzodiazepines for immediate control and intravenous Phenytoin for continued control.

Any patient who convulses in the ED after having convulsed at home is in status.

If there is NO past history of seizures the cause must be established on the same visit.

A history of epilepsy suggests non-compliance and exhaustive investigations are unnecessary.

Alcohol withdrawal

Seizures occur within 60 hours of withdrawal or reduction in the intake of alcohol – a decision usually made on a Sunday hence the occurrence of these seizures usually on a Monday or Tuesday.

Factors favouring Syncope:

1. A past or current history of myocardial ischaemia, diabetes, permanent pacemaker, aortic stenosis, anti-hypertensive therapy or previous episodes in the erect or sitting position. When syncope occurs in the supine position consider arrhythmias as the cause.
2. Sweating, pallor, dizziness and/or vomiting and an attempt by the patient to prevent the fall favours syncope. If the syncope is due to an arrhythmia the above prodrome will not occur. Bradycardia during the episode favours syncope.
3. Examination for pallor (GIT bleed), arrhythmias, BP (supine and erect), a CNS and cardiac examination should be done.

An ECG should be done in ALL patients.

There are TWO causes of Syncope:

A.) Cardiac – minority in young patients, almost a third in the elderly.

i.) Myocardial infarction

ii.) Arrhythmias (tachy and brady)

iii.) Obstruction of the circulation through the cardiac chambers eg. Pulmonary Embolism,

Aortic Stenosis.

B.) Non- Cardiac

i) The commonest cause is VASOVAGAL SYNCOPE.

The symptoms discussed in 2. are typical.

Always occurs in the erect or sitting position.

Extreme bradycardia in this situation is often mistaken for a cardiac arrest. I suspect that some successful extrahospital resuscitations are actually episodes of vasovagal syncope.

Elevating the legs after placing the patient in a supine position will result in rapid recovery.

Advise these patients to avoid attacks by lying down or placing the head between the knees or if not able to sit (eg. in a crowded lift) to cross the legs and contract every muscle in the lower limbs. Refer to a cardiac clinic if attacks occur frequently.

ii.) Less common causes

- Micturition syncope (in males for obvious reasons)
- Cough Syncope (COPD)
- Post hypotension (drop of 15mmHg or more) due to:
 - a.) volume depletion.
 - b.) Anti hypertensive tricyclic or phenothiazine therapy.
 - c.) Autonomic neuropathy

Note that patients with diabetes experience frequent syncopal episodes but NEVER are they due to Hypoglycaemia (which causes coma, convulsions or confusion).

Causes of Syncope in diabetes are due to:

1. volume depletion (osmotic diuresis)
2. anti hypertensive therapy
3. arrhythmias
4. Ischaemic heart disease
5. Upper GIT bleeds (aspirin use).
6. Autonomic neuropathy (ankle reflexes will always be absent).

STRANGE BEHAVIOUR

Nurses are the first staff members to note observations and presenting complaints in ED's. Confusion, psychosis and hysterical behaviour they label as strange behaviour and I have decided to adhere to their description for want of a better term.

These patients are brought to hospital by friends or relatives, by the police or paramedics with or without relatives. Obtain, if possible, a detailed medical and psychiatric history (including epilepsy, drugs, substances, recent injuries, travels etc.) If possible assess vitals and blood sugar. If the patient is a diabetic and violent, restrain physically and administer IV glucose after obtaining a blood sample.

If the patient is a known epileptic, obtain drug levels and administer a benzodiazepine IV.

If the patient has an old depressed fracture of the skull you are probably dealing with a postictal state.

If the patient is violent and abusive do not get too close or touch the patient (if not restrained) as he or she may consider it a threat.

Delirium vs Psychosis

Your next step is to distinguish organic from functional causes.

The presence of any or all of the following 3 suggests a delirium:

1. Disorientation for time, place and person
2. Decline or fluctuation in the level of consciousness.
3. Fever, tachycardia, sweating, tremulousness, hypertension, hyperreflexia with excessive motor activity (trying to get off hospital trolleys, removing IV lines etc) and signs of HIV/AIDS.

Clearly a patient with a known psychiatric disorder with any or all of the above 3 symptoms and signs should be considered to be delirious.

Causes of Delirium: (as in coma)

1. Intracranial

a.) without focal signs – postictal state, meningitis, encephalitis, subarachnoid haemorrhage.

b.) with focal signs

- postictal state secondary to mass lesions (inflammatory, malignant) or Todd's paralysis

- chronic subdural
- cerebral haemorrhage.

2. Extracranial

- a.) organ failure** (liver, renal, respiratory disorders)
- b.) infections** (especially in the elderly)
- c.) metabolic and endocrine disorders** (eg. Hypoglycaemia, hyponatremia, thyroid disease.
- d.) exogenous** – drug side effect, substance abuse, withdrawal or overdose.

The commonest causes of confusion in young people who are HIV negative are substance abuse, the postictal state and intracranial infections.

In the elderly extracranial infections, electrolyte and metabolic disorders are more common than intracranial disorders.

Delirium with focal signs warrants a CT scan.

Delirium Tremens is a serious disorder with a high mortality if not admitted and treated appropriately. IV dextrose solutions to maintain hydration, large doses of IV Thiamine (300 to 400 mg), large doses of benzodiazepines to induce sleep for at least 48 hours, and prevention of aspiration are key elements of therapy.

Withdrawal seizures are treated with benzodiazepines and not standard anticonvulsants.

Receptive Aphasia

Often misdiagnosed as delirium. Usually an elderly patient with risk factors for cerebrovascular disease (diabetes, hypertension, atrial fibrillation). Answers are inappropriate. Comprehension is poor. No excess motor activity and does not require restraining. Mild facial, right arm drift and ® Babinsky may be present in right-handed patients. You will know the patient's handedness by noting that the dominant hand has a wider thumb nail.

CHAPTER TEN

MUSCULO-SKELETAL DISORDERS

1. **Acute Low Backache(LBA)**

Is it mechanical or not (inflammatory, malignant)?

Mechanical:

weight-bearing aggravates LBA (worse on sitting, standing, walking)

Non-mechanical:

Present both on weight-bearing and on lying down. Retroperitoneal malignancies (pancreas, sarcoma) frequently present with backache which is always worse when lying down. The patient sits up all night and no position relieves the pain.

Inflammatory condition causes marked stiffness, especially in the morning.

LBA of short duration in the elderly is often serious. Note that urinary tract infections do not cause LBA!

An opiate injection is superior to a NSAID (except in inflammatory causes).

There are 3 causes of Acute LBA:

a.) Serious causes

More often in middle age to elderly.

Consider:

- osteoporosis with fractures (steroid dependent, elderly)
- Metastasis (lung, breast, prostate)
- Retroperitoneal malignancies eg. Pancreas or bleeds (Warfarin therapy).
- Dissecting aortic aneurysm (Hypertension)
- Inflammatory - Tuberculosis and rheumatological (marked stiffness often relieved by activity).

Careful examination (abdomen, spine and lower limbs) and X-rays will assist in the diagnosis in almost all patients.

Refer appropriately.

b.) Backache with root signs

Most often due to disc prolapse (young to middle age) or spinal metastases(middle age to elderly).

The Straight Leg Raising test is often positive (note that SLR may be negative in high lumbar disc prolapse – hip extension may be painful in this setting - and diabetic sciatica)

Know your dermatomes:

- Knee (L3)
- Inside leg (L4)
- Outside leg, dorsum of foot and big toe (L5)
- Little toe and sole of foot (S1)

Reflexes:

- knee L4 – note that an absent knee jerk means a L4 root lesion or a femoral neuropathy. If the adductor reflex is present it is a femoral neuropathy eg. Diabetes.
- ankle S1

Note: There is no reflex to test L5 – assess the power of big toe extension.

Best method to elicit ankle reflexes in a patient in severe pain is to ask the subject to lie on the side he or she finds most comfortable with hip and knees flexed. The reflexes are then easily elicited.

Some patients present with pain in a lower limb with no backache. If you find no local cause (arthritis, sepsis, vascular insufficiency, oedema) consider a spinal cause and examine the peripheral nervous system as mentioned.

Patients with root signs require an MRI a.s.a.p.

c.) None of the above.

Usually not serious.

May well be a partial disc herniation with no root signs or degenerative disease.

Do an ESR, X-rays and refer to an orthopaedic OPD if the pain persists.

2. Shoulder Pain

a.) Shoulder disorders

The most common of which are rotator cuff syndrome and frozen shoulder.

Injuries will be self evident.

Haemarthrosis will occur only with bleeding disorders or injuries.

In rotator cuff syndromes the pain occurs on active abduction between 30' to 120', passive abduction is painless, resisted abduction is painful from 0' and the pain is worse when sleeping on the affected side.

Subacromial steroid/ local anaesthetic injection gives immediate relief in almost all patients.

With a frozen shoulder there is pain and limitation of all movements. Refer to a

physiotherapist.

b.) Referred pain

I.) From cervical spine disorders (usually degenerative).

The pain is often worse over the trapezius and radiates to the shoulder (C5) or thumb (C6) or index and middle finger (C7) or ring and little finger (C8).

Shoulder movements are painless.

Extension of the neck and/or rotation induces pain in the neck.

Pressure on the head with the neck extended induces pain in degenerative cervical spine disorders.

Rarely, pain is felt only in the supraspinatous region, especially at night, and may be the first symptom of a Pancoast tumour. Finger clubbing, a palpable scalene node or a Horner's syndrome may be present.

II.) Pain may be referred from:

- the heart (angina)
- oesophagus (reflux with spasm)
- diaphragmatic irritation.

Note: shoulder movements are painless

3. Acute Arthritis

Acute gout and septic arthritis are the commonest causes

In males, and especially if they suffer from hypertension, gout is the most likely cause.

Joints affected are the wrist, knee, big toe and ankle.

Females develop gout in the postmenopausal state or when on diuretics.

In septic arthritis there are systemic signs of infection (high fever, sweating, chills) not seen in gout.

A common error is that of diagnosing a septic prepatellar bursa as an arthritis – in septic arthritis there is marked limitation and pain on movement of the joint.

Rheumatoid and reactive arthritis is more often polyarticular.

4. Patients who ache all over

This is a not uncommon presentation at the primary care level and sometimes on the

patient's insistence referred higher up the chain.

A good history and physical examination will often reveal the aetiology. Conditions to consider:

- an occult infection eg. Infective Endocarditis. Always enquire about recent travels. The ESR/CRP will be raised.
- an occult malignancy eg. pancreas, prostate, renal and haematological malignancies (myeloma).
- Hypothyroidism
- Chronic Renal Failure (CRF) – remember that any patient with anaemia and hypertension suffers from CRF until disproven.
- Polymyalgia Rheumatica (rare disorder, occurs in the elderly, marked stiffness of joints, high ESR) and early rheumatoid arthritis.
- Parkinsonism (rigidity without tremors). You will uncover cogwheeling by flexing and extending the wrist while the patient is moving the head from side to side.

Having excluded the above the most likely diagnosis is Depression.

I have seen many patients diagnosed as “fibromyalgia” who turn out to suffer from one of the above conditions. Diagnosis and treatment of “fibromyalgia” should be left to acupuncturists and those with adequate malpractice cover.

CHAPTER ELEVEN

LOSS OF WEIGHT

These patients should ideally be seen in a medical OPD but frequently end up in EDs where doctors can initiate investigations and procedures before referral. A thorough history and examination (especially looking for pallor, jaundice, lymphadenopathy, abdominal palpation and rectal examination) will suggest a diagnosis.

The usual suspects are:

1. Tuberculosis (anaemia is always present) – Note that respiratory examination may be normal in patients with PTB.
2. HIV/AIDS
3. New onset or poorly-controlled Diabetes (often due to an infection especially pulmonary TB)
4. Thyrotoxicosis (no anaemia)
5. COPD (no anaemia)
6. Various malignancies (anaemia often present)

Less common causes are:

1. Inflammatory bowel disease
2. Chronic Rheumatic disorders
3. Parkinsonism with tremors

There are **3 abdominal malignancies** that are often overlooked or difficult to diagnose:

1.) Gastric cancer without dyspeptic symptoms.

Anorexia is a predominant symptom.

Look for anaemia and a VT node.

Gastroscopy **asap**

2.) Carcinoma of the tail of Pancreas

The predominant symptom is mid dorsal or upper lumbar back pain radiating to the left flank and is especially worse at night. These patients are often misdiagnosed as left sided pyelonephritis. The white cell count may be elevated. CT Abdomen is the investigation of choice.

3.) Abdominal Lymphoma with involvement of bowel and or lymph nodes. CT Abdomen, haematological investigations and referral to gastroenterology department for a definitive diagnosis.

Substance abuse and anorexia nervosa are not uncommon causes of weight loss and should be considered after excluding the above.

*CHAPTER TWELVE***FACTORS CAUSING ACUTE AND SUBACUTE DETERIORATION
OF CHRONIC MEDICAL DISORDERS**

Failure to recognise the factors that precipitate an acute deterioration and attributing it to non-compliance is the reason why there is often a poor response to therapy.

1. Diabetes

- Infections (urinary, respiratory, skin and subcutaneous tissue)
Pulmonary TB is more common in poorly controlled diabetes than the general population (excluding HIV). It is worth remembering that patients with diabetes on treatment with unexplained weight loss should have a chest X-Ray to exclude TB; and patients with TB on treatment with weight loss should have a blood sugar done to exclude diabetes.
- Myocardial Infarction
- Pregnancy
- Inappropriate change in therapy by health practitioners eg. discontinuing insulin for oral therapy
- Non-compliance (diet and/or drugs) for various reasons if none of the above is found. Note that most South Africans believe that only carbonated soft drinks are disallowed and help themselves to liberal amounts of fruit juices.

2. Heart Failure

- Cardiac causes
 - Acute Ischaemia
 - Arrhythmias
 - Infections – infective endocarditis, rheumatic carditis
 - Uncontrolled Hypertension
 - Acute valvular dysfunction
- Non-cardiac causes
 - Anaemia, pregnancy or thyrotoxicosis
 - Pulmonary embolism or infections
 - Drugs (NSAIDS, recent Beta Blocker initiation, verapamil)
 - Not adhering to a low salt diet and fluid restrictions, inadequate or no therapy.

3. **Chronic Obstructive Pulmonary Disease**

- Respiratory Infections in 95% or more
- Pneumothorax (reduced or absent breath sounds in one lung field)
- Ischaemic left ventricular dysfunction with heart failure(extremities will be cold)
- Pulmonary embolism
- Environmental pollution

4. **Chronic Renal Failure (CRF)**

- Dehydration (failure to concentrate urine is the commonest renal functional abnormality), hence diarrhoea in CRF invariably results in deterioration.
- Urinary tract infections (more common in CRF)
- Uncontrolled hypertension.
- Nephrotoxic drugs and NSAIDs use. NSAIDs in patients on ACE inhibitors are absolutely contraindicated.
- If the CRF is due to a systemic disorder, consider a relapse eg. Systemic Lupus

*CHAPTER THIRTEEN***MANAGING BLEEDING/PROLONGED INR IN PATIENTS
ON WARFARIN**

Patients who bleed from the gut or the urinary system should be investigated for an underlying cause after correcting the INR.

Patients are referred to ED's for three reasons:

A.) Prolonged INR

The lab informs the patient to report to the ED asap. The patient is not bleeding.

1. INR - 4-6 : omit Warfarin for 24hours, and recommence the next evening.

2. INR – 6-10: omit warfarin for 48hours and repeat INR and treat as in 1. Advise the patient to stay at home for 48hours. If the patient does not have a valve replacement you may give 1mg Vit K orally (Vit K from an ampoule is well absorbed orally and is as effective as the IV route).

3. INR > 10 : administer 1mg Vit K orally. Omit Warfarin and admit overnight. Repeat INR after 48hours and treat as in **2**. If the patient is not keen on admission AND is not using public transport and has adequate family support allow to rest at home for 48 hours and repeat the INR and treat as in **2**.

B.) Overt bleeding and a prolonged INR

1. Correct blood loss
2. Administer FFP.
3. Administer 1mg Vit K as in **A.**)
4. Admit and investigate asap.
5. Omit Warfarin

C.) Covert bleeding

Patients present with headache, backache, abdominal pain, swelling and pain in a limb or a joint swelling.

If the INR is elevated admit and treat as in **B.**)

Joint swelling may be due to a haemarthrosis or gout. Acute gout in patients on Warfarin should be treated with a short course of Prednisone (20-30g/d x 5days) and simple analgesics.

EM staff should always do an urgent INR in patients on Warfarin under the following circumstances:

1. overt or covert bleeding occurs.
2. If the primary condition for which the Warfarin was indicated deteriorates eg, Atrial Fibrillation and an embolic event, pulmonary embolism symptoms deteriorate.
3. If the patient has not had an INR done for > 1 month.
4. If you plan a surgical procedure.

Advise the patient against the use of NSAIDS and cough syrups (the most common offenders).

*CHAPTER FOURTEEN***POISONING AND OVERDOSE**

Document vital signs and the GCS in all patients.

Parasuicide in the elderly, those who live alone, substance abusers, recent bereavements or those who have suffered a financial disaster and those with major psychoses must be referred to a psychiatrist as soon as they are physically cleared.

The rest should be referred before discharge to a social worker or psychiatrist. If this facility does not exist release the patient into the custody of a family member and arrange an early referral.

Inquire about:

1. what was ingested (fairly reliable)
2. how much was taken (unreliable)
3. when was it taken (fairly reliable)

All patients, **except** those who ingested:

1. Petroleum products
2. Acids (battery)
3. Caustics (detergents-drain cleaners)

receive 100g of activated charcoal a.s.a.p.

Gastric lavage, never for the 3 mentioned above, is only done if you suspect a toxic dose and seen within the hour.

If the patient is drowsy or comatosed – protect the airway, pass a nasogastric tube, aspirate the contents (repeatedly if necessary – with fluids) and administer 100g of charcoal.

Repeat 50g of charcoal, 4 hourly for 24 hours if the drugs ingested delay gastric emptying (anticholinergics, opiates) or are slow release compounds. Toxicology screens are expensive and should not be requested.

- a.) All patients should have a paracetamol level done at 4hours or later unless you are absolutely certain that it was not ingested. If you see the patient more than 12 hours after the paracetamol overdose and you suspect more than 7.5 grams was taken commence the antidote immediately. Liver damage will be detected between 48 to 72 hours of the

overdose (elevated ALT or prolonged INR). Continue with the antidote if liver damage has occurred.

b.) A toxic dose of tricyclic antidepressant will result in any of the following within 6 hours of the overdose:

- i. coma or convulsions
- ii. respiratory depression with desaturation (the most serious effect)
- iii. tachycardia, hypotension
- iv. QRS width of more than 100 msec. in a limb lead.

Administer Sodium Bicarbonate (the antidote) to raise urinary pH to 7 (Measuring blood pH is wasteful).

The rest of the treatment is supportive.

c.) Refer for charcoal haemoperfusion if the patient has taken a massive dose of Barbiturates (GCS ↓ requiring ventilation)

Theophylline (levels more than 400)

d.) Petroleum product ingestion may result in a lipid pneumonia - the respiratory rate will be increased. Do not X-ray if the rate is normal.

e.) Organophosphate poisoning:

- not a toxic dose if the pupils are not constricted
- the heart rate not slow
- no fasciculation occurs on contracting the facial muscles.

Large doses of Atropine are required if a toxic dose has been ingested.

Observe all organophosphate poisoned patients for at least 24 hours. The antidote is obidoxime (administered within 4 hours).

f.) Caustic and acid ingestions must be referred for endoscopy. No charcoal please.

g.) INH overdose causes grand mal convulsions within an hour of ingestion. Administer pyridoxine and benzodiazepines. Theophylline and tricyclic overdoses also cause convulsions.

h.) Iron overdose

Half a tablet of Ferrous Sulphate per kg is a toxic dose and 1 tablet (40mg of elemental iron) is a potentially fatal dose.

There are 4 phases of iron overdose:

1st phase – within 6 hours of a toxic dose vomiting, severe abdominal pain, shock will occur.

2nd phase – 6-24 hours – asymptomatic.

3rd phase – 24-72 hours – if a toxic dose was ingested and no antidote given – acute renal failure and/or hepatic necrosis may occur.

4th phase – jejunal strictures which will manifest months or years later as intestinal obstruction.

Treatment:

Abdominal X-ray will confirm radio opaque tablets and if symptomatic do a gastric lavage and instill bicarbonate (50ml of 8.4%)

Collect a urine sample in a tube and tape to the IV tubing – empty the bladder.

Administer 1-2 g of IM desferrioxamine, IV dextrose and collect urine hourly for 4 hours – compare to the initial sample - a vin rose colour is the indication for IV desferrioxamine in a high care unit.

- i.) Bites – envenomation with snake bites will occur within 6 hours in most cases.
Spider bites envenomation may be delayed for up to 24 hours.

CHAPTER FIFTEEN

INTERPRETATION OF SOME COMMON INVESTIGATIONS in EDs

The Chest X-Ray

1.) In HIV/AIDS patients cavitary TB is rare with low CD4 counts.

Hilar and paratracheal lymphadenopathy occurs only with low CD4 counts.

2.) Cardiomegaly in patients who are HIV positive is due to

- pericardial effusion (moderate to massive) due to TB.
- anaemia of many months duration (mild to moderate)
- unrelated cardiac conditions. Some clinicians believe that dilated cardiomyopathy is more common in HIV positive individuals.

3.) A very small cardiac silhouette is due to :

- Hyperinflation
- HIV/AIDS (usually with signs of marked weight loss)
- Addisons Disease
- Anorexia Nervosa

4.) Multiple healed rib fractures with no past history of injury is suggestive of alcoholism.

An infiltrate in this setting is usually due to TB.

5.) In Heart Failure right-sided or bilateral pleural effusions are commonly seen. Effusions that occur only on the left are usually due to a second pathology. Note that in cor pulmonale effusions are always due to a second pathology.

6.) Pericardial Effusions in the elderly are usually due to malignant disease of the lung or breast. In elderly females consider myxoedema if there is no evidence of malignant disease.

The Full Blood Count

A low Hb

Low MCV means iron deficiency means blood loss (GIT or uterine). Dietary deficiency and post-gastrectomy syndrome are rare causes. Thalassaemia occurs in people of Mediterranean or Asian descent and most importantly the red cell count is elevated.

A high MCV

Megaloblastic anaemia, chronic liver disease, alcohol abuse, hypothyroidism, haemolytic anaemia and rarely due to drugs.

A normal MCV

Chronic Renal Failure and Chronic Sepsis will account for the majority. If none of the above and the RDW is elevated consider a mixed deficiency. If the RDW is normal request a reticulocyte count – a low count suggests a bone marrow disorder - a high count haemolytic anaemia and if normal continue the search for an occult infection or malignancy or a chronic rheumatic disorder.

Neutrophil Leucocytosis

Infections account for the majority. If there is no obvious infection always consider the following:

1.) **Tissue Necrosis:**

- of muscle (heart and skeletal) eg. after an MI, grand mal convulsions, prolonged coma.
- of bowel and limbs (gangrene)
- of tumours (lung, gut, liver, pancreas etc.)

2.) **Acute bleed** (joints, muscles, brain, gut) and **acute haemolysis**.3.) **Acute Pulmonary Oedema, Acute Asthma, Diabetic Ketoacidosis and exogenous glucocorticoids.**4.) **Haematological malignancies.****Elevated LDH**

If the ALT is elevated the source is the liver.

If the CK is elevated the source is muscle.

If the patient is anaemic you are probably dealing with a haematological disorder (leukaemia, lymphoma, megaloblastic or haemolytic anaemia).

If the patient is dyspnoeic consider Pulmonary Embolism or PCP.

Note that jaundice in a patient with heart failure is usually due to an Ischaemic Hepatitis due to a recent hypotensive episode. The clue is the disproportionate elevation of the LDH to other liver enzymes.

HYPONATREMIA occurs in:

1. **dehydration** – fluid replacement (urine Na will be less than 20mEq)

2. overhydration: heart failure, nephrotic syndrome, renal failure (fluid restriction)

3. euhydration – SIADH, diuretic usage or Addisons (urine Na will be $>20\text{mEq}$)

- in SIADH the urea will be low

- in Addisons the urea will be high

- with diuretic use the serum potassium is usually low.

Dehydrated patients with Hyponatremia are treated as follows: $(140 - \text{Na}) \times 2 \text{ ECF vol}$
 $(2 \times 15\text{L}) = \text{deficit of Na in mEq.}$

Administer this amount as normal saline in 24 hours. (eg. deficit of 600 mEq of Na = 4L of normal saline). The first few litres are given rapidly until no postural drop in BP occurs.

In SIADH the treatment of choice is fluid restriction. If the patient is comatosed or convulsing or vomiting administer Hypertonic Saline. Do not raise the Na by more than 10mEq per day eg. $10 \times 2\text{ECF (30)} = 300\text{mEq of Na}$. Administer this amount over 24 hours as Hypertonic Saline under close supervision. (3% Hypertonic Saline contains 510 mEq of Na/ L and 5% contains 850 mEq of Na/L – 1g Saline = 17mEqNa).

Pre renal failure - the urea is far more elevated than the creatinine (dehydration, heart failure)

Intrinsic renal failure – there is a proportionate increase of urea and creatinine.

In crush injuries (usually after assaults) with renal failure the creatinine is far more elevated than the urea.