UPPSALA UNIVERSITY
Department of Women's and Children's Health
Section for International Maternal and Child Health (IMCH)

Staircase Diagnostics

especially in relation to low income countries

Gunnar Holmgren, Section for International Maternal and Child Health, Uppsala University, Sweden

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Postal address
University Hospital
Entrance 11
S-751 85 UPPSALA, Sweden

Visiting address
University Hospital, Entrance 11

E-mail: IMCH.sekretariat@kbh.uu.se
http://www.kbh.uu.se/imch

Phone
+46 18 66 59 96

From June 2000
+46 18 661 59 96

Fax
+46 18 50 80 13

VAT
SE20210029320

Staircase diagnostics

Gunnar Holmgren, Section for International Maternal and Child Health, Uppsala University, Sweden. October, 2001

All sound therapy in medicine is built on a reliable diagnosis. However the pathway to reach this diagnosis may vary widely depending on the resources available. A good patient history and careful clinical examination are paramount, and the past experience of the clinician plays a decisive role. Osler[1] the father-figure in clinical medicine in the early part of this century emphasized that most of medicine is learnt from patients at the bedside. "To study the phenomenon of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all".

It is likely that within the scope of internal medicine 80-85% of the diagnosis comes from the history, a further 7-15% from the clinical examination and around 8% per cent from technical investigations[2].

According to Sandler[3] 95-99% of investigations showed no unexpected results and two thirds of diagnoses could have been arrived at with a more careful history. Borgenhammar[4] has estimated that in Sweden investigations swallow 30% of the total costs for a hospital and in only 2% of patients is the treatment so radically changed because of the investigation result that the patient's health is significantly improved. And yet investigation of ill patients is a huge drain on the resources of all countries.

Sackett[5] suggests that clinicians assign diagnoses in one of four ways:

1. Pattern recognition (Gestalt). This is mainly of value in uncommon conditions where certain features are rare in other conditions and so give a clue to this specific disease.
2. Arborization (algorithm). Diagnosis can proceed down but one of a large number of preset paths by a method in which the response to each diagnostic inquiry automatically determines the next inquiry to be carried out and ultimately the correct diagnosis or course of action.
3. The complete "history and physical" (exhaustion). This is how students learn clinical medicine but this exhaustive approach is prohibitively inefficient if used throughout professional life.
4. The hypothetico-deductive method. Here the clinician formulates from the earliest clues about the patient a "short list" of potential diagnoses or courses of action followed by the performance of those clinical (history and examination) and paraclinical (laboratory) manoevres that will best reduce the length of the list.

The limits of diagnostic investigation vary widely from high to low-income-countries. Sometimes there are acceptable cheaper ways of coming to a conclusion. These are especially useful at the peripheral level and when resources are absent.

In the rest of this compendium some suggested acceptable alternatives are given, complementing and in situations of severe financial constraint replacing the standard investigative pathways.

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3. Sandler G. Costs of unnecssary tests. BMJ 1979 7 July. 2: 21-24
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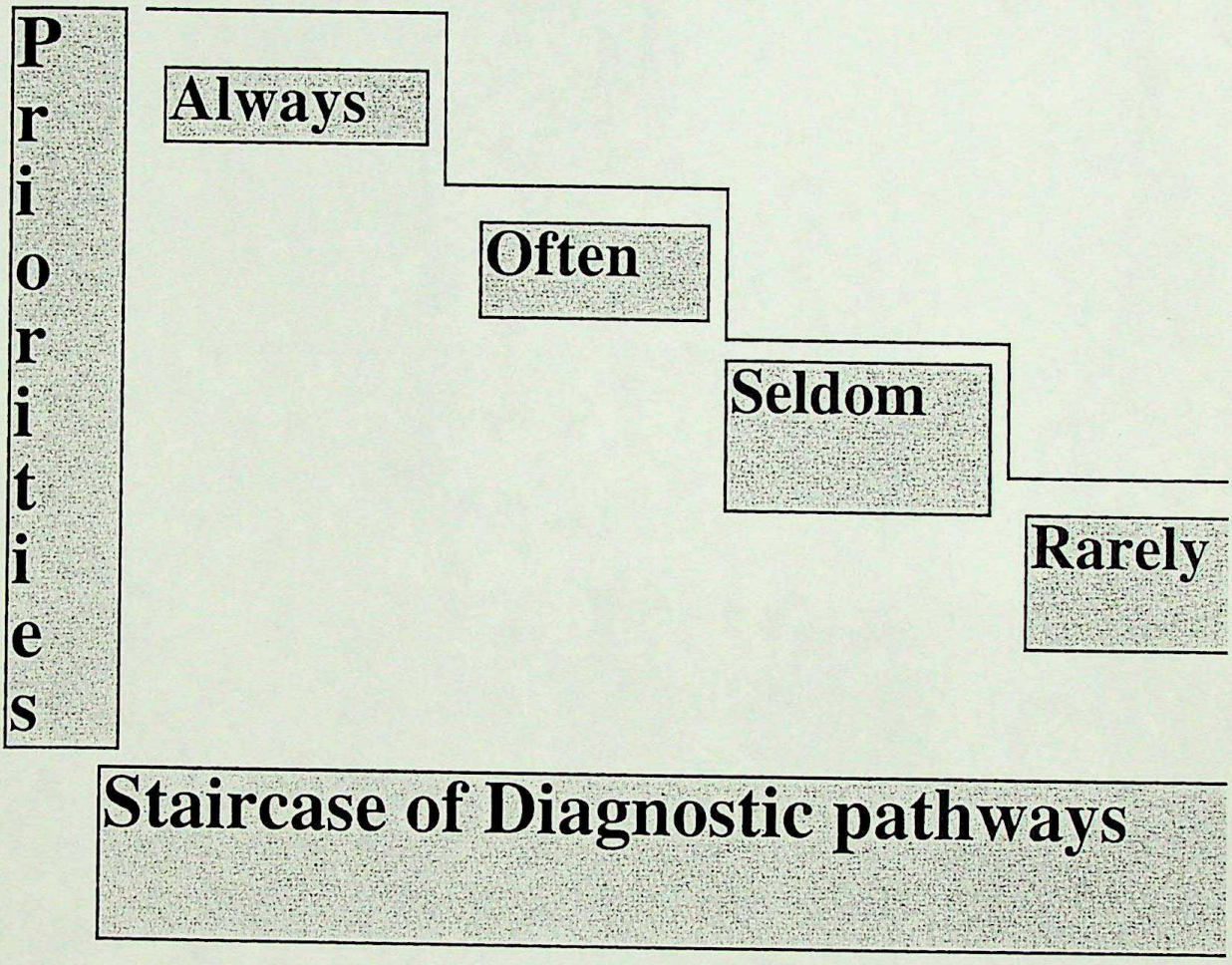

N.B. The section numbers shown after each symptom or disease entity follows the sections as given in the BNF.

Contents

Staircase diagnostics

Acute Watery Diarrhoea in childhood *1*

Major cause of child morbidity and mortality. Four categories to be identified: Acute watery diarrhoea, persistent diarrhoea, dysentery, chronic diarrhoea. Accurate history of onset, duration, severity, appearance of stool, presence of blood and mucus. On the basis of this classifying into one of above 4 categories. When classified as acute watery diarrhoea the two commonest causes are rota virus and Enterotoxigenic E. coli (ETEC) the latter with an adenylate cyclase like cholera toxin. Other E. coli strains are Enteroinvasive (EIEC) giving an inflammatory enteritis with blood and pain, Enterohaemorrhagic (EHEC) giving haemorrhagic colitis or haemolytic uraemic syndrome and Enteroadherent (EAEC) giving a watery diarrhoea.

Always assess degree of dehydration and classify to determine treatment

Decision on category of treatment (Plan A,B or C) after clinical assessment for state of consciousness, sunken eyes, ability to drink, skin turgor (either: normal, or goes back slowly after pinching, or very slowly - more than 2 secs.). Categorize into ***no dehydration*** (A), ***some dehydration*** (B) or ***severe dehydration*** (C). See appendix 11 for chart.

Often add simple laboratory investigations when these are available.

Combine above with microscopy of stool. If stool shows many white cells likely to be salmonella, shigella, Campylobacter or EIEC even if blood and fever are absent. If few white cells present likely to be due to rota virus, ETEC or food poisoning. If Giardia or trophozoites of E. histolytica found start specific treatment with metronidazole.

Seldom add best diagnostic practice disregarding cost.

Combine steps 1 and 2 with addition of stool culture, latex agglutination test for rota virus in stool.

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Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.

Polyacrylamide gel electrophoresis with silver stain (PAGE-SS) or PCR in diagnosis of rota-virus.

b. Poor diagnostic practice which is now obsolete.

Waiting for definitive diagnosis before initiating rehydration therapy

References

Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. Oxford University Press. Oxford.

Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases 4th Ed. 1995 Churchill Livingstone, New York.

Management of Childhood Illness. Treat the Child. WHO and UNICEF 1995 WHO/CDR/95.14.D

Staircase diagnostics

Persistent Diarrhoea in childhood *1*

Once good coverage of rehydration in acute watery diarrhoea has been achieved, persistent diarrhoea is a major cause of child morbidity and mortality. Persistent diarrhoea defined as diarrhoeal episodes that began acutely but continue at least 14 days. Often associated with malnutrition. Often in the first year of life, more likely when animal milk is given to the child. Sometimes pathogens are isolated and the most likely are: Shigella, Salmonella, Campylobacter jejuni, Entero-adherent E. Coli (EAEC), Cryptosporidium and Vibrio cholerae.

Always focus on clinical diagnosis first even **where microscopy is available.**

Accurate history of onset, duration, severity, appearance of stool, presence of blood and mucus, fever, vomiting, convulsions, state of consciousness, amount of urine passed, thirst, ability to drink. Check child for nutritional status with weight for age, weight for height and mid-upper arm circumference if between 1-5 years. Check stool for blood and reducing substance if simple test strips are available.

Often add simple laboratory investigations where available.

Combine above with microscopy of stool to look for plentiful white cells. If present evidence of inflammatory enteritis caused by Shigella, Salmonella, EIEC or EAEC (see under Watery diarrhoea). May favour use of antibiotics. Check for Giardia or trophozoites of Entamoeba histolytica. If present treat specifically. Check urine with microscopy to look for white cells and nitrite strip test to look for evidence of urinary tract infection.

Seldom add best diagnostic practice disregarding cost.

Combine steps 1 and 2 with addition of stool, urine and blood culture and sensitivity.

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Black,R.E. Persistent diarrhea in children of developing countries. Pediatric Infect. Dis. J. 1993 12:751-761

Evaluation of an algorithm for the treatment of persistent diarrhoea: a multicentre study. Bull. of WHO 1996. 74:479-489

Persistent diarrhoea Update from CDD at WHO No 4 March 1989

Staircase diagnostics

Dysentery in Childhood *1*

Major cause of child morbidity and mortality in situations of overcrowding such as refugee camps. Defined as acute diarrhoea with blood in stool. Likely cause Shigella, Salmonella, EIEC, Campylobacter jejuni, amoebic dysentery.

Always focus on clinical diagnosis first even **where microscopy is available.**

Accurate history of onset, duration, severity, appearance of stool, presence of blood and mucus, fever, vomiting, convulsions, state of consciousness, amount of urine passed, thirst, ability to drink. Clinical assessment for state of consciousness, sunken eyes, ability to drink, skin turgor (either: normal, or goes back slowly after pinching, or very slowly - more than 2 secs.). Categorize into ***no dehydration, some dehydration*** or ***severe dehydration.*** See Appendix 11.

Check stool to confirm that blood is present. If macroscopic blood is absent check with strip test if available to see if microscopic blood is present. Check child's general condition looking for evidence of systemic invasion with high fever, rapid pulse, cold extremities, pallor etc. These will favour the use of antibiotics early and i.v. fluids.

Often add simple laboratory investigations where available.

Combine above with microscopy of stool to check for presence of large numbers of WBC. If so evidence favours bacterial cause. May favour antibiotic treatment. Check for trophozoites of Entamoeba histolytica and if present treat specifically.

Seldom add best diagnostic practice disregarding cost.

Combine steps 1 and 2 with culture and sensitivity of stool. Serotyping of Salmonella if found.

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Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.
b. Poor diagnostic practice which is now obsolete.
c. Contra-indicated diagnostic practice.

e.g. Delay in getting stool to laboratory since organisms (esp. Shigella) is delicate and perishes in an acid medium. Must be refrigerated or placed in a buffered transport medium if there is to be delay.

References

Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. Oxford University Press. Oxford.

Staircase diagnostics

Chronic Diarrhoea *1*

Here diarrhoea often starts more slowly and continues for many weeks.

Always focus on clinical diagnosis first even **where microscopy is available.**

Accurate history of onset, duration, severity, appearance of stool, presence of blood and mucus, fever. Check for fever, wasting and blood in stool. Try to categorize into one of following three groups:

a). Chronic diarrhoea, wasting and fever: likely to be due to AIDS, TB bowel or visceral leishmaniasis

b). Chronic bloody diarrhoea: likely to be due to intestinal Schistosomiasis, heavy intestinal fluke, trichuris, neoplasm of large bowel, amoebiasis or ulcerative colitis.

c). Chronic fatty diarrhoea: likely to be due to tropical sprue, gluten enteropathy, giardiasis, abdominal lymphoma, TB of abdominal lymph nodes and chronic pancreatitis or pancreatic lithiasis.

Often add simple laboratory investigations where available.

Combine above: *Always* with microscopy of stool looking especially for intestinal schistosmiasis, flukes, giardia or trichuris, trophozoites of Entamoeba histolytica, excessive fat cells

Seldom add best diagnostic practice disregarding cost.

Combine steps 1 and 2 with addition of colonoscopy, barium enema, fat estimations in stool, if indicated jejunal biopsy looking for gluten enteropathy changes, spleen puncture if visceral leishmaniasis is suspected, Mantoux reaction or accelerated BCG test for TB.

N.B. rarity of tropical sprue, gluten enteropathy, ulcerative colitis in African subjects.

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Staircase diagnostics

Cholera *1*

Caused by Vibrio cholerae with two major biotypes: classical and El-Tor, the former being the more severe. The clinical case : infection ratio is 1:6 for classical and as low as 1:50 in El Tor. The closest clinical mimic is Enterotoxigenic E.coli (ETEC)

Always focus on clinical diagnosis first even **where microscopy is available.**

In epidemics the diagnosis is obvious in most clinical cases but in sporadic illness the ***strongest predictors*** of cholera are rapid onset of painless watery diarrhoea which soon goes over to rice-water stools and in many, effortless vomiting. When fluid loss is severe, muscle cramps especially of the calf muscles are common. In the severe cases progress from onset to shock takes 4-12 hours with death following in 18 hours or more without treatment. Once shock has set in, the clinical picture is characteristic with a collapsed cyanotic patient with no peripheral pulses, pinched facies and scaphoid abdomen with extreme loss of turgor of the skin. The voice is weak, high-pitched and often nearly inaudible. Usually the patient remains surprisingly oriented but apathetic. Adults with cholera have no fever.

Often add simple laboratory investigations where available.

Microscopy of stools show no leucocytes or erythrocytes unless there has been a preceding inflammatory bowel disease. Dark-field microscopy shows rapidly moving comma-shaped bacilli in a fresh specimen. These are immobilized with group and type specific antisera.
A new rapid colorimetric immunodiagnostic kit Cholera SMART can be used without laboratory equipment with high specificity and sensitivity.

Seldom add best diagnostic practice disregarding cost.

Culture the stool on TCBS agar where they grow as distinct opaque yellow colonies.
Distinguish two major serotypes: Inaba and Ogawa by slide agglutinatioin with type specific antisera. El Tor is distinguished from classical cholera because it is resistant to polymyxin B.

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Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases 4th Ed. 1995 Churchill Livingstone, New York 1934-1943.

Hasan JA et al.. A novel kit for rapid detection of Vibrio cholerae O1. J Clin Microbiol. 1994 32: 249-252

Staircase diagnostics

Peptic ulcer *1*

Abdominal pain is one of the commonest presenting symptoms and the history is paramount. Below are given features which give a clue to peptic ulcers but see also Appendix 5 for an approach to other causes of abdominal pain. Infection of the gastroduodenal mucosa with Helicobacter pylori is present in virtually all patients with duodenal ulcers and in 80% of those with gastric ulcers. There is likewise a link with this organism in gastric carcinoma. All cases need assessment at the level of first referral hospital or above.

Always focus on clinical diagnosis first even **where endoscopy is available.**

Suggesting duodenal ulcer: epigastric or back pain occurs when the stomach is empty and often wakes the patient in the early hours of the morning. Relieved by milk, bland food, antacids and H_2 antagonists. Smoking and alcohol make the pain worse. If the ulcer penetrates backwards especially into the pancreas pain may be only felt in the back.

Suggesting gastric ulcer: epigastric pain comes on soon after food. Patient may be afraid to eat. Relieved by vomiting and by antacids.

Suggesting gastric carcinoma: The pain is constant and not periodic i.e. it never goes away completely. It is uncommon below the age of 40 yrs. Rare to coexist with duodenal ulcer. Often poor appetite, epigastric fullness after a meal, bad taste in the mouth, unpleasant eructations, loss of weight, anaemia. May find hard lymph nodes in the left supraclavicular fossa or deposit in the pouch of Douglas. N.B. many have no symptoms until late in the disease.

At this level where advanced surgery is unlikely, if the history is typical give trial of therapy for an ulcer: see Staircase Pharmaceuticals. However be aware that gastric carcinoma may have relief of symptoms, but at this level the chances of curative surgery are virtually non-existent.

Often add good diagnostic practice where investigative facilities are available.

Add to the above: endoscopy, if available, looking for an ulcer, neoplasm or gastritis. Otherwise do barium meal which is less reliable than endoscopy.

Seldom add best diagnostic practice disregarding cost.

Prior to endoscopy assess serology for H. pylori (ELISA for IgG antibody) and 13C urea breath test. If these are negative, duodenal ulcer is ruled out and endoscopy is not needed.

N.B. In HIV/AIDS a common mimic to peptic ulcer is Kaposi sarcoma of the stomach.

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Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. OUP. Oxford. 1877-1891

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Staircase diagnostics

Hypertension 2

*Hypertension is not a disease but a risk factor. It is defined as a sustained diastolic blood pressure > 100 mm on 3 readings each a week apart. There are genetic factors so a family history is essential. In 95% the cause is unknown. In the remaining 5% the causes are renal, endocrine disease, coarctation, or pre-eclampsia in the second half of pregnancy. Concentrate more advanced investigation for the younger (under 30 yrs). See below for the technique of BP assessment.**

Always focus on clinical diagnosis first even **where laboratory is available.**

There are no inevitable clinical signs and symptoms of hypertension until complications such as a stroke have appeared. However especially when the diastolic is above 110 mm the person may complain about dizziness and headache, they may have heavy nose bleeds, signs of heart failure or have protein in the urine on routine testing.

To make a diagnosis of hypertension take three BP readings one week apart. Make the diagnosis straight away if the diastolic is more than 110 after resting. With any abnormal reading check BP in both arms at the first visit. Check the femoral pulses. Listen over the renal arteries. Check the urine for protein. Look at the fundi for silver wiring, A-V nipping, exudates, haemorrhages.

Often add good diagnostic practice where a laboratory is available.

Add to the above: urea, electrolytes and creatinine. Usually normal unless secondary renal damage has occurred. Urinary casts, haematuria and proteinuria usually indicate that the hypertension has entered the malignant phase or reflects primary renal disease. ECG may show left ventricular hypertrophy and provides a good criteria for the severity of the hypertension.

Seldom add best diagnostic practice disregarding cost.

Especially in younger hypertensives rule out the rarer causes such as renal (3%) and renovascular (1%) hypertension suggested if there is polyuria, nocturia, accelerated phase hypertesion or hypertension in someone under 30 yrs. Look for known family history of renal disease or other known vascular abnormalities. Add to the above, microscopy of urine looking for granular or cellular casts. Culture the urine. Renal ultrasound may show polycystic kidneys or obstructive uropathy. If two kidneys are of unequal length more than 1.5 cms listen extra carefully for renal bruit. Consider renal arteriography. IV urography is especially valuable in diagnosis of chronic pyelonephritis with renal scarring (the most likely cause of hypertension in people under 20 yrs.). Renal biopsy is only indicated when other renal diseases have been ruled out and to determine whether renal impairment in a patients with accelerated phase hypertension is due to primary kidney disease which could be treated specifically or whether it is a secondary phenomenon.

If the person describes episodic feelings "as if about to die" consider phaeochromocytoma (<1%) and do 24-hour urinary VMA x 3. Urinary free cortisol is found in Cushing's syndrome.

***Technique:** **the BP is taken with the patient sitting or reclining comfortably in a relaxed atmosphere. Apply an appropriate sized cuff (>half the arm's circumference) with its midpoint over the position of the maximal pulsation of the brachial artery. Determine the systolic pressure by palpation and after letting the pressure down completely, place the stethoscope lightly over the artery. Pump up the cuff to 30 mm more than the point at which the radial artery disappeared. Release at a rate of 2-3 mm per sec. When the first regular sound is heard take this as the systolic pressure and when the sounds completely disappear (phase V Korotkoff) take this as the diastolic pressure, both to the nearest 2 mm. Repeat.**

References

Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. OUP. Oxford. 2527-2553

Oxford Handbook of Clinical Medicine. Hope, RA. et al. 1996. 3rd Edition. OUP. Oxford.

Staircase diagnostics

Heart failure 2

This is failure of the heart to pump enough blood for the needs of the body tissues. This may be due to high ouptput failure where the demands of the tissues are increased e.g. anaemia, thyrotoxicosis and beri-beri or low-output failure where there is damage to the heart muscle, valves or a septal defect, hypertension, lung disease, pulmonary embolism, or irregular heart beat. All cases need assessment at the level of first referral hospital or above.

Always focus on clinical diagnosis first even **where ECG and Xray are available.**

Assess on symptoms and signs. ***High output failure:*** *symptoms* of breathlessness esp on exertion, palpitations and swelling of the ankles. *Signs* of increased pulse vol. and raised systolic pressure, warm extremities, ankle oedema and rales in the chest. Look for pallor (anaemia), tremor (thyrotoxicosis), ask about diet (beri-beri), alcohol intake (beri-beri).

Low output failure more common: mainly **left-sided failure** dominated by pulmonary oedema. *Symptoms* exertional dyspnoea, orthopnea, paroxysmal nocturnal dyspnoea, wheeze (cardiac asthma), cough, haemoptysis and fatigue. *Signs* tachypnoea, tachycardia, basal rales, third heart sounds, pulsus alternans (alternating large and small pulse pressure), cardiomegaly, peripheral cyanosis, pleural effusion. Check BP, heart rhythm, any murmurs.

Right-sided failure *Symptoms:* peripheral oedema, abdominal discomfort, nausea, fatigue, wasting. *Signs:* raised JVP, hepatomegaly, pitting oedema, peripheral cyanosis. Check for evidence of lung disease, pain of pulmonary embolus, heart murmurs, heart rhythm.

In the common cardiomyopathy that is dominant in most parts of Africa there is usually both right and left sided failure with a dilated heart (the diagnosis is by excluding other causes).

Often add good diagnostic practice where investigative facilies are available.

Add to the above: Chest Xray looking for other pulmonary disease or signs of pulmonary oedema: alveolar odema (Bat's wings), cardiomegaly, Kerley B lines and pleural effusion; ECG looking for arrhythmia, evidence of myocardial infarction or ischaemia;
flattened T waves, arrhythmia in cardiomypopathy usually of the dilated variety.

Seldom add best diagnostic practice disregarding cost.

Add echocardiography which is especially valuable in dilated cardiomyopathy showing a globally hypokinetic, dilated heart. Also it shows valve damage, pericardial disease.

In selected cases cardiac catheterization and radionuclide ventriculography may be indicated.

Heart failure can be graded according to the New York Heart Association classification:

NYHA I Organic heart disease without symptom

NYHA II Mild heart failure with breathlessness and tiredness more than expected only after fairly vigorous physical activity e.g. running, climbing, heavy body work

NHYA III Moderate heart failure with breathlessness and fatigue after light physical activity e.g. walking up a slight slope; in the later stages fatigue even after dressing/undressing.
Sometimes divided into **A:** can walk 200 metres on the level without difficulty and
B: worse than this.

NHYA IV Severe heart failure with breathlessness and fatigue even at rest. Increasing symptoms at even the slightest activity.

This grading is especially of value in following the effect of treatment and in prognosis. Grade I has a yearly mortality of 5% whereas Grade IV has a yearly mortality of 40-50%. Early diagnosis and treatment can reduce these figures.

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References

Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. OUP. Oxford. 2228-2238
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Staircase diagnostics

Myocardial infarction 2

Edgar Borgenhammer the Swedish Health Planner has described the three phases of ill-health in Sweden: the diseases of poverty, the diseases of wealth and the diseases due to lack of trust. Many countries are in a transition phase from the diseases of poverty to the diseases of wealth and many countries have now a double burden of both kinds of diseases. MI is a classical disease of wealth which often comes in the first generation after leaving poverty. See Appendix 6 for approach to Chest pain. All cases need assessment at the level of first referral hospital or above.

Always focus on clinical diagnosis first even **where ECG and laboratory are available**

Suggesting diagnosis of MI: Many will have had prodromal symptoms of undue fatigue or shortness of breath. Once the infarct has occurred the typical history is that of crushing (or tightness or bursting sensation) central or retrosternal pain often radiating into neck and upper limbs, persisting >20 mins and not relieved by glyceryl trinitrate; often accompanied by nausea and sometimes vomiting, feeling of impending death, sometimes breathlessness (however N.B. some older patients may have little or no pain with an infarct). In the equally severe pain of pericarditis the feeling of impending doom is not prominent.

Signs may be remarkably few if any: a third heart sound, tachycardia, sweating, pallor, raised venous pressure. In large infarctions, hypotension, circulatory collapse, arrhythmias and shock. True shock that is not just due to the vasovagal reflex has a very poor prognosis (80% mortality).

N.B. MI is virtually ruled out in a rural African patient. It is extremely rare in the poorer urban African community. More likely if symptoms occur in someone of a European, American or an Indian extraction and in well-off Africans especially if they have lived on a Western diet for many years.

Often add good diagnostic practice where investigative facilities are available.

Add to above: ECG assessment. Regional ST elevation or new Q-waves appearing are strongly suggestive. In a simpler lab check SGOT or AST and serum creatinine kinase and follow levels over the first days.

Seldom add best diagnostic practice disregarding cost.

Assess CKMB. Doubling in levels of CKMB in 3-6 hours after onset is diagnostic. An echocardiogram can be a useful tool after treatment has been started to assess left ventricular wall motion. Later a routine chest Xray is of value (only done as an emergency if a dissecting aneurysm is suspected).

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Oxford Handbook of Clinical Medicine. Hope, RA. et al. 1996. 3rd Edition. OUP. Oxford.

Staircase diagnostics

Upper respiratory infection with pharyngitis *3*

It is important to try to distinguish between viral pharyngitis and acute streptococcal pharyngitis. Approx 50% of episodes of URTI caused by rhinovirus followed in frequency by coronavirus, influenza, parainfluenza virus, RSV, adenovirus and enterovirus. However 12 million people have acute rheumatic fever or rheumatice heart disease complicating infection (usually tonsillitis) by Lancefield Group A ß-haemolytic Streptococcus pyogenes and 400 000 die each year as a result.

Always. Simple diagnostic practice based on careful clinical assessment.

Clinical signs suggesting **streptococcal pharyngitis**: tonsillitis with exudate and/or enlarged anterior cervical lymph node; grossly enlarged painful tonsils which are asymmetrical with glottal speech. More likely in age group 5-15 yrs. With scarlatina or in a strep. epidemic differentiation is easy.

Suggesting **viral pharyngitis**: marked nasal stuffiness and coryza, an irritating cough and hoarseness (URTI); during influenza epidemic, coryza, pharyngitis, cough, hoarseness, fever and general body pain (influenza); tonsils covered by a membrane with offensive breath and many lymph nodes enlarged (infectious mononucleosis); vesicles in the mouth and gingivitis (herpes simplex).

Often. Good diagnostic practice with good cost-effectiveness.

Combine *Always* with culture of a throat swab and/or rapid antigen detection method using latex agglutination or enzyme immunoassay. If throat culture yields more than 50 colonies almost certainly an acute infection and not a carrier. If less than 10 could be a carrier.

Seldom. Best diagnostic practice disregarding cost.

As in *Often*.

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Staircase diagnostics

URTI and Otitis media in childhood 3

URTI is the commonest acute illness of childhood. In 80-90% infection is confined to the upper airways and in the rest there is involvement of the lower tract. The peak incidence is in infancy at 6-8 months but it remains frequent up to 5 years. There are on average 5-7 episodes per year. If there are siblings at school the rate increases to 9/yr. Commonest organism: rhinovirus, then adenovirus, coronavirus, influenza, and RSV.

In acute Otitis media 60% are caused by bacteria (pneumococcus and H. influenzae dominate; rarely streptococcus, staphylococcus are found. Moraxella catarrhalis is prominent in some areas), the rest by viruses. However most bacterial infection follows a viral illness. The peak incidence is in the first 3 years of life. By 3 years more than 2/3 will have had at least one episode and one third will have had 3 or more episodes.

Always. Simple diagnostic practice based on careful clinical assessment.

Assess acute airways infection according to IMCI method: *see under URTI and pharyngitis.*

Acute otitis media is defined by presence of fluid in the middle ear together with signs and symptoms of acute illness. If a child complains of ear-ache or deafness check both ears with a otoscope. If there is true acute otitis media the drum is red, inflamed often bulging with injection of blood vessels and loss of the light reflex. The presence of fluid is assessed by pneumatic otoscopy: the motion of the tympanic membrane is correlated with squeezing the rubber bulb attached to the otoscope. If fluid is present the movement is dampened or absent. If there is a diffuse pinkness or redness with a normal light reflex and normal mobility on pneumatic otoscopy regard this as a milder otitis probably due to a viral illness without secondary bacterial infection. *See Appendix 12.*

Often. Good diagnostic practice where investigative facilities are available.

If in doubt about giving an antibiotic take a white cell count and differential and CRP

Seldom. Best diagnostic practice disregarding cost.

Can do a careful thin needle aspiration of the middle ear to culture the organism.

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References

Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases 4th Ed. 1995 Churchill Livingstone, New York 579-585.

Staircase diagnostics

Pneumonia in childhood 3

In low-income-countries this is the biggest cause of mortality in children under 5 years

Always focus on clinical diagnosis first even **where Xrays and laboratory are available.**

Use of the Integrated Management of Childhood Illness (WHO) criteria for establishing the diagnosis in children i.e. fast breathing (>60 up to 1 month, >50 1-12 months, >40/min. in 1-5 year-olds) +/- indrawing of the lower chest wall during inspiration. Presence of cyanosis and/or inability to drink as signs of severe pneumonia. See appendix 12 with chart.

Often. Good diagnostic practice with good cost-effectiveness.

Use of above, combined with auscultation, percussion and if possible Chest Xray. (however note lack of value of Chest Xray in ambulatory children*)

Seldom. Best diagnostic practice disregarding cost.

Use of above, combined with culture of blood, and sputum in older children, blood gases especially arterial pO_2 . Spirometry where there is obstructive element. Diagnostic bronchoscopy rarely needed in community acquired pneumonia (except in suspected obstruction) but may be necessary in nosocomial and ventilator-associated pneumonia.

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Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.

Percutaneous lung aspiration (but pneumothorax caused in 10%, usually insignificant). Thoracoscopy and lung biopsy under direct vision.

b. Poor diagnostic practice which is now obsolete.

Transtracheal aspiration through cricothyroid membrane. May cause mediastinal emphysema and significant bleeding

c. Contra-indicated diagnostic practice.

Invasive investigation in a child with a bleeding tendency.

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Staircase diagnostics

Lobar pneumonia in adults 3

Community acquired pneumonia is a common cause of morbidity and mortality in low-income-countries. It accounts for 10% of adult medical admissions in Sub-Saharan Africa. There is a mortality of around 5-10% despite antibiotics. Most patients with lobar or segmented pneumonia are infected with Strep. pneumoniae and Haemophilus influenzae. There is a link with alcoholism, smoking and pre-existing heart disease and a strong link with HIV infection.

Always focus on clinical diagnosis first even **where Xrays and laboratory are available.**

Over 90% of patients with a lobar pneumonia have a cough and 60% have pleuritic chest pain. Almost all are tachypnoeic i.e. >25/min. On auscultation most have crepitations over the affected lobe and bronchial breathing usually develops in at least half. The most valuable test is to use the pointing sign: ask the patient to put his hand to his mouth and give a big cough and then point to the site of chest pain. By inadvertently stretching the parietal pleura by the forced cough the localization of the pneumonia increases from 75% to 90%. Jaundice is seen in 5-25% especially when the lobe affected is on the right side. Diarrhoea is seen in 10-25% and muscle tenderness in up to 60% with an enlarged tender liver in up to 60%. The patients in danger are the old, those with multiple lobes involved, those that are HIV positive, those with hypothermia and hypotension and a low WBC.

Often. Good diagnostic practice where investigative facilities are available.

Chest Xray to look for pleural fluid, lung abscess and an enlarged heart. Do a white cell count and differential and CRP. However in 25% the WBC is normal and in 5% it is below normal. Do a Gram's stain on the sputum looking for gram positive diplococci (Strep. pneumoniae).

Seldom. Best diagnostic practice disregarding cost.

Use of above, combined with culture of blood, and sputum, blood gases especially arterial pO_2. Spirometry where there is obstructive element. Diagnostic bronchoscopy is rarely needed in community acquired pneumonia (except in suspected obstruction) but may be necessary in nosocomial and ventilator-associated pneumonia. Rarely there may be a place for transtracheal aspiration through the cricothyroid membrane in order to get a sample for culture. But this may cause mediastinal emphysema and significant bleeding.

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Staircase diagnostics

Lung abscess 3

This describes a suppurative lung infection with necrosis of tissue, seen as a rounded opacity on Chest Xray and often with a fluid level. It is usually caused by aspiration of debris or fluid and anaerobic bacterial contamination. There is a strong link with alcoholism, drug abuse, smoking, epilepsy and stroke. Post-anaesthesia may be a cause when the skill of the anaesthetist is limited. Bronchial obstruction due to bronchial carcinoma may be present. In childhood it is often caused by a foreign body such as a groundnut. All cases need assessment at the level of first referral hospital or above.

Always focus on clinical diagnosis first even **where Xrays and laboratory are available.**

Suspect it in a patient with an illness of sudden onset with shivers, fever, cough and pleuritic pain. After one week the abscess may discharge blood-stained sputum into a bronchus. This is coughed up and is often foul-smelling because of the anaerobes. There may be localized bronchial breathing and crepitations and later some may show amphoric breathing. Clubbing develops rapidly. Empyema develops in 20-30%.

Often. Good diagnostic practice where investigative facilities are available.

Chest Xray. Look for a round shadow with a fluid level. Look for pleural fluid, any shadow consistent with bronchial carcinoma. Do a white cell count and differential and CRP.

Seldom. Best diagnostic practice disregarding cost.

Use of above, combined with culture of blood, and sputum, blood gases especially arterial pO_2 Consider bronchoscopy if there is any suspicion of obstruction. It may also be needed for culture specimens. Percutaneous transthoracic aspiration sometimes called for. If there is pleural fluid take a sample for culture.

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Staircase diagnostics

Asthma 3

Asthma is characterized by variable airways obstruction with often normal lung function between attacks. It is today seen as an inflammatory disorder of airways mucosa, with bronchial hyper-responsiveness as a consequence of inflammation rather than the primary phenomenon. In children it is related to atopic allergy with production of large amounts of IgE in response to allergens. Often adult asthma arises in non-atopic individuals. In children the initiating trigger is often an infection with rhinovirus, unusually vigorous exercise, exposure to cold air, allergens such as house dust mite, or animal furs, plant pollens (however more often the eye and upper airways effect dominates). Less commonly there may be response to food or taking an NSAID such as aspirin

Always focus on clinical diagnosis first even **where Xrays and laboratory are available.**

The three pointers in the history are sudden onset of breathing difficulty, with a wheeze brought on by some clear-cut trigger. The cardinal symptom of asthma is wheezing usually coming in attacks (however a small group of asthmatics never wheeze). This wheeze is polyphonic i.e. is present on inspiration and expiration and is often not detectable at rest or heard only on exercise or forced expiration. Asthmatics often find it more difficult to breathe in than out even though most doctors think the opposite. Cough confined to the night suggests asthma.

Ask about other signs of atopic disease: eczema, hay fever, family history of asthma.

Check for any signs of heart failure to rule out cardiac asthma which may confuse.

Often. Good diagnostic practice where investigative facilities are available.

The cardinal investigation is the peak expiratory flow (PEF) using a flow meter. If this is reduced (<80% of expected) and marked improvement (>20%) comes after inhaling a bronchodilator (β-2-agonist) then the diagnosis is virtually certain. If there is <20% improvement after inhaling a bronchodilator but there is more than 20% improvement after treating with inhaled steroids over a period of time, then asthma is still the most likely diagnosis. If neither improve the PEF, the most likely diagnosis is chronic obstructive airways disease. In asthma patients if the PEF is followed throughout a day without an attack the highest levels are in the evening and lowest between 3-5 a.m.

Seldom. Best diagnostic practice disregarding cost.

Investigation of possible allergens is worthwhile in younger asthmatics but the former enthusiasm for desensitization procedures has waned. Some sensible easily achieved measures to reduce exposure to allergens are worthwhile.

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Staircase diagnostics

Headache 4

This is the commonest symptom bringing a patient to a general practitioner who is likely to see 1000 headaches a year and one cerebral tumour every 5-10 years. Febrile illnesses such as malaria, influenza, typhoid, trypanosomiasis, meningitis and rickettsial diseases need to be excluded first before going on to investigate other causes of headache. Most of diagnosis comes from the history especially if the patient is allowed to develop the details themselves. Tension headaches are the commonest cause of chronic headaches. Referral to a neurologist or lab tests and other investigations rarely needed.

Always. Simple diagnostic practice based on careful clinical assessment.

Accurate history of timing, site, aggravating, relieving factors, preceding and accompanying symptoms. Try to fit into one of the following categories:

A. Acute single episode. If bilateral with drowsiness, neck stiffness, headache worse on moving eyes, photophobia suspect meningitis, encephalitis or subarachnoid haemorrhage even if fever is absent.

B. Acute recurrent attacks. Look for classical groups of symptoms suggesting one of the following:

Migraine. Paroxysmal headaches with 2 of 4 features: unilateral, nausea, family history, focal symptoms e.g. flashing lights, fortification spectra.

Cluster h. daily bouts of severe pain in one eye radiating to forehead, temple, or cheek, usually wakens suddenly at night ('alarm-clock' h.). Relieved by intranasal 4% lidocaine on same side.

C. Sub-acute onset. If patient is over 55 and expatriate exclude *giant cell arteritis*. Look for tender thickened pulseless temporal arteries and do ESR. May have fever. May be accompanied by rheumatism in shoulder or pelvic girdle.

D. Chronic headache. This is the commonest group and *tension headache* is by far the commonest of all. Worse in evening, sense of pressure, feeling of tightness, Arm-chair test: tell patient to relax completely and imagine that they are sitting in an arm-chair while you hold their arms up. Then let go. Those with tension h. keep arms suspended at same level.

Intra-cranial disease. Headache worse in morning, aggravated by sitting up or standing up, by coughing or straining. Later associated with vomiting. May be focal signs. Late onset epilepsy very suggestive.

Often. Good diagnostic practice when clinical skills are available.

Combine *Always* with careful neurological examination looking for papilloedema, cranial nerve plasies (esp. VIth), abnormal reflexes. Check BP, pulses, look for tender temporal artery. Where encephalitis or meningitis possible do LP with CSF assessment.

Seldom. Best diagnostic practice disregarding cost.

In rare cases where history and signs are suggestive of intra-cranial disease combine steps 1 and 2 with addition of CT scan or magnetic resonance imaging (MRI).

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Not recommended diagnostic procedure

b. Poor diagnostic practice which is now obsolete.

Labelling chronic headache as being due to low-grade malaria if blood slides are consistently negative.

c. Contra-indicated diagnostic practice.

Doing LP in the presence of raised intra-cranial pressure

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Staircase diagnostics

Coma 4

In a malaria area cerebral malaria is the most important condition to diagnose early because delay has such devastating consequences and a surprising number do well on treatment. Main treatable causes in Africa are malaria, hypo and hyperglycaemia, poisoning with carbon monoxide, alcohol, opiates or organophosphates, meningitis, brain abscess, extradural and subdural haematomas, septicaemia, head injury, eclampsia. All cases need assessment at the level of first referral hospital or above. Use Glasgow or Blantyre coma scale to follow course. See appendix 2.

Always. Simple diagnostic practice based on careful clinical assessment.

Focus on history: in a holoendemic or hyperendemic area for malaria ask the family of anyone over 6 years where this person has been living for the last 6 years. If it is in the holoendemic area then malaria is excluded as a cause for the coma even if a few parasites are found in the blood smear. If the person has been living away from the holoendemic area, or is under 6 years, then cerebral malaria must be considered. Is there any history of injury, fever, diabetes, alcohol abuse, exposure to carbon monoxide?

Check for focal motor signs (supratentorial causes suggests stroke, tumour or abscess), neck stiffness, BP, pupils (abnormal pupil responses suggests infratentorial cause e.g. brain-stem stroke or raised intra-cranial pressure) (pin-point pupils that respond to light found in opiate poisoning. N.B. need hand-lens to see this). Symmetrical pupils responding to light suggests metabolic cause.

If no history is available and malaria has been excluded with a blood slide, give 50ml of 50% glucose iv after taking blood for glucose. If there is any risk of alcoholism give thiamine 50 mg i.v. If there is any risk of opiate poisoning give naloxone 1 mg i.v.

Often. Good diagnostic practice where clinical skills are available.

Combine *Always* with careful neurological examination looking for papilloedema, cranial nerve palsies (esp. VIth), abnormal reflexes, (cerebral malaria may show divergent squint, invariably absent abdominals, brisk jaw jerk, 15% have retinal haemorrhages) . If no history of injury or grand mal convulsion, no papilloedema, no severe hypertension and no hypoglycaemia do an L.P. One third of these remaining will have meningitis. If CSF negative check for typhoid, trypanosomiasis (in certain areas only), evidence of herbal overdose.

Seldom. Best diagnostic practice disregarding cost.

Where evidence suggests intra-cranial disease combine steps 1 and 2 with addition of CT scan or magnetic resonance imaging (MRI).

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Staircase diagnostics

Stroke 4

A stroke results from either ischaemic infarction of part of the brain (80%), or from intracerebral haemorrhage (15%) or from a subarachnoid haemorrhage (5%). In high income countries this is the commonest reason for disability in adults and the 3rd commonest cause of death. It is a major cause of invalidity and death also in low-income-countries since the same degree of hypertension is a greater risk factor in black subjects. The most powerful risk factors are hypertension (70%), Diabetes mellitus and smoking. Age and obesity also increase the risk. Reduced level of consciousness at 24 hrs after onset is the most reliable indicator of poor prognosis. One year after the first stroke 33% are dead, 22% are dependent and 45% are independent. All cases need assessment at the level of first referral hospital or above.

Always. Simple diagnostic practice based on careful clinical assessment.

A stroke manifests as rapid onset of focal brain damage (within minutes). Sometimes there is step-wise progession over hours. Often the focal damage relates to the distribution of the affected artery. In cerebral hemisphere infarcts (50%) the common presenting symptoms are contralateral hemiplegia (flaccid weakness with the limb falling like a dead weight), sensory loss, homonymous hemianopia*, dyphasia** (if the lesion affects the dominant hemisphere). After a while flaccid paralysis goes over to spastic with all the signs of an upper motor neurone lesion. In lacunar syndromes (25%) small infarcts around the basal ganglia, thalamus and pons may cause pure motor, pure sensory, mixed motor and sensory signs or ataxia. In brainstem infarctions (25%) there are a wide range of effects with quadriplegia, disturbances of gaze and vision, and a locked-in syndrome where the patient is aware but unable to respond.

In all cases check BP, pulses, listen for carotid bruit, heart murmurs and check rhythmn, check reflexes including grasp reflexes and plantars, check level of consciousness, look for neck stiffness, check motor and sensory function, fundi.

Try to differentiate haemorrhage from infarct (accurate in 90%): **favouring haemorrhage:** 2 or more of the following including apopleptic onset 1. Loss of consciousness within hours, 2. Headache within 2 hrs. 3. Vomiting. 4. Neck stiffness. 5. Bilateral extensor plantar reflexes 6. BP significantly raised 24 hours after admission.

Favouring infarct: previous TIA or stroke, stepwise onset, heart disease with atrial fibrillation or mitral stenosis, atheroma markers (angina, claudication, diabetes); history of hypertension.

Mass lesions (tumours etc) are more likely to present with convulsions, neurological deficits which were prolonged in onset or discontinuous in evolution.

Often. Good diagnostic practice where investigative facilities are available.

Besides the above, try to localize the lesion. Easier with occlusion than haemorrhage.

1. Anterior cerebral artery: weak numb contralateral leg with milder effects of arm.
2. Middle cerebral artery (and internal carotid artery): contralateral hemiplegia and sensory loss mainly of face and arm. Dyphasia** if the dominant hemisphere is affected. Dyspraxias*** if the non-dominant hemisphere is affected.
3. Posterior cerebral artery: contralateral homonymous hemianopia*.
4. Vertebrobasilar circulation: hemianopia, cortical blindness, diplopia, vertigo, nystagmus, hemi- and quadriplegia, sensory symptoms on one or both sides, cerebellar symptoms, drop attacks, coma.
5. Brainstem vascular syndromes: this may cause a variety of syndromes e.g. lateral medullary

syndrome: severe vertigo with vomiting, dysphagia, nystagmus on looking to the side of the lesion, ipsilateral hypotonia and ataxia, ipsilateral paralysis of soft palate, ipsilateral Horner's syndrome, dissociated sensory loss (analgesia to pinprick on ipsilateral face and contralateral trunk and limbs.

A lumbar puncture has no place in the assessment of a stroke unless there is meningism to rule out meningitis or confirm a sub-arachnoid haemorrhage

Seldom. Best diagnostic practice disregarding cost.

Computerized tomography of the brain to rule out haemorrhage before commencing asprin.
Ultrasound of heart valves, carotid and subclavian arteries

*Homonymous hemianopia: patients lose vision on half of the visual field on the side opposite to the lesion.

** Dysphasia: impairment of speech with lack of coordination and failure to arrnge words in their proper order.

*** Dyspraxia: partial loss of ability to perform coordinated acts

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Staircase diagnostics

Epilepsy 4

Defined as recurrent transient attacks of abnormal cerebral function resulting from excessive discharge of neurones. 1 in 200 have recurrent fits in Europe with probably double this number in Africa due to brain damage, infection or parasites (e.g.cysticercosis). In the majority no underlying cause found. Focal epilepsy should always be regarded as symptomatic of some underlying condition.

Always. Simple diagnostic practice based on careful clinical assessment.

Get enough detail in the history to fit the epilepsy into one of the following categories:

Generalized seizures: a). Tonic clonic (grand mal): fits start with a tonic phase where all groups of muscles are strongly contracted. Tonic contraction can force expiration of air past tense vocal cords to give an epileptic cry. Consciousness is lost immediately and the patient falls with arms flexed and legs extended. The teeth are clenched and because respiratory movements cease the subject becomes cyanosed. There is loss of sphincter control. After 10 secs the tonic phase goes over to the clonic phase. Movements initially very rapid, gradually slowing to violent generalized shaking. The tongue may be bitten. This phase last 30 secs to 2 minutes. It then ceases and deep noisy respirations follow and then the subject sleeps.

b). Tonic only: often seen in children. Very rare in adults.

c). Akinetic: this is rare. The subject loses consciousness, falls but has neither tonic nor clonic spasms.

d). Typical absences (petit mal). These always start in childhood and most often found between 5-15 years. There are sudden periods of blankness, sometimes associated with rapid blinking and myoclonic jerks of limbs. The subject does not fall and is often unaware of the attack. These rarely last for more than a few seconds. There is a family history in half. This is relatively rare in Africa. The EEG pattern is diagnostic (3/sec spike and wave pattern).

Partial seizures: a). Simple motor seizures. Arise from the motor cortex of the frontal lobe. Jerking movements on contralateral side of face, limbs or trunk. Occasional spread of seizure with so-called "march" Jacksonian spread. Usually confined to one part of the body. There may be a resulting Todd's paralysis.

b). Simple sensory seizures. These cause tingling or numbness in the contralateral part of the body and arise in the sensory cortex. There may sometimes be a sensory "march".

c). Simple partial seizures. If they arise in the temporal lobe there may be olfactory hallucinations usually of an unpleasant odour, epigastric discomfort, feeling of dread, déja vu or jamais vu. In the occipital lobe it may cause visual hallucinations (rare)

d). Complex partial seizures. These usually arise in one of the temporal lobes. There is altered perception with epigastric sensation or olfactory hallucinations (aura) followed by the subject staring blankly, failing to respond to questions or commands, lip smacking or grimacing. Short periods of semi-purposeful automatic behaviour may occur.

e). Secondary generalization. This starts as a partial seizure then spreads to involve the whole brain with a generalized seizure usually after some seconds.

Often. Good diagnostic practice where clinical skills are available.

Add to above clinical assessment Xray of the skull for cysticercosis cysts which can be treated.

EEG where this is available especially in children with likely petit mal. Syphilis test.

Seldom. Best diagnostic practice disregarding cost.

CT scan for any of the types of epilepsy with a focal lesion.

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Staircase diagnostics

Depression 4

Clinical diagnosis from history is the only way of making this diagnosis but some tests can be necessary to rule out organic diseases that may mimic depression e.g. hypothyroidism.

Always. Acceptable diagnostic practice that can be taught at every level.

Look for clinical pointers to diagnosis:

Central features: low mood, reduced energy and activities, loss of enjoyment

Biological features: early morning wakening, diurnal mood variation, loss of appetite and weight, complaints about constipation, loss of libido, amenorrhoea

Cognitive features: pessimistic thoughts and feeling hopeless, guilty recollections, suicidal ideas. There may occasionally be delusions or hallucinations.

N.B. These features have to be weighed against the normal feelings of sadness that everyone feels and especially those who are or have been recently ill.

In many countries in Africa organic symptoms such as abdominal, chest or back pain or headache are usually prominent in the presenting complaints. Other symptoms will have to be actively sought in order not to miss the underlying depression.

Often. Good diagnostic practice where clinical skills are available.

Add to above, tests to rule out organic conditions that mimic depression e.g. hypothyroidism, Cushing's syndrome, dementia, alcohol abuse, pellagra, hypopituitarism.

Seldom. Best diagnostic practice disregarding cost.

Referral to a psychiatric specialist of difficult cases where other psychiatric conditions may be present such as bipolar illness with mania, schizophrenia, anxiety and agitation.

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Staircase diagnostics

Schizophrenia 4

Patients with schizophrenia will benefit greatly from specialist psychiatric supervision from primary diagnosis to follow-through of treatment. Unfortunately this is usually not available in many settings in low-income-countries and so a generalist may need to do the best possible. Clinical diagnosis from history is the only way of making this diagnosis. A consistent feature is a person who describes a subjective experience with a vivid sense of personal meaning out of proportion to what most listeners feel. Some tests and assessment can be necessary to rule out organic and other psychiatric diseases that may mimic schizophrenia. All cases need assessment at the level of first referral hospital or above.

Always. Acceptable diagnostic practice that can be taught at every level.

Suspect the diagnosis when one of the following group of symptoms is found:

Two syndromes are common:

an acute syndrome with delusions, hallucinations, and disordered thinking (positive symptoms)

chronic syndrome with apathy, slowness, and social withdrawal (negative symptoms).

First rank symptoms: Hearing thoughts spoken aloud, "third person" hallucination, somatic hallucinations, thought withdrawal or insertion, thought broadcasting, delusional perception, feelings or actions experienced as made or influenced by others.

All levels can start to suspect the condition when a typical picture emerges. Refer the patient to the most capable clinician that is available to make a definitive diagnosis and to initiate treatment.

Often. Good diagnostic practice where clinical skills are available.

Add to above, tests and psychiatric assessment to rule out organic and psychiatric conditions that mimic schizophrenia e.g. tertiary syphilis (general paralysis of the insane), acute brain syndrome, dementia with paranoid features (including pellagra), Cushing's syndrome, personality disorders, psychotic affective disorders, drug abuse.

Seldom. Best diagnostic practice disregarding cost.

Referral to a psychiatric specialist unit of difficult cases where other psychiatric conditions may be present such as severe personality disorders, psychotic affective disorders. Occasionally use of CT-scans and MRI to rule out focal lesions in the brain.

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Staircase diagnostics

Sexually Transmitted Diseases (STD) *5*

The major diseases apart from HIV are Chlamydia, gonorrhoea, syphilis, herpes, chancroid, papilloma, trichomonas, and candida. Less common causes include Lymphogranuloma venereum, granumoma inguinale and pubic lice.

There are 5 ways of establishing a diagnosis in an STD: visualizing the organism, antibody tests, culturing the organism, searching for antigens by non-amplified and amplified means, and clinical assessment including the ***syndromic approach.***

Always. Acceptable diagnostic practice that is cheap but less dependable **where microscopy is unavailable.**

Accurate history of symptoms including onset, duration, presence of abdominal pain, ulcer: ?painful, urethral discharge, burning micturition, and in women vaginal discharge, last menstrual period. Try to fit into one of four **syndromes:**

a). Urethral discharge: easier to define in men than women. Design flow-chart of treatment to cover gonorrhoea first and then if no improvement chlamydia.

b). Vaginal discharge: a more nebulous group with many causes but design a flow-chart to treat gonorrhoea and trichomonas first and if no improvement, candida and chlamydia

c). Genital ulcer: treatment schedule to cover syphilis and chancroid. If no improvement check for evidence of herpes and if diagnosed give symptomatic relief.

d). Lower abdo pain: design treatment flow chart to cover major causes especially in women e.g. salpingitis and pelvic inflammatory disease (gonorrhoea and chlamydia dominate), ectopic pregnancy and other causes of peritoneal irritation such as appendicitis. Refer those who cannot be treated at the peripheral level.

Often. Good diagnostic practice with good cost-effectiveness.

Combine Always. with careful clinical assessment to distinguish STD's including Gram's staining of urethral discharge with microscopy looking for intra-cellular diplococci (gonorrhoea), microscopy of vaginal and cervical discharge looking for trichomonas, candida. RPR or VDRL for syphilis. Make treatment more specific than in Always

Seldom. Best diagnostic practice disregarding cost.

Chlamydia diagnosed by culture or DNA probe or amplified antigen detection tests (PCR) Gonorrhoea detected by culture. Syphilis identified by serologic tests: VDRL for screening and then TPHA or FTA-ABS or EIA tests as confirmatory test. Chancroid shown by culture, but difficult and expensive. Trichomoniasis and candida by direct microscopy of wet preparations.

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Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.

Chlamydia detected by newer amplified antigen tests such as ligase chain reaction test on urine. Gonorrhoea detected by enzyme-linked assays, immunofluorescence and DNA probes. Syphilis tested by EIA tests such as CAPTIA IgG, IgM or BioElisa Syphilis test. Chancroid detected by DNA probes, PCR and antigen-capture methods.

b. Poor diagnostic practice which is now obsolete. e.g. Wasserman reaction for syphilis

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Staircase diagnostics

HIV infection and AIDS *5*

As yet no good algorithms have been worked out for a clinical diagnosis of HIV. However the WHO criteria for clinical diagnosis of AIDS from Bangui can be used at the later stage.

Always. Acceptable diagnostic criteria for AIDS in adults but with low sensitivity and specificity (Bangui criteria). At least two major signs and one minor sign in the absence of other causes of immunosuppression such as cancer or severe malnutrition. Similar criteria were worked out for children see below*.

Main Symptoms Weight loss exceeding 10%; Chronic diarrhoea for more than 1 month; Chronic fever for more than 1 month

Minor Symptoms Cough for more than 1 month; Widespread itchy dermatitis; Recurring "shingles" - Herpes Zoster; Candida (fungal) infection of mouth, oesophagus; Widespread herpes simplex; Generalized lymphadenophathy (check esp. sub-occipital nodes)

Kaposi Sarcoma or Cryptococcal Meningitis are Pathognomonic for AIDS.

Often. Good diagnostic practice where a laboratory is available.

Add to above, HIV testing for *antibody* using two different rapid HIV tests such as HIVChek, SERODIA-HIV, HIV dipstick etc. Several of these can be used a number of times if the result is negative. Pre and post counselling is essential unless HIV test used only for testing donated blood. Prior to blood transfusion a single test is adequate since these tests have such high sensitivity (their specificity is almost as high).

Seldom. Best diagnostic practice disregarding cost.

Look for virus using PCR. Check for *antibody* using latest ELISA test with a follow-up of a different ELISA or Western blot. Follow the clinical course with CD4 counts and when these fall below 500 per mm^3 consider triple therapy; Follow viral load with PCR and when above 10 000 per mm^3 consider triple therapy. When CD^4 below 200 per mm^3 define as AIDS (USA). Otherwise use CDC clinical criteria.

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*Clinical case definition for AIDS in children: needs two major signs and two minor signs **Major** instead of 10% weight loss have in this defn. "Weight loss or abnormally slow growth". **Minor** signs includes: confirmed maternal HIV infection, repeated common infections (otitis, pharyngitis etc). Not included: herpes zoster or simplex. The rest of the criteria are the same.

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Staircase diagnostics

AIDS Dementia Complex 5

Invasion of the brain by HIV often affects the frontal lobe early. It needs to be distinguished from opportunistic infections of the brain some of which are treatable in the early stages at reasonable cost even when retro-virals are unaffordable. It closely mimics Alzheimer's disease but is usually at a different age. Since toxoplasmosis is the cheapest and easiest of brain invasive opportunistic infections in HIV to treat some start with empirical treatment in an acute brain syndrome even without a diagnosis.

Always. Acceptable diagnostic practice that can be taught at every level.

Suspect the diagnosis when an HIV-infected person shows any significant mental changes.

Test with three simple bed-side tests

* Short-term memory using 5 objects for 2 minutes. Then cover over and ask for patient recall
* Fine motor function - pick up 2 pins on a flat surface
* coordination (cerebellum) - tap the table quickly

Often. Good diagnostic practice where clinical skills are available.

* **Earliest changes**: damage to the frontal lobe causing "Frontal Lobe Release". Test with:
  * "snout" reflex: the mouth forms a snout when the lips are lightly touched or tapped.
  * palmo-mental reflex: light stroking of the palm of the hand causes muscle twitching on the same side of the lower face.
* 10% have paresis of one of the cranial nerves III, IV, VI, VII.

L.P. to look for Cryptococcus neoformans with indian ink staining of CSF.

CMV invasion may show as "white cottage cheese with ketchup" appearance of retina i.e. soft exudates with bleeding. Treatment extremely costly.

Toxoplasmosis often presents with fits.

Seldom. Best diagnostic practice disregarding cost.

MRI to look for evidence of toxoplasmosis: multiple brain abscesses showing as ring-enhancing lesions. Check for toxoplasma antibody. If negative, toxoplasmosis of brain less likely.

Lymphoma of brain looked for with CT scan.

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Staircase diagnostics

Pulmonary Tuberculosis in Childhood *5*

In areas where both malnutrition and TB are common establishing the diagnosis of active TB in a malnourished child is extremely difficult. In such areas up to 20% of all severely malnourished children have active TB and in the older malnourished child (over 3-4 years) the majority have active TB unless famine or severe food shortage is the underlying factor. In the same areas 90% of children with active TB are malnourished. The normal diagnostic pathways are unattainable. All cases need assessment at the level of first referral hospital or above.

Always. Acceptable diagnostic practice that is cheap where Xrays are unavailable.

Treatment flow-chart. Good nutrition rehabilitation of all malnourished children, accompanied by antibiotic therapy in severe malnutrition. If no response within 3 weeks start TB treatment using 2-3 drugs with specific TB effect (INAH, Pyrazinamide, ethambutol). If no response after 2 further weeks, check for HIV.

Often. Good diagnostic practice with good cost effectiveness.

Chest Xray often gives a pointer to the diagnosis but is more difficult to assess that in an adult. Use BCG as diagnostic tool looking for rapid response. Give standard intra-cutaneous BCG for that age. If reaction shows >10mm within 4-6 days TB is likely; if more than 15 mm almost certainly TB.

Alternatively use **points scoring system** see appendix 1.

Seldom. Best diagnostic practice disregarding cost.

Where TB culture is available use gastric washings or a laryngeal swab or bronchoscopy with culture of material obtained. Complement this with PCR enhancement. Careful assessment of Chest Xrays with tomography where available. In a non-malnourished child use Mantoux test. Persistently asymmetrically enlarged lymph nodes can be biopsied and examined histologically.

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Staircase diagnostics

Pulmonary tuberculosis in adolescents and adults *5*

Caused by Mycobacterium tuberculosis, M. bovis or M. africanum. TB causes 9 million new cases of illness per year and 3 million deaths, of whom half a million are children. Developing laboratory facilities to stain and assess sputum for AAFB is highly cost-effective in breaking the infection cycle since each undiagnosed and untreated case of open pulmonary TB will infect 10 others each year.

Always. Clinical criteria which make diagnosis likely without laboratory facilities

Early TB may be without symptoms. When symptoms occur, a history of contact with someone with open pulmonary TB is a strong pointer. Once active TB illness supervenes the typical features are long-standing cough (more than 3 weeks) usually with mucoid, purulent or blood-stained sputum, loss of weight, fever at first in the evening and then more generally, night sweats, malaise. Check the mid-upper-arm-circumference (MUAC) which may show wasting.* Clinical assessment may show asymmetry of chest wall and its movement, enlarged asymmetrical lymphadenopathy especially in the supra-clavicular fossa, stony dulness to percussion if there is pleural effusion, with absent tactile fremitus. Crepitations and bronchial breathing especially over the upper lobes. Tachycardia out of proportion to fever is common. However no diagnosis made on clinical findings alone.

Often. Good diagnostic practice where a laboratory is available without culture.

Ziehl-Neelsen staining of at least 2 morning sputum samples** for AAFB if possible using household bleach to enhance yield: see Appendix 10 and ref. below. Diagnosis by ZN staining alone has high specificity but low sensitivity. In those positive, follow-up sputum for AAFB after 2 months treatment and then at 6 months. Chest Xray may help in diagnosis of sputum negative patients. A normal Xray virtually rules out pulmonary TB. Signs of active PTB on Chest Xray: cavity, soft shadows, extensive shadows, progression of shadows on serial follow-ups.

Seldom. Best diagnostic practice disregarding cost.

Culture the sputum in liquid media e.g. BACTEC and use nucleic acid probes or PCR to get a rapid diagnosis while waiting for the culture. Tomography to visualize small lesions. Pleural biopsy in pleural effusions. Bronchoscopy and laryngeal swabs to get specimens for PCR and culture when other tests fail. Use of firefly luciferase reporter mycobacteriophage assays.

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* Three categories of malnutrition from MUAC:

	Undernourished		Severe wasting		Extreme wasting	
	Male	Female	Male	Female	Male	Female
	<230	<220	<200	<190	<170	<160 mm

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Staircase diagnostics

Suspected typhoid and paratyphoid fever *5*

The enteric fevers are caused by Salmonella typhi and paratyphi but even by other invasive Salmonella species and occasionally by Yersinia and Campylobacter species. All of these give prolonged fever i.e. more than a week. Most of the information below relates to invasive Salmonella and especially S. typhi. There are an estimated 33 million cases of typhoid globally each year with half a million deaths. See also Appendix 8 Diagnosing acute and chronic fevers.

Always. Acceptable diagnostic criteria for clinical diagnosis when laboratory unavailable (a careful clinician makes the right diagnosis in 80%).

Prolonged fever but without rigors, with a slow onset rising stepwise to a plateau around 39-40° after one week accompanied by headache and abdominal pain (esp in the ileal region). In whites look for rose spots (pink sparce maculopapular lesion on the abdominal wall fading on pressure with a glass slide) at the end of the first week of fever. More likely in typhoid fever than in the other enteric fevers . Cough and chest signs with a normal chest Xray is supportive evidence. Conjunctivitis seen in a few. Relative bradycardia in the first week, splenomegaly and hepatomegaly. At the onset of the third week the patient looks more toxic with mental dullness or a delirious confusional state. ***Features against*** the diagnosis: herpes labialis, lymphadenopathy.

One recent study suggests that ***the most powerful clinical predictors*** of typhoid are a stepladder pattern of rise of temperature up to at least 39°, loose motions, relative bradycardia and coated tongue. Moderately powerful predictors were headache, splenomegaly and hepatomegaly. Most had more than one week of fever. Commonest age affected: young adults and older children.

Often. Good diagnostic practice where a laboratory is available.

Add to above, white cell count and differential where leucopenia after initial leucocytosis favours the diagnosis, with WBC < $9x10^6$/L and polymorphs<$3.5x10^6$/L. N.B. majority have WCC in the normal range. Absence of eosinophils in an area with many helminths is strongly suggestive. Blood culture (43%+) in the first week or bone marrow culture (95%+) at any stage are the best methods of establishing the diagnosis. Widal test with rising antibodies against O-antigen in the early stages or against H-antigens later is suggestive but many false positives occur. Stool and urine cultures are often negative. Slide typhoid agglutination test in one study was 98% sensitive for typhoid.

Seldom. Best diagnostic practice disregarding cost.

ELISA test for antibodies against S.typhi somatic antigen can be a useful addition to bone marrow culture which is the gold standard for diagnosis.

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Staircase diagnostics

Suspected urinary tract infection 5

There are only two primary pathogenic organisms in the urinary tract: E. coli and Staph. saprophyticus. All the other pathogens occur predominantly in those with predisposing factors with the commonest being Klebsiella spp., Enterobacter spp., Proteus mirabilis and Enterococcus spp. There is a clear distinction between uncomplicated and complicated UTI. Complicated includes all men, those with an abnormal urinary tract, those with depressed immune response including Diabetes mellitus and those with impaired renal function. Upper and Lower UTI present differently.

Always. Acceptable diagnostic criteria for clinical diagnosis when laboratory unavailable . ***Look at the urine with good light: if it looks clear it probably is clear.***

Children 0-5 yrs. Ask about offensive urine, pain at micturition, fever, haematuria and abdominal or flank pain. In upper UTI may show vomiting, high fever and is ill-looking.

Children over 5 yrs and adults: symptoms and localization of pain often more specific i.e. over suprapubic are in lower UTI and over flanks in upper UTI. In old people may only be confusion.

Examine for temp., tenderness in lower abdo or flank. Check urine with nitrite urine strip. Especially useful in morning urine, if the urine has been incubating for at least 4 hrs. Specificity almost 100%. Sensitivity 50-80%. Negative with Staph. saprophyticus and Enterococcus spp.

Often. Good diagnostic practice where a laboratory is available.

In uncomplicated UTI in women and older girls no further test are needed and response to short-course therapy is the next step. In others add to above, leucocyte esterase test on urine if nitrite test is negative. Sensitivity 73-99% Specificity 91-100%. May be falsely positive if child has a concomitant respiratory infection. Microscopic examination by an experienced microscopist of a mid-stream urine specimen after careful cleaning of the genitalia. Clear excess of leucocytes (>10 leucocytes/mm^3) is strong evidence of UTI. Add CRP, white cell count (count above $10x10^9$ is significant) and differential if available. CRP raised above 20 mg/l in upper UTI in children and above 50 mg/l in adults. It is normal in lower UTI. Dipslide of mid-stream urine is of use in adults (less useful in children) where nitrite test is negative but only useful in E. coli infections.

Seldom. Best diagnostic practice disregarding cost.

If culture of urine is possible only use this with complicated (including small children, all men, nosocomial UTI and pregnant women), recurrent or suspected upper UTI . Take specimen from mid-stream morning urine that has been incubating at least 4 hours. Only a count above 100 000 cfu/ml is significant unless symptoms are convincing. Many advocate suprapubic puncture if possible with a full bladder for urine culture especially in doubful UTI in small children since any growth is significant and mixed growth is unlikely. Blood culture of value in suspected upper UTI. **In sterile pyuria remember possiblity of TB using TB culture and/or PCR amplifying methods.**

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Staircase diagnostics

Suspected malaria fever *5*

In Africa this is the biggest cause of mortality and morbidity in children under 5 years ·

Always. Acceptable diagnostic practice that is cheap but less dependable **where microscopy is unavailable.**

In a child ***between 1-9 years living in a holoendemic malaria area with fever for at least 2 days,*** use of the two questions formulated by Rooth (less useful in other malaria areas e.g. hyperendemic, mesoendemic and hypoendemic but history of intermittent fever still highly sensitive but not very specific):

a. Has your child at any stage in this febrile illness been completely fever free? If the answer is yes, 98% will be suffering from clinical malaria and with this question 78% of the children with malaria will be identified.

b. Has your child got any symptoms suggestive of a viral illness (specify e.g. coryza or rash of measles)? If the answer to this question is yes, more than 90% will **not** be suffering from clinical malaria. See also Appendix 8 diagnosing acute and chronic fevers.

Often. Good diagnostic practice with good cost-effectiveness.

Combine *Always* with good thick and thin blood slides, well stained and accurately examined being aware that in a semi-immune population in a holo-endemic area there is a cut-off level below which presence of parasites is usually irrelevant to the present illness (400 parasites/µL i.e. approximately 5 parasites per 100 white blood cells). Quality assurance of microscopy is important to avoid both false negatives and false positives.

Seldom. Best diagnostic practice disregarding cost.

Combine steps 1 and 2 with use of *Para*Sight-F (only for falciparum parasites) and/or PCR and/or QBC malaria test (cannot distinguish between malaria species) and/or test for antigen HRP-2 (Malaquick) in a doubtful case of plasmodium falciparum in a non-immune. These may have a place where reliability of the blood films is in question but in the long run improving the diagnosis by blood films is more important than relying on expensive extras.

Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.

Cheaper versions of *Para*Sight-F

b. Poor diagnostic practice which is now obsolete.

Depending on a single blood slide for excluding suspected malaria in a non-immune on prophylaxis

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Staircase diagnostics

Distinguishing between Cerebral malaria (CM) and Meningitis in comatose children 5

Main CM problem in hyperendemic and holoendemic malaria areas is in children aged 2-6years. Can virtually be ruled out in any child over 6 years or adult who has lived continuously in such an area. In people living in areas with unstable malaria or in other non-immune people, this is a problem that occurs at any age.

Always. Acceptable diagnostic practice that can be taught at every level.

Those with a short history of fever up to 3 days likely to be CM. Those with significant neck stiffness and fontanelle fulness likely to be meningitis. Both frequently have had convulsions. Those with abnormal eye signs (divergent squint, dysconjugate eye movements), extensor posturing or opisthotonus, hyperventilation and absent abdominal reflexes likely to be CM. Jaw jerk brisk, plantar reflexes often extensor, pout reflex usually present, mild neck stiffness common; neck rigidity never seen in CM. Blood slide more likely to show plentiful parasites in CM but in rare cases a negative slide does not rule it out. It needs to be repeated if CM suspected. Meningitis cases may also have parasites. High white cell count likely to favour meningitis.

Often. Good diagnostic practice where clinical skills are available.

L.P. to examine CSF. No CM has more than 10 cells/mm. No meningitis has less than 50 cells/mm. If no lab available can test CSF with Combur9 urine reagent strips. If cells are positive in the lowest range this gives a doubtful diagnosis. Higher WBC confirms meningitis. Follow all cases with Blantyre or Glasgow coma scale (see appendix 2) which gives prognostic rating. CM with deep coma, absent corneal reflexes, recurrent seizures, hyperventilation, extensor posturing all associated with poor outcome.

Seldom. Best diagnostic practice disregarding cost.

Check for lactic acidosis which shows a poor prognosis in CM .

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Staircase diagnostics

Leprosy 5

Caused by Mycobacterium leprae. There are an estimated 2 million cases in the world of whom just under 1 million are registered. The annual incidence is 500 000/yr. Much of the dramatic improvement in leprosy control started in 1982 with the introduction of Multi-Drug Therapy.

Always. Clinical criteria which make diagnosis almost certain even in the absence of a laboratory (look carefully in daylight, palpate the nerves and test sensation with a wisp of cotton wool and a pin)

Very early *indeterminate leprosy* shows as a skin lesion which has lost the ability to sweat. These patients may complain of nasal stuffiness and oedema of the feet.

Later stages depend on the body's immune response.

With good immune response, *TT leprosy* shows as asymmetrical anaesthetic skin lesions without hair with enlarged often tender nerves especially over the cooler parts e.g. ulnar, superficial radial, median, great auricular, lateral popliteal and posterior tibial.

Poor immune response *LL leprosy* shows as small symmetrical skin lesions with numerous infiltrates and in the early stages normal sensation. At a later stage the nerves are often symmetrically enlarged and "glove and stocking" anaesthesia develops.

In between these extremes are *borderline BB* cases and even *BT* and *BL* with intermediate characteristics.

Often. Good diagnostic practice where a laboratory is available.

Add to the above the following: in any suspected leprosy patient and especially where LL, BL and BB leprosy is suspected take skin smears from the lobes of the ear and the edge of any skin lesion and scrapings from the nose. Stain with Ziehl-Neelsen method. Some value of lepromin test to distinguish good immune response (positive reaction to injected leprosy protein) and poor immune response (no reaction).

Seldom. Best diagnostic practice disregarding cost.

Add to the above the following: inject suspected material with bacilli into the 9-banded armadillo or the foot pads of immunologically deficient mice to check the diagnosis when this is in doubt and in order to study drug resistance patterns where this is suspected.

Search for M-leprae using gene amplification; combined ethidium-bromide staining and probe hybridization

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Staircase diagnostics

Suspected urinary schistosomiasis *5*

Caused by Schistosoma haematobium and endemic in certain countries with inadequate sanitation and contact with water where snails of Bulinus genus reside. Man is the only definitive host.

Always. Acceptable diagnostic practice that is cheap but less dependable **where microscopy is unavailable.**

An indirect method of diagnosis using a history of haematuria, visual haematuria or microhaematuria at the 1+ and 2+ positivity limit by reagent strips. Microhaematuria is the most useful in an endemic area, with very high sensitivity and specificity. Useful both in preliminary screening of communities and even individuals in a community programme esp. in children.

Often. Good diagnostic practice with good cost-effectiveness.

Combine *Always* with microscopy of sedimented or centrifuged urine specimen preferably passed between 12 noon and 14.00 hrs. on 3 occasions. Quantitative estimates can be made passing 10 ml aliquot of urine through nucleopore filter and counting the eggs trapped. Rectal biopsy of value even in suspected S. haematobium infestation (use rectoscope and curette, pulling mucosa over end of rectoscope and cutting a small piece with curette). In males can look for schistosomes in semen as a non-invasive test.

Seldom. Best diagnostic practice disregarding cost.

Combine steps 1 and 2 with addition where doubt still exists of enzyme immunoassay using crude-egg antigen extract (SEA) or a purified component (CEF6).

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Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.

Enzyme immunoassay using circulating anodic antigen (CAA) or circulating cathodic antigen (CCA)

b. Poor diagnostic practice which is now obsolete.

Depending on a single urine specimen alone to exclude the diagnosis.
Insisting on exercise before passing the specimen

c. Contra-indicated diagnostic practice.

Relying on the earlier enzyme immunoassays to be a marker of current infection in someone who has been treated for a previous infection.

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Staircase diagnostics

Diabetes mellitus *6*

This is likely to become a major cause of ill-health in the future as more affluence comes to certain sections of communities in many countries. In some communities e.g. those of Polynesian-Micronesian stock 40-50% of some groups over 50 yrs. have abnormal glucose tolerance tests.

***Insulin Dependent Diabetes Mellitus (IDDM)** definition: any symptomatic patient with a random plasma glucose of 11.1 mmol/L (200 mg/dl) or a fasting plasma glucose >6.7 mmol/L and/or a 2 hour postglucose level >10.0 mmol/L. IDDM defined as any patient who without insulin injection becomes ketoacidotic within days. Usually in younger people. More common in white Caucasians.*

***Non-Insulin Dependent Diabetes Mellitus (NIDDM)** definition: any patient with a fasting plasma glucose >6.7 mmol/L ((120 mg/dl) and/or a 2 hour postglucose level >10.0 mmol/L (180 mg/dl).*

Malnutrition-related diabetes mellitus (MRDM) is seen in tropical countries and shares features of the above two types.

Always. Simple diagnostic practice where a laboratory is not available.

Suspect the diagnosis in any person with a history of polyuria, polydypsia and, in many younger diabetics, weight loss. There is often fatigue and ill-health and susceptibility to infections. They may have noticed fungal infection of the vagina or glans penis. They may even have noticed how ants are attracted to any drop of their urine on the ground. In IDDM, acetone may be smelt on their breath. This may worsen to diabetic coma where the person becomes drowsy and dehydrated sometimes with abdominal pain and deep sighing breathing. The pulse is often weak and rapid. Without treatment this may go on to deep coma and death.

Check the urine for sugar using any of the test strips that measure glucose. Double the use of these strips by cutting them in half longitudinally. If available check blood glucose using dextrostix.

Often. Good diagnostic practice where a laboratory is available.

In anyone found to have glucose in the urine, check the fasting plasma glucose. If this is normal consider a Glucose Tolerance Test (if glucose continues to be found in the urine) and specifically the plasma glucose level 2 hours after ingestion of 75gms glucose.

Follow the progress of IDDM by checking twice daily blood glucose levels aiming at fasting < 8 mmol/L, and 2 hours postprandial < 10 mmol/L and if available monthly Glycated haemoglobin GHb within 4 S.D. of normal. Once stable levels are achieved, follow less often.

Seldom. Best diagnostic practice disregarding cost.

As above but refine follow-up by 4 daily checks of self-monitored blood glucose aiming at preprandial levels of 3.9-6.7 mmol/l and a normal glycated haemoglobin (GHb) concentration until a stable control is achieved.

N.B. 1. Viral and bacterial infections can cause glucose insensitivity and so diagnostic tests for diabetes should not be carried out during or just after infections.

2. Whole-blood glucose values (as measured by dextrostix) are 10-15% lower than plasma glucose levels, and capillary levels are 7-8% higher than venous blood levels.

3. Glucose levels in the SI units compare with conventional units as follows: 4-6mmo/L =72-108mg/dl

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Staircase diagnostics

Ante-natal monitoring 7

Much of monitoring of pregnant mother in the ante-natal period has come about by tradition and is now being questioned. More specific monitoring with less visits but making these more accurate is the goal. See also Appendix 5 Ante-natal clinics.

Always. Acceptable diagnostic practice that can be taught at every level.

4-6 visits during the ante-natal period with high quality measurements is adequate.

Obstetric history: ask about risk factors. Check height. <152 cms is a risk factor.

Blood Pressure. Urine for protein (split the strips to get twice value) and glucose. Check for pallor of palms and soles and mucous membranes.

Check symphysis-fundus length after emptying the bladder to look for foetal growth retardation or twins/hydramnios.

Often. Good diagnostic practice where clinical skills are available.

Add to above: Check Hb. Check for syphilis with RPR or VDRL screening tests.

Seldom. Best diagnostic practice disregarding cost.

Check growth of baby, position, any sign of abnormalities and normal position of placenta with ultra-sound.

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Staircase diagnostics

Monitoring Labour 7

Three of the main causes of maternal mortality will be reduced if all labours are kept short (bleeding post-partum, pueperal sepsis and obstructed labour). Accurate monitoring of the progress of labour is central to initiating early any interventions to speed up labour and thereby to reduce the Caesarean section rate as well as avoiding the above three complications. This makes the diagnosis of abnormal labour early.

Always. Acceptable diagnostic practice that can be taught at every level.

Simplified partogram only following established labour in the active phase (from when the cervix is 3 cms dilated and fully effaced in a primip). A simpler partogram need only have the cervicogram, foetal heart rate pattern monitored with a foetal stethoscope, and assessment of downward progress of the presenting part (if this is the head, measuring it in fifths above the pelvic brim). Use of alert line and action line to initiate action early when labour is abnormal especially in a primip. See appendix 4.

Often. Good diagnostic practice where clinical skills are available.

Full partogram following even the latent phase of labour together with monitoring of contractions, pulse, BP, state of liquor, temp. (see appendix 3)

Seldom. Best diagnostic practice disregarding cost.

Complementing monitoring of high risk babies with electronic monitoring of foetal heart sounds and even scalp blood sampling. However this may increase the number of unnecessary Caesarean sections.

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Staircase diagnostics

Anaemia *9*

Establishing an accurate haemoglobin and haematocrit are the cornerstone of diagnosis. Globally the main causes of anaemia are iron deficiency (due to lack in diet, abnormal loss of blood e.g. heavy hookworm infestation), haemolytic anaemia (due to malaria or an abnormal Hb such as HbS), and folate deficiency in malnourished children and in pregnancy (suspected when a blood film shows macrocytosis and a shift to the right of neutrophils).

Always. Acceptable diagnostic practice that is cheap but less dependable.

Combining clinical impression from examination of palms, conjunctival mucous membranes and tongue with Hb estimation using a BMS Haemoglobinometer (sensitivity of 78% and specificity of 96% when used accurately). No electricity or chemicals needed. Saponin sticks for haemolysis of sample can be made locally very cheaply from match-sticks and a tiny smear of ordinary soap. Long battery life but instrument and its slides need careful cleaning and storage when needed. Skill of getting estimation accurate takes time to develop.

Often. Good diagnostic practice with good cost-effectiveness.

Haemoglobin assessed using photometry colorimetry. Needs electricity (unless a batery operated unit is available) and chemicals (sensitivity approaches 100% and specificity >90%). Combine with haematocrit using microhaematocrit tube and centrifuge and a careful examination of a blood film to look at the red blood cells (see introduction).

Seldom. Best diagnostic practice disregarding cost.

Hb. estimation for field work using a HemoCue haemoglobinometer (can be battery operated). (sensitivity approaches 100% specificity 94%). Laboratory estimation using a Coulter counter. Haematocrit estimation as above in Often. Serum folate levels when indicated.

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Not recommended diagnostic procedure

a. Diagnostic investigation that is being developed.

Cheaper renewables with HemoCue Hb.meter

b. Poor diagnostic practice which is now obsolete.

Clinical impression alone or estimation using Talqvist filter paper.

Copper sulphate drop method other than for screening procedures e.g. prior to large scale blood donations to screen out anaemic subjects.

c. Contra-indicated diagnostic practice.

Prolonged squeezing of finger or earlobe when extracting drop for Hb. estimation.

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Topley, E. (1984) Haematological services in developing countries. Medical laboratory Sciences, 41, 389-399

Staircase diagnostics

Blood Grouping and Cross-matching *9*

Restricted use of blood transfusion because of HIV and Hepatitis risks is strongly recommended. Have written policy for hospital. Testing for HIV and Hepatitis B is obligatory if risks are to be avoided.

Always. Acceptable diagnostic practice that is cheap.

Where laboratory facilities are unavailable (such as at night or at the weekend in smaller rural hospitals) in an emergency consider use of Eldon cards for ABO+ rhesus blood grouping, and similar rapid HIV/Hepatitis B testing. Blood grouping alone will give 98% safety if the right group is transfused even without cross-matching. If there is any chance of doing a quick cross-match this adds marginally to the safety. Otherwise careful supervision during transfusion looking for significant reactions.

Often. Good diagnostic practice where clinical skills are available.

Laboratory based ABO+ rhesus grouping, HIV/Hepatitis B testing and cross-matching in a standard way. May add RPR for syphilis screening.

Seldom. Best diagnostic practice disregarding cost.

Add testing for Hepatitis C where this occurs and even syphilis screening test such a VDRL.

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Staircase diagnostics

Rheumatoid arthritis *10*

Prevalence is around 1% in most parts of the world. More common in females than males 3:1. Some races less affected e.g. rural Africans much less affected, certain North American Indian tribes more affected e.g. Chippewa and Yakina. Diagnosis based on 4 out of 7 criteria of which the clinical features must have been present for at least 6 weeks. In 10% of those affected, the disease starts with explosive speed. The disease is characterized by exacerbations and remissions.

Always. Acceptable diagnostic practice that is cheap without lab facilities.

Diagnosis based on at least 4 ot the 7 following criteria: morning stiffness at least 1 hour, swelling of at least 3 joints, swelling of the wrist, m-p or proximal i-p joints, symmetrical swelling, typical Xray changes in the hand-joints, subcutaneous nodules, positive RA-factor. The first 4 of these must have been present for at least 6 weeks.

N.B. Two out of the seven criteria cannot be tested for at this level.

Often. Good diagnostic practice where clinical skills are available.

Look for typical Xray changes in the hand-joints

Check for positive RA-factor in blood test.

Seldom. Best diagnostic practice disregarding cost.

Check for HLA-Dw4 or HLA-Dw14 which are positively associated with the disease (Dw-10 and Dw-13 are negatively associated).

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References

Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. Oxford University Press. Oxford.

Staircase diagnostics

Osteoarthritis *10*

OA is a disease process of synovial joints characterized by focal areas of loss of hyaline articular cartilage, associated with increased activity in marginal and subchondral bone. It is symptomatic three times more often in women and the mean age of onset is 50 years.

Always. Acceptable diagnostic practice that is cheap.

Looking for the typical features i.e. pain on movement worse at the end of the day; background pain at rest; stiffness; joint instability. Most commonly affected joints are DIP joints, 1st metacarpophalageal joints, 1st metatarsaophalageal cervical and lumbar spine, next the hip, then knee.

Often. Good diagnostic practice where diagnostics are available.

Xray of the affected joint looking for asymmetrical narrowing of the joint space, increased density of the bone next to the articular surface (subchondral bone sclerosis), marginal osteophytes and subchondral cyst formation. Blood tests to rule out metabolic disease.

Seldom. Best diagnostic practice disregarding cost.

Bone scintigraphy which in more severe disease is abnormal. Raised C-reactive protein and hyaluronate are markers of more severe disease.

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Staircase diagnostics

Gout *10*

Gout is the main example of crystal arthropathy where due to hyperuricaemia, crystals of sodium monourate are deposited in joints and cause inflammation. The attacks may be precipitated by trauma, surgery, starvation, infection and diuretics. Probably 95% of hyperuricaemic subjects remain asymptomatic throughout their lives.

Always. Simple diagnostic practice.

If the patient has a classical history of podagra with an acute exquisitely painful metatarsophalageal joint of the big toe, the diagnosis is clear especially if this responds dramatically to colchicine or to an NSAID such as naproxen.

Often. Good diagnostic practice where investigative facilities are available.

Check the fasting serum uric acid level twice. If this is more than the reference level for the sex and age it confirms the diagnosis*. The true diagnosis is however made on aspiration of the inflamed joint and finding the strongly birefrigent needle-shaped crystals 5-20μm in length within cells or freely in fluid. There are markedly increased numbers of neutrophils in the turbid fluid. This aspiration may often give dramatic relief by reducing the pressure in the inflamed joint.

Seldom. Best diagnostic practice disregarding cost.

In young overproducers check for a purine enzyme defect. Check renal function.

*In males the normal range is between 210-480 μmol/L (3.5-8 mg/dl) depending upon age.
In females the normal range is between 150-390 μmol/L (2.5-6.5 mg/dl) depending upon age.

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Staircase diagnostics

The Red Eye *11*

There are 3 common groups of causes of red, painful or irritating eyes: infections of various kinds, allergy and injury. There are three important but rare conditions: acute glaucoma, iritis and episcleritis all of which usually cause severe pain and loss of vision.

Always. Acceptable diagnostic practice that is cheap.

Where ophthalmology services are unavailable base diagnosis on history and examination: Four questions are central:

a) Any injury or f.b. in eye?

b) When did the symptoms start? Acute recent onset likely to be acute viral or bacterial infections.

c) Is there any loss of vision? Likely to be a sign of more serious disease e.g. corneal damage, glaucoma or iritis.

d) What other symptoms? e.g. URTI or measles.

A red eye where the redness comes in from the outer edge and involving the inside of the eyelids is likely to be a conjunctivitis either viral or bacterial. If the discharge is purulent it is likely to be bacterial.

A chronic red eye is likely to be trachoma or allergy. If accompanied by itching it is more likely to be allergy. If on the inside of the eyelid there are many small swellings it is almost certainly allergic. If there are vessels running across the inside of the eyelid it could be trachoma.

Often. Good diagnostic practice where clinical skills are available.

Add to above, culture of any purulent discharging red eye to identify the organism and its antibiotic sensitivity. Tonometry for any eye where raised pressure may be a possibility. Any corneal damage further examined using fluorescein to look for a corneal ulcer. This may show a dendritic ulcer caused by herpes simplex.

Seldom. Best diagnostic practice disregarding cost.

Examination of eye where diagnosis is obscure by a ophthalmologist with ophthalmological microscope and other special instruments.

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References

Sandford-Smith J. Eye Diseases in Hot Climates. Wright. Bristol 1986

Appendix 1

Clinical points system for paediatric pulmonary TB	Points
Cough for 4 weeks (excluding whooping cough)	1
"Pneumonia" not improving after 3 weeks treatment	1
Unexplained fever more than 3 weeks	1
Malnutrition rehabilitated with food as outpatient - no change	1
Malnutrition rehabilitated with food as inpatient - no change	2
Onset of PEM after age of 3 years	3
Onset of PEM after age of 4 years	5
Measles followed by prolonged ill-health for 2 months	2
Family member sputum positive for AAFB	4
Cervical glands enlarged, rubbery, painless	4

Interpretation of clinical points system

1 or 2 points - further control after 1 month

3-4 points - refer if possible for further investigation

5 - start TB treatment.

Appendix 2 Glasgow Coma scale

		Score
Eyes open:	spontaneously	4
	to speech	3
	to pain	2
	never	1
Best verbal response:	oriented	5
	confused	4
	inappropriate words	3
	incomprehensible sounds	2
	none	1
Best motor response:	obeys commands	6
	localizes pain	5
	flexion to pain:	
	withdrawal	4
	abnormal	3
	extension to pain	2
	none	1
	Total	3–15

This score has been modified to be applicable to children, including those who have not learned to speak.

Eye movements:	directed (e.g. follows mother's face)	1
	not directed	0
Verbal response:	appropriate cry	2
	moan or inappropriate cry	1
	none	0
Best motor response:	localizes painful stimulus[a]	2
	withdraws limb from pain[b]	1
	nonspecific or absent response	0
	Total	0–5

These scales can be used repeatedly to assess improvement or deterioration.

[a] Rub knuckles on patient's sternum.
[b] Firm pressure on thumbnail bed with horizontal pencil.

Blantyre Coma scale

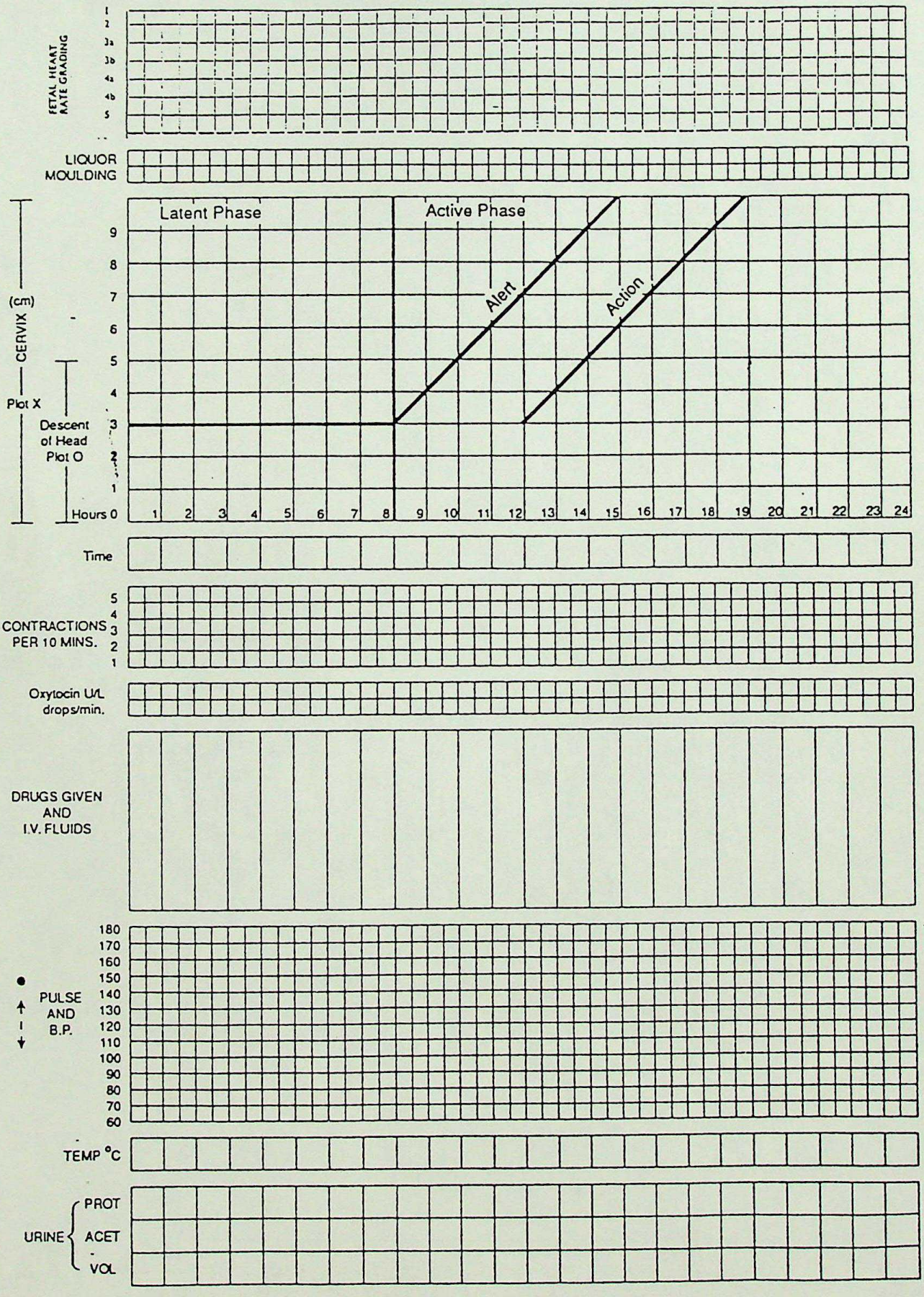
Response	Score
Best motor response	
Localizes painful stimulus (pressure with blunt end of pencil on sternum or supraorbital ridge)	2
Withdraws limb from painful stimulus (pressure with horizontal pencil on nail bed of finger or toe)	1
No response or inappropriate response	0
Best verbal response	
Cries appropriately with painful stimulus or, if verbal, speaks	2
Moan or abnormal cry with painful stimulus	1
No vocal response to painful stimulus	0
Eye movement	
Watches or follows (e.g., mother's face)	1
Fails to watch or follow	0

NOTE. Data are from [13]. The Blantyre coma score is calculated by adding the scores for each response (maximum score = 5; minimum score = 0).

Appendix 3 WHO Partogram

Name ..... Gravida ..... Para ..... Hospital No. .....

Date of admission..... Time of admission ..... Ruptured membranes ..... hrs.



Proposals for a simpler Partogram

1. Discard the latent phase on the partogram and start only when the labour has entered its active phase i.e. 3 cms dilated cervix, fully effaced in a primiparous patient (same criteria in multipara but must train staff to assess the cervix accurately without dilating it up more than it is already dilated). Make this start at time 0 (this is going back to the original partogram of Philpott).

2. Easier assessment of foetal heart rate pattern than Philpott has in his 5 grades and two sub-grade levels. Have only 3 grades:

a. Foetal heart counted *immediately after the contraction.* The rate is more than 100/min. and any slowing of the heart rate returns to normal within 30 seconds. This is reckoned as a normal heart rate.

b. Foetal heart counted *immediately after the contraction.* The rate is over 100 but there is slowing immediately after each contraction that takes longer than 30 seconds to return to normal. This is abnormal and intervention is necessary to relieve foetal distress.

c. Foetal heart is continuously under 100/min. This is a sign of severe foetal distress which if not relieved very quickly could cause permanent damage to the baby.

3. Only measure the blood pressure every 4 hours just prior to each vaginal assessment.

4. Discard trying to assess the level of the head except for those who have gone over the action line and then do this assessment with the most experienced member of the team. Alternatively try to get a simpler method of assessing the level of the head. (Doing it in the traditional way with relation to the spines is very misleading in Africa with a lot of cephalo-pelvic disproportion and moulding of the head).

5. Discard assessment of the number and strength of the contractions until the partogram has gone over the action line and then do this in relation to stimulation with oxytocin stimulation.

6. Every labour ward to have a board with cut out rings of diameters from 1 to 9 cms so that every vaginal assessment of cervix dilatation is followed by a comparison with the board. No partogram should be introduced until this is in place. No member of the team should do an assessment without this comparison, in order to get the best possible accuracy.

Appendix 4

Ante-natal clinics

It is important that these clinics base their work on the best evidence available of what are the important things to measure and the important treatment to give. In new studies from Zimbabwe it has been shown that 4-6 visits during the ante-natal period with high quality measurements is as good as many more visits.

1. Get a good obstetric history. Asking about risk factors such as previous stillbirths, Caesarean sections or instrumental deliveries, post-partum or ante-partum haemorrhage, prolonged labour, hypertension in pregnancy. Check the height of the woman. If she is under 152 cms and especially if she is under 146 cms. this may be a risk factor.

2. Blood Pressure. This must be taught and retaught with quality control to make the measurement as accurate as possible. A Blood Pressure of 140/90 mm or above especially in the latter half of pregnancy is significant and needs follow up. If the systolic pressure rises more than 30mm or the diastolic pressure more than 15 mm after the 20th week this is indicative of pregnancy induced hypertension.

3. Testing of urine for protein. This is probably only needed in someone with a raised blood pressure. You can split the strips lengthwise to get twice their value. Schistosomiasis often gives proteinuria and confuses the picture. Check for sugar in urine.

4. Check the Haemoglobin. This must be done in the best way that is possible with the funds and laboratory facilities available. Hemocue is best but very expensive in purchasing price and in running costs. BMS is cheap but needs a lot of training and motivation to make it accurate. However all staff should be trained in assessing pallor of the conjunctiva, palms and tongue even if Hb measurements are done.

5. Check the Symphysis-Fundus height after the bladder has been emptied. This is mainly of value if there is an accurate history of the last menstrual period in which case the S-F height can be compared with the normal. However following the S-F height is also of value. Even a single measurement at the commencement of labour is of value since it gives some idea of the size of the baby. In a baby with longitudinal lie an S-F height of 30-38 cms estimates a baby of between 2.5-4 kg. unless there are twins or there is hydramnios.

6. Supplement with iron and folic acid especially in communities with a high anaemia prevalence. If the prevalence of hookworm is high, check the stool for hookworm. If found give an anti-helminthic that is safe in pregnancy e.g. pyrantel.

Iron supplementation: new evidence shows that **one tablet once or twice weekly** is virtually as good as three tablets a day with vastly better compliance. Only in rare cases with severe iron deficiency anaemia is it worth giving more or alternately total-dose infusion of iron. The dose of folic acid is too high. 1 mg daily is enough but since all the tablets are 5 mg a compromise is giving 1/2 tablet (i.e. 2.5mg) daily.

7. In a community with high prevalence of STDs check for syphilis with RPR or VDRL.

Appendix 5

Abdominal Pain

The most important assessment is to decide how ill the patient appears and whether immediate operation or medical treatment or referral is indicated. Most patients with abdominal pain have some temporary complaint that will get better on its own provided they get encouragement and support and sometimes symptomatic relief. Thus constipation, minor indigestion, the pain related to menstruation or ovulation are limited in their duration and threat. However certain causes of abdominal pain are significant and can even be life-threatening.

Thus in children with abdominal pain two treatable causes must be sought urgently: pneumonia presenting in an unusual way and urinary tract infection, usually pyelonephritis.

With a patient who presents with abdominal pain and looks ill the following causes should be sought:

The commonest causes (95%) of an acute abdominal crisis

peritonitis from salpingitis (usually cervical excitation tenderness and often rebound tenderness in the lower abdomen. May be purulent vaginal discharge)

ruptured ectopic pregnancy (generalized rebound tenderness, pallor, sometimes shoulder tip pain, free blood on abdo aspiration and often a history of fainting and amenorrhoea).

torsion and volvulus (usually marked tympanic abdo distension with absolute constipation and late tenderness)

obstruction due to a strangulated or incarcerated hernia (usually absolute constipation of both faeces and flatus, in the early stages colicky then continuous pain; check hernia sites)

intestinal parasites (ascaris in a mass in children, usually retain bowel sounds of obstruction)

Food poisoning (often affects a group and usually vomiting and diarrhoea supervenes);

perforation of a peptic ulcer (sudden onset; classically a board-like rigid abdomen that is acutely tender with marked rebound tenderness. Erect Chest-Xray shows gas under the diaphragm); ,

amoebic liver abscess (marked tenderness on compression of the rib-cage overlying the liver),

pancreatitis (may mimic perforated peptic ulcer but usually the patient is not lying so still and often sits forward to find pain relief. Marked central abdo pain and vomiting),

appendicitis (rare in rural Africa. Marked anorexia, mild fever, rebound tenderness over the site or tender mass p.r. on right side),

renal calculi (Pain often colicky and very severe, starts in the loin and radiates down to the groin); , *adhesions* (scar of previous operation,usually causes partial obstruction with colicky pain, distension, nausea and sometimes vomiting), ,

cholecystitis (rare in rural Africa; usually stone impacted in the cystic duct. Murphy's sign present*) ;

*Murphy's sign: lay two fingers over the right upper quadrant. Ask the patient to breathe in. This causes pain and arrest of inspiration when the inflamed organ impinges on the fingers.

Not all abdominal pain comes from the abdominal organs. Here are 5 common non-abdominal or atypical causes of abdominal pain

1. "Psychiatric" causes including anxiety, depression, impotence. Ask about related symptoms.
2. Sickle-cell anaemia and mediterranean fever. Here the Hb abnormality causes circulatory changes in the abdomen which mimic an acute abdomen.
3. Irritation of the diaphragm caused by Bornholm disease (pleurodynia) or lobar pneumonia or a myocardial infarction. These may mimic pleurisy, a perforated DU or even appendicitis.
4. Periodic syndrome in children. The child complains of central abdominal pain of acute onset, feeds are refused, the child becomes pale and within 2-3 hrs starts to vomit. The pain persists about 1 day. The stool remains normal but fever may occur. the abdomen remains soft on palpation.
5. Recurrent pain of childhood. This differs from the above in that it is not associated with vomiting.
6. More uncommon or rare causes: diabetic ketoacidosis, porphyria, root pain of herpes zoster before vesicles appear, tertiary syphilis with lightning pains of tabes dorsalis.

Acute abdomen where pain may be missing or minimal

1. Older patients with peritonitis from e.g. appendicitis
2. Sub-acute haemorrhage from an ectopic. This is rare but important to recognize: the pale woman with "ascites" where in fact the belly is full of blood.
3. An acute abdomen in an infant. The symptoms may be lacking or misleading.

Abdominal disease where the pain may be elsewhere

1. Back pain in carcinoma of the body of the pancreas, or oesophagus or a peptic ulcer penetrating the pancreas.
2. Shoulder tip pain e.g. in a ruptured ectopic or a ruptured spleen. Usually there is associated severe abdominal pain but sometimes not.
3. Pain in the chest neck and arms e.g. due to the pain of gastric distension, likewise oesophagitis.

Ryle's 10 questions are important in assessing the pain: its character, severity, situation, localization, paths of reference, duration, frequency, special times of occurrence, aggravating factors and relieving factors.

Appendix 6 Chest Pain

There are two main groups: those with acute pain who look ill and those who look well but have a history of pain that needs to be investigated. Below are important causes out of the 85 possible diagnoses!

1. Pleural pain. This is usually a sharp stabbing pain localized to the area of the chest overlying the inflamed pleura and made worse by inspiration, coughing. Diaphragmatic pleurisy often radiates to shoulder tip. Look for underlying pneumonia. Pleural friction rub sometimes heard over the area on deep inspiration.
2. Oesophageal pain: associated with hiatus hernia or acid reflux. Typically central chest or neck pain often burning in nature but may mimic MI or angina. Usually brought on by large meal, bending or lying flat. Usually relieved by antacids. May show up on barium swallow with head down tilt to promote reflux. Provoked by acid infusion into the lower oesophagus (8-10 cms above oesophageal hiatus).
3. Spontaneous pneumothorax. Pain + dyspnoea disproportionate to degree of lung collapse. Worse if there is underlying lung disease e.g. emphysema. Apex to opposite side. Hyper-resonance on percussion with diminution of breath sounds.
4. Pericarditis: similar pain to angina and MI but sharper. Persistent soreness in neck and shoulders. Worse pain on inspiration and relieved on leaning forwards. Pericardial friction rub may be heard in some positions. In tamponade paradoxical pulse, JVP raised, quiet heart sounds. ECG may show ST elevation concave upwards.
5. Angina pectoris. This is virtually unknown in rural Africa. However it is now appearing in some urban settings. The pain is usually dull, heavy crushing or constricting under the sternum. Commonly radiation to neck, shoulders and inner aspects of upper arms. May radiate to epigastrium, back, jaw and teeth. Relieved by rest for 3-5 mins and rapid relief with nitroglycerine.
6. Unstable angina: recent onset of progressive pain not completely relieved by rest and may wake the patient from sleep.
7. Myocardial infarction: see section under MI
8. Effort syndrome: often accompanying symptoms of shortness of breath, palpitations, fatigue and often left inframammary pain. May be intermittent stabbing pain or more prolonged dull aching pain associated with overlying tenderness. Often follows exercise. Response to forced hyperventilation may be dramatic. Normal ECG.
9. Aortic aneurysm and dissection: acute emergency with severe tearing chest pain radiating to shoulders, neck, back and lower limbs. Often accompanying hemiparesis, disappearance or inequality of pulses and shock. Underlying atherosclerosis with hypertension is common cause in Westerners but rarely tertiary syphilis can cause this.
10 Miscellaneous: *Bornholm disease* with shoulder-tip pain; *herpes zoster* before vesicles appear; *carcinoma of lung* usually a deep-seated dull ache but if there is a secondary in a rib: sharp localized pain. *Spinal pain* causing root stimulation. *Tabes dorsalis* "like a bolt from the blue". *Tietze's syndrome:* costochondritis with a tender junction of (usually) the 2nd rib and the cartilage worse on motion, coughing.

Ryle's 10 questions can also be used here in assessing the pain: its character, severity, situation, localization, paths of reference, duration, frequency, special times of occurrence, aggravating factors and relieving factors.

Appendix 7 Cough

The cough reflex is initiated by stimuli of irritant receptors in the larynx, trachea and major bronchi.

Character of the cough

The type of cough may give a clue as to the likely cause:

A *dry cough* with an irritative hacking quality short and often repeated is heard in inflammatory conditions of the pharynz, tracheobronchitis and in early pneumonia.

A *harsh cough* is heard in laryngitis e.g. in croup.

A cough with a long whoop-like inspiratory sound is heard in laryngeal and tracheal inflammation as in whooping cough or certain viral infections in children e.g RSV

N.B.The characteristic cough in the early stages of whooping cough before the whoop starts is a *paroxysm* of coughing without an initial inspiration (in bronchitis the child almost always breathes in first before coughing). If a paroxysm of coughing ends with vomiting pertussis is likely.

A prologed *lowing cough* like the sound of cattle is heard when the abductor muscles of the vocal cords are paralyzed. This is usually due to pressure on the left recurrent laryngeal nerve by lesions in the thorax e.g. carcinoma of the bronchus or oesophagus, enlarged hilar nodes or an aortic aneurysm.

A hard metallic *"brassy" cough* is heard when lesions press on the trachea but spare the nerve.

Timing of the cough

A cough that wakes the person up in the small hours of the night suggests asthma even if there is no wheeze.

A cough with copious sputum on arising in the morning is characteristic of chronic bronchitis but may occasionally occur in asthma.

A bout of coughing after eating or on lying down after food suggests oesophageal, pharyngeal or neuromuscular disease causing aspiration into the lungs.

Cough brought about by change of posture suggests bronchiectasis, chronic lung abscess, TB with a cavity or a pedunculated tumour. In bronchiectasis free production of sputum may occur at any time of the day.

A dry cough which persists over many weeks might be a sign of a neoplasm.

A cough persisting for more than 3 weeks with mucoid, purulent or blood-stained sputum especially if there is any evening fever or night sweats or any weight loss suggests pulmonary TB.

Nature of the sputum

Airway mucus is 95% water and it's characteristics come from the glycoprotein

Non-infected mucoid sputum is clear, white or jelly-like. Exceptionally viscid sputum is sometimes seen in asthma and in pertussis. In other asthma sufferers the sputum is clear and frothy (like "surf").

In infection, cells and micro-organisms give a yellow colour which changes often to green. This does not necessarily mean bacterial infection since the same process occurs in viral infection.

In most lower respiratory infections pus is mixed with mucus to produce mucopurulent sputum.

Pure pus can be coughed up from a lung abscess or stagnant bronchiectatic cavities. This is often offensive because of anaerobes. "Anchovy sauce" brown-coloured sputum points to an amoebic lung abscess (rare).

Blood in the sputum always needs investigation. However in Europe only 50% have a final diagnosis. First establish that it is definitely coming from the lungs and not from the nose or GIT. In true haemoptysis there is usually froth due to mixed air and the blood is bright red and is alkaline. In haematemesis it is dark brown and acid.

Common causes are bronchitis, bronchiectasis where the blood is often mixed with mucopurulent sputum. In pneumonia it is classically rust coloured. In TB frank blood in otherwise mucoid sputum is common. Sudden haemoptysis is seen in pulmonary embolism with infarction. In neoplasms there may be daily streaking of the sputum with blood or more substantial bleeding with clots. In pulmonary oedema the sputum is often pink and frothy. Mitral stenosis may cause recurrent haemoptysis.

The most important investigation if the cough with haemoptysis has gone on for 3 or more weeks is a stain for AAFB.

Appendix 8a Diagnosis of an acute febrile illness

This is a major challenge to clinical acumen at all levels but especially when laboratory facilities are very limited. Fevers can be divided into acute (<2 weeks) and chronic (>2 weeks). There are two obligatory tests in many settings in the tropics:

1. Thick film for malaria parasites
2. Total and differential white blood count (WBC).

However a positive malaria slide in a holoendemic area for malaria may not mean much unless the number of parasites exceeds 400/μL (this is approximately 5 parasites per 100 white blood cells). Even if parasites were present, if the WBC was raised and there was a polymorphonuclear leucocytosis (PNL) then another diagnosis must also be present.

Acute fever with a negative malaria blood film

1. *PNL present:* 5 most likely diagnoses are pyogenic infection, leptospiral infection, relapsing fever, amoebic liver abscess, and acute connective tissue disease.
2. *PNL absent:* 3 most likely diagnoses are viral infection, rickettsial infections and typhoid.

Acute fevers with PNL and localizing symptoms

Clinical pointers to the diagnosis include: severe sore throat (Strep tonsillitis or diphtheria), cough, pleuritic pain , rusty sputum (pneumonia), severe pain and swelling in a joint (pyogenic arthritis), frequency, dysuria and loin pain (pyelonephritis), severe headache and neck stiffness (meningitis), localized pain over a bone (osteomyelitis), lower abdominal pain with tenderness (pelvic sepsis), bloody diarrhoea (bacillary dysentery or campylobacter infection), marked localized lymphadenopathy (local sepsis, plague), sharply defined cutaneous inflammation (erysipelas) or ill-defined subcutaneous inflammation (cellulitis).

Acute fever with PNL and no obvious localizing features

1. Septicaemias of various kinds e.g. staphylococcal, strep or meningococcal; acute bacterial endocarditis
2. Infections with Leptospira and Borrelia; tick-borne relapsing fever; louse-borne relapsing fever.
3. Acute non-typhoid Samonella sepsis even without GIT symptoms. Check for splenomegaly.

Acute fevers without PNL with a negative blood film

1. Look for indicators pointing to typhoid fever *see typhoid section.*
2. Some rickettsial infections have pointers e.g. a rash or as in African tick typhus (R. conorii) - headache + eschar and lymphadenopathy. An outbreak in an army or school may give a clue.
3. Most viral fevers have a non-specific picture but dengue gives a double-humped fever pattern. Hepatitis before the jaundice may give nausea and pain over the liver.

Acute fever with haemorrhagic rash

There are two main groups of causes:

1. Viral haemorrhagic fevers (dengue, Chikungunya, Ebola, rift valley fever)
2. Acute meningococcal septicaemia. Material aspirated from the spots show meningococci on staining in 80% of cases.

Acute fever with anaemia

The two commonest causes are malaria and infection in a patient with pre-existing anaemia due to diet, hookworm, sickle cell anaemia, thalassaemia or with G6PD deficiency. Rarer causes are babesiosis and bartonellosis.

Appendix 8b Diagnosis of a chronic febrile illness

In many countries an important diagnosis not to miss is HIV infection with or without opportunistic secondary invaders. *See section on HIV*

Fever for more than 2 weeks. This may include some of the above especially typhoid.

1. Chronic fever with PNL

With a sustained fever think of deep sepsis (e.g. sub-phrenic, periappendicular, diverticular), amoebic liver abscess (check for tenderness on rib-cage compression), erythema nodosum leprosum (check ear lobes and cool parts of face for nodules, and swelling of nerves). With a relapsing pattern think of cholangitis or relapsing fever.

2. Chronic fever with eosinophilia

Most likely causes: invasive Schistosoma mansoni and japonicum infections; invasive Fasciola hepatica infection

3. Chronic fever with neutropenia

Most likely causes malaria, disseminated TB, visceral leishmaniasis, brucellosis.

4. Chronic fever with normal WBC

Most likely causes: typhoid, localized TB, brucellosis, secondary syphilis, trypanosomiasis, toxoplasmosis, subacute endocarditis, systemic lupus erythematosus, chronic meningococcal septicaemia.

5. Chronic fever with variable WBC picture

Tumours, reticuloses, drug reactions

If the fever has a relapsing pattern think of :

malaria, visceral leishmaniasis, trypanosomiasis, relapsing fever, brucellosis, filiariasis, cholangitis.

Reference for appendices 9a. and 9b.

Bell DR. Lecture notes on Tropical Medicine 4th Ed 1995 130-138 Blackwell Science. Oxford

Appendix 9 Fever of unknown origin

This is defined as an illness of more than 3 weeks duration with a fever of more than 38.3°C (101°F) on several occasions and with 1 week of adequate evaluation without result. Four distinct groups of prolonged fevers have been suggested: Classical, nosocomial, neutropenic (<1000PNL/mm^3) and HIV.
Most of the studies of FUO have been done in developed countries and so priorities differ very markedly. However it is worth noting the most common causes found:
1. Classical: malignancies (lymphomas i.e. Hodgkin's and Non-Hodgkin's lymphoma, leukaemias, other haematological malignancies and solid tumours e.g. renal cell carcinoma, hepatoma and atrial myxoma), infections (abscesses esp in the abdomen, mycobacterial, endocarditis UTI, CMV, EBV, HIV, amoebic abscess, visceral leishmaniasis, brucellosis, inflammatory conditions (Still's disease, polyarteritis nodosa, SLE, temporal arteritis, polymyalgia rheumatica, rheumatic fever), undiagnosed habitual hyperthermia.
2. Nosocomial: nosocomial infections, post-operative complications, drug fevers.
3. Neutropenic: majority due to infections but cause only found in 40-60%.
4. HIV related: HIV effect, typical and atypical mycobacteria, CMV, lymphoma, toxoplasmosis.

The most treatable cause of FUO which is otherwise life-threatening is disseminated TB often without the characteristic miliary pattern on Chest Xray, or extrapulmonary disease without clearly localising features. Many have a negative tuberculin test. The ESR is usually raised and anaemia is common. Sputum for AAFB is only positive in one fourth. However lung and liver biopsies show granulomas in 80-90% of the disseminated group.
In a setting where TB is common and TB culture or biopsy facilities are unavailable the most valuable final test is a trial of TB therapy using drugs which do not affect other organisms e.g. INAH, ethambutol and pyrazinamide. If there is no change in the fever pattern within one week (and absolutely within two weeks) this is not TB. If there is a good response, add rifampicin to prevent any danger of resistance building up.

In a high-income-country the **minimum diagnostic evaluation** includes a good history and repeated examinations, complete blood count with differential and platelet count, liver function tests, urinanalysis with microscopic examination, Chest Xray, ESR, Antinuclear factor, rheumatoid factor, angiotensin converting enzyme, routine blood culture (x3) while not receiving antibiotics, cytomegalovirus IgM antibodies or virus detection in blood, heterophile antibody test in children and young adults, tuberculin skin test, CT of abdomen or radionuclide scan, HIV antibodies or virus detection assay.

References

Oxford Textbook of Medicine. Weatherall et al. 1996. 3rd Edition. OUP. Oxford. 1015-1020
Arnow PM and Flaherty JP. Fever of unknown origin. Lancet 1997; 350: 575-580
Durack D Street A. Fever of unknown origin - reexamined and redefined. In: Remington J. Swartz M. eds. Current Clinical Topics of Infectious Diseases. St. Louis. MO: Mosby-Year Book Inc.; 1991:35

Appendix 10 Method of improving stain for AAFB

Mix 1-2 mls of sputum with an equal volume of household bleach (4-5% NaOCl) in a 10 ml screw-capped tube. Incubate the mixture at room temperature for 10-15 mins and shake this regularly. Then add 8 mls distilled water and centrifuge the sample. Discard the supernatant and then prepare slides from the remaining material. Stain with the standard Ziehl-Neelsen method and then examine with bright-field microscopy at 1000 x magnification.
This should increase the number of specimens with AAFB discovered by straight staining and microscopy by around 100%.

Reference

Gebre N et al Improved microscopical diagnosis of pulmonary TB in developing countries. Trans Roy Soc Trop Med Hyg 1995 89: 191-193

Does the child have diarrhoea?

IF YES, ASK:	*LOOK AND FEEL:*
• For how long? • Is there blood in the stool?	• Look at the child's general condition. Is the child: Lethargic or unconscious? Restless and irritable? • Look for sunken eyes. • Offer the child fluid. Is the child: Not able to drink or drinking poorly? Drinking eagerly, thirsty? • Pinch the skin of the abdomen. Does it go back: Very slowly (longer than 2 seconds)? Slowly?

Classify DIARRHOEA

for DEHYDRATION	Two of the following signs: • Lethargic or unconscious • Sunken eyes • Not able to drink or drinking poorly • Skin pinch goes back very slowly.	**SEVERE DEHYDRATION**	▸ If child has no other severe classification: - Give fluid for severe dehydration (Plan C). OR ***If child also has another severe classification:*** ***- Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way.*** ***Advise the mother to continue breastfeeding.*** ▸ ***If child is 2 years or older and there is cholera in your area, give antibiotic for cholera.***
	Two of the following signs: • Restless, irritable • Sunken eyes • Drinks eagerly, thirsty • Skin pinch goes back slowly.	**SOME DEHYDRATION**	▸ Give fluid and food for some dehydration (Plan B). ▸ ***If child also has a severe classification:*** ***- Refer URGENTLY to hospital with mother giving frequent sips of ORS on the way.*** ***Advise mother to continue breastfeeding.*** ▸ Advise mother when to return immediately. ▸ Follow-up in 5 days if not improving.
	Not enough signs to classify as some or severe dehydration.	**NO DEHYDRATION**	▸ Give fluid and food to treat diarrhoea at home (Plan A). ▸ Advise mother when to return immediately. ▸ Follow-up in 5 days if not improving.
and if diarrhoea 14 days or more	• Dehydration present.	**SEVERE PERSISTENT DIARRHOEA**	▸ Treat dehydration before referral unless the child has another severe classification. ▸ Refer to hospital.
	• No dehydration.	**PERSISTENT DIARRHOEA**	▸ Advise the mother on feeding a child who has PERSISTENT DIARRHOEA. ▸ Follow-up in 5 days.
and if blood in stool	• Blood in the stool.	**DYSENTERY**	▸ ***Treat for 5 days with an oral antibiotic recommended for Shigella in your area.*** ▸ Follow-up in 2 days.

If referral is not possible, manage the child as described in **Management of Childhood Illness, Treat the Child, Annex: Where Referral Is Not Possible, and WHO guidelines for inpatient care.*

ASSESS AND CLASSIFY THE SICK CHILD AGE 2 MONTHS UP TO 5 YEARS

ASSESS

CLASSIFY

IDENTIFY TREATMENT

ASK THE MOTHER WHAT THE CHILD'S PROBLEMS ARE

- Determine if this is an initial or follow-up visit for this problem.
  - If follow-up visit, use the follow-up instructions on *TREAT THE CHILD* chart.
  - If initial visit, assess the child as follows:

CHECK FOR GENERAL DANGER SIGNS

ASK:	*LOOK:*
• Is the child able to drink or breastfeed? • Does the child vomit everything? • Has the child had convulsions?	• See if the child is lethargic or unconscious.

A child with any general danger sign needs *URGENT* attention; complete the assessment and any pre-referral treatment immediately so referral is not delayed.

USE ALL BOXES THAT MATCH THE CHILD'S SYMPTOMS AND PROBLEMS TO CLASSIFY THE ILLNESS.

THEN ASK ABOUT MAIN SYMPTOMS:

Does the child have cough or difficult breathing?

IF YES, ASK:	*LOOK, LISTEN, FEEL:*
• For how long?	• Count the breaths in one minute. • Look for chest indrawing. • Look and listen for stridor. (CHILD MUST BE CALM)

→ *Classify COUGH or DIFFICULT BREATHING*

If the child is:	Fast breathing is:
2 months up to 12 months	50 breaths per minute or more
12 months up to 5 years	40 breaths per minute or more

SIGNS	CLASSIFY AS	TREATMENT (Urgent pre-referral treatments are in bold print.)
• Any general danger sign or • Chest indrawing or • Stridor in calm child.	**SEVERE PNEUMONIA OR VERY SEVERE DISEASE**	▸ ***Give first dose of an appropriate antibiotic.*** ▸ ***Refer URGENTLY to hospital.****
• Fast breathing.	**PNEUMONIA**	▸ ***Give an appropriate antibiotic for 5 days.*** ▸ Soothe the throat and relieve the cough with a safe remedy. ▸ Advise mother when to return immediately. ▸ Follow-up in 2 days.
No signs of pneumonia or very severe disease.	**NO PNEUMONIA: COUGH OR COLD**	▸ If coughing more than 30 days, refer for assessment. ▸ Soothe the throat and relieve the cough with a safe remedy. ▸ Advise mother when to return immediately. ▸ Follow-up in 5 days if not improving.