GENERAL INTRODUCTION

"Discard in the first instance all attempts to identify or to name, and try instead to read the malady, tracing the symptoms to the seat of their cause, and discerning the nature of the morbid process by their character and course"

William Gowers, 1892.

'Do not touch the patient - state first what you see; cultivate your powers of observation'

Sir William Osler1849-1919

THE CRUX OF CLINICAL METHOD

To learn clinical diagnosis, you must be able to think in a relatively structured manner. You are now at a crucial point in your medical training. If you take the wrong path along the cross-roads from here on, you may miss the whole point of how helpful your preclinical science training can be in diagnosing and managing real patients. Possible paths are as follows:

1. Let us forget about all those basic medical sciences and get on with 'real' medicine. With the basics behind us, we can now learn the real facts, come to know the important conditions in medicine and to diagnose them by recognizing their symptoms and signs. Thus, mitral valve narrowing is*characterized* by shortness of breath, a loud first heart sound and an apical mid-diastolic murmur. According to this approach, as long as we collect the symptoms and signs accurately, we should be able to recognize the diagnosis, i.e. re-cognize the disease pattern to give our diagnosis a probability rating accordingly. Now, at times this is true, particularly when the diagnosis is fairly obvious. As Thomas Lewis said of cardiac murmurs, we often diagnose their origin just as we recognize a dog by its bark. But there are many pitfalls and much frustrartion in this approach, particularly for any student starting without any patterns from which to re-cognize in this way. In any case, when a patient presents with any variant of a disease - and this is more often the rule than the exception - we finish up trying to put square pegs into round holes. And, of course, such an approach could never allow us to re-cognize any new disease. Our aim should be to describe as accurately as possible the condition suffered by the individual patient before us on the basis of his own symptoms and signs. This is a far safer way, for pattern-recognizers so often fall into the trap of thinking they have seen the 'true' pattern of the disease early on, and then, because of that bias, are not able to even see findings that don't fit into the pattern. As you gain experience, you will come to re-cognize partial or even complete

diagnostic patterns, but this is not the way to begin. Our approach is essentially to gradually 'corner' the diagnosis.

2. The extreme pattern-recognizer takes the view that the pre-clinical sciences such as physiology etc. play no primary part in clinical diagnosis. A common approach there, perhaps more dangerous because more subtle, is to say that we should use our knowledge of physiology in that, having made the diagnosis (by pattern-recognition), we can then explain the various disease aspects in physiological terms, e.g. aortic valvular incompetence causes cardiac dilatation because..... Now this may sound reasonable, and is useful in demonstrating your understanding of the pathophysiology of what I call the "chain of mechanism" in disease, but in real clinical situations it is not at all as helpful in diagnosis as you may think, because in effect, it 'tells the story backwards' i.e. gives an explanation of the diagnosis after the event. Indeed, as a stand alone exercise, it can merely be a way of just showing off!

3. Much has been written about clinical logic and problem-based learning but, for all that, their methodology has never really been defined. It is usually proposed to be diagnosis by a 'hypothetico-deductive' process, i.e. as you go along, various thoughts arise and are tested by subsequent questions and observations, with many hypotheses/diagnoses being rejected until a best conclusion is drawn. But how you are supposed to come by these ideas or hypotheses is not discussed, and because of this, the whole approach is little different from the "pattern-recognition" method discussed above, and certainly not very helpful to the beginner-student. The same can be said about much of what goes by the name of "problem-based" diagnosis.

One of the real problems in synthesizing clinical diagnosis is the amount of information we have to deal with, including the present illness, the past history, family history, social background, examination findings etc. If one goes into appropriate detail, we can have so much information that we may be unable to see the wood for the trees. Then it is very difficult to make a diagnosis "all-of-a-piece" by any "inductive" or other process of reasoning. Because of that, what we need is to handle the clinical information within a small number of separate sub-categories which are to a large extent independent, but which, when put together, severally describe all aspects of the condition or diagnosis.

Now, I suggest that you can do this by approaching medical diagnosis in the categories you have already learnt in your major pre-clinical disciplines namely:

1. WHERE is the problem? ANATOMICAL DIAGNOSIS = the organ system involved.

2. WHAT, is the pathological nature of the process? GENERAL PATHOLOGICAL DIAGNOSIS.

3. HOW does it affect the patient? FUNCTIONAL OR PHYSIOLOGICAL DIAGNOSIS.

4. WHY did this particular patient get it? AETIOLOGICAL DIAGNOSIS underlying the condition.Subsumed under this category is also the question of WHO has the illness, and why it occurred WHEN it did. The WHO relates to the type of patient concerned, in particular to the social and psychological contexts of the presentation. The WHEN aims to focus on its precipitating factors.

The value of this approach is that it allows you to focus down and 'corner' the diagnosis within each category on less than the total body of information, and so gives you a much greater chance of reaching the correct conclusion. Also, each of these aspects does indeed contribute importantly to clinical diagnosis and, when taken together, the four categories severally describe all the elements of full diagnosis. By contrast, when the "pattern-recognizer" pins some "off-the-rack" diagnostic label on a patient, it so often lacks one or other of these categories, particularly the Functional and Aetiological ones. Thus, we often hear that a patient with a heart attack has had an acute myocardial infarction (Anatomical and general Pathological diagnoses), but where is the comment about how this has affected him functionally (did he have secondary heart failure or ventricular dysrhythmias, for example?); and what were the long term factors predisposing to his condition and a repeat of it, and the more recent ones precipitating it? (Aetiological diagnosis: e.g. in this case was there pre-existing 'cardiiovascular risk marker' pre-disposing to atheroma, and some stressful life event precipitating the episode itself?)

The important point here is that by using this approach, you can TAILOR-MAKE a diagnosis to the INDIVIDUAL patient as you go along, rather than force him into some pre-conceived diagnostic pigeonhole read about in a text book.

Like Kipling, we are trying to learn by simplification:

"I keep six honest serving men,

(They taught me all I knew.)

Their names are What and Why and When

And How and Where and Who."

From R. Kipling's "Just So Stories"

Essentially, what we do is to hone down on particular categories of diagnosis at any given time, and in this way are less likely to be overwhelmed by the information we have so painstakingly collected.There are also principles we can build up within each of our four categories to help reach a

diagnosis in a more logical or real PROBLEM-SOLVING way. In some respects, this is much like solving a jigsaw puzzle where we realise that certain pieces are clearly related to, say, sky, others to trees, and others to men, houses etc., and which can all be put together and solved as subunits before going on to the solution of the overall puzzle itself. Actually, once you have solved the problem in each of the four categories, you will sometimes see that they can be put together in different ways. And now that you only have a limited number of combinations to deal with, you will often be able to see such possibilities. For example, one may see hypertension associated with renal disease, and this can be looked at both ways around i.e. as hypertension causing renal disease, or being its effect.

An important LESSON is that when you are in a quandary in such respects, always look for what came first in time, and this is one way in which careful history taking is so important.

Throughout the tutorials and chapters which follow, we will approach patients in a way that helps us formulate the categories of Anatomical, Pathological, Functional (Physiological) and Aetiological diagnosis. In the remainer of the Online Reference/Book, the emphasis will be on the physiological. This is deliberate not just because of the importance of physiology to the Functional or 'How?' but because it also often helps define the system involved, and therefore delineate the Anatomical diagnosis. When the system involved is hidden from our clinical examination, it may be especially difficult directly to determine the Anatomical diagnosis, so the *only* clinical information we have about Anatomy is the indirect physiological. Thus, in a patient with vague and atypical chest pain and shortness of breath, a history of discomfort on lying flat (orthopnea) and attacks of shortness of breath at night (paroxysmal nocturnal dyspnoea) gives the clue to an impairment of function of the left heart, and therefore to the heart as the anatomical site of the diagnostic problem. Indeed, in some areas such as the central nervous system, our Anatomical diagnosis rests almost entirely on our knowledge of (neuro-)physiology.

THE FOUR DIAGNOSTIC CATEGORIES

Anatomical Diagnosis (WHERE?)

Information about this category is given to you from both the clinical symptoms and the physical signs, the proportion contributed by each depending on the particular system involved. Thus, with exposed organs the examination yields most information, but with hidden ones the history may be more important. In taking a history, always define, as the patient unfolds 'his-story', the major symptom or symptoms involved, as well as their site (and in the case of pain, the radiation), their duration and time-intensity relationships, and their quality, as well as aggravating, precipitating and relieving factors. Among these symptoms, the site of the major problem (and its radiation if pain), as well as the precipitating, aggravating and relieving factors, are particularly important in making rational deductions about the anatomical site of the problem, because they often point in a common sense way to the organ involved (e.g. chest pain aggravated by respiration suggests involvement of the parietal pleura, anatomically).

The site and radiation of (referred) pain can help on the basis of the sensory innervation of the various internal organs, although that this innervation is often so variable that one does have to come to recognise various patterns and variations both in the distribution of referred pain and its quality, e.g. in myocardial ischaemia/infarction. Particularly is this so in relation to the quality of pain, because though very important, there is little knowledge about why pains from certain organs have particular characteristics, and we are almost entirely reliant on recognizing its pattern e.g. the "heavy" or "crushing" chest pain of myocardial infarction. But often deductions of a more logical nature can be made by unearthing knowledge about precipitating, relieving or aggravating factors. For example, central retrosternal chest discomfort, precipitated by exercise and relieved by rest would, on rational grounds, suggest something to do with the heart or upper respiratory tract, i.e. *mediastinal* systems put under load by exercise. Similarly, aggravation of any retrosternal pain on swallowing would point more to the oesophagus, and alteration of the symptoms by position would suggest pericardial involvement (because different positions may vary the relationship of the visceral to parietal pericardium rubbing together).

Hierarchic Approach to Diagnosis

This brings out our second principle of diagnoses which holds within ALL of our four diagnostic categories, i.e.:

ALWAYS TRY TO ANALYSE CLINICAL INFORMATION BEGINNING FROM THE BROADEST LEVEL FIRST AND ONLY NARROWING TO THE LEVELS OF DETAIL SUBSEQUENTLY.

Thus if a patient has an RETROSTERNAL chest pain, we can be fairly certain that he has a:

MEDIASTINAL COMPLAINT. Don't jump too early to the conclusion that it is of cardiac origin, just because that is common. Its precise site, radiation and quality may help define its origin, but in obscure cases we should at least be able to corner the problem by uncovering the aggravating, relieving and precipitating factors.

In the history, ask about associated phenomena because these can be very important in defining the anatomical site as well as the physiology of the problem. Thus, in the patient above with obscure chest pain, symptoms of left heart failure would strengthen the case for the pain having a cardiac origin.

Contribution of Physical Examination to Anatomical Diagnosis

Sometimes the evidence concerning Anatomical diagnosis is direct, either obvious to the patient himself, or evident to the medical examiner. In the latter respect, it is important to know the

SURFACE ANATOMY of different organs, in particular the various lobes of the lung, the abdominal organs, and the positions for cardiac auscultation, etc.

When the clinical evidence is more indirect, physiological derangements of function may be all that are available to indicate the anatomical site of the lesion. In this respect, it is particularly important to know the sensory and motor dermatome nervous distributions for the body, including where pain is referred from the various internal organs to the external surface of the body, as well as the different types of pain, e.g. colic with blocked abdominal tubes. If you know the sensory (often sympathetic) afferent innervation of the various organs, you can often work out just what the site of the lesion will be. Therefore in reverse, you will be able to work out what organ is involved when you are dealing with referred pain of a certain dermatome distribution.

Pathological Diagnosis (WHAT?)

Tthe general clinical pathological nature of the condition. As far as clinical pathology is concerned we can really only recognise general pathology such as inflammation, neoplasia, ischaemia/infarction due to vascular obstruction or haemorrhage, other hollow tube obstruction or rupture; and degenerative, traumatic, hereditary/constitutional/familial and allergic/immunological conditions.

The onset, duration, and time-intensity relationships of the symptom, and the presence or absence of weight loss, as well as local and general evidence of inflammation, are all-important in deriving the clinical pathological diagnosis.

(a) If of very sudden, dramatic onset, this signifies either a HYPER-ACUTE mechanical event or, in most medical cases, the sudden obstruction or rupture of a hollow tube somewhere within the body. If rapidly reversible, rupture is unlikely, and obstruction more probable - in the case of vascular obstruction, either embolic or thrombotic. Duration helps define clinical Pathology even further in such situations. Thus, a short-term interruption of blood supply may only result in ischaemia or damage of the organ involved, whereas longer-term interruption can result in its infarction or death. Obviously, this cut-off line between damage and infarction will vary greatly between tissues (e.g. minutes in the brain, hours in the skin, 1-3 hrs in the case of the heart).

(b) Onset over a period of hours or days is referred to as ACUTE. This includes acute inflammatory as well as other conditions such as allergic ones. The clues to INFLAMMATION should always be looked for, namely the GENERAL evidence (fever, lassitude, general malaise, sweats, shivers etc.), as well as LOCAL evidence, including tenderness, pain, swelling, heat, redness and loss of function in an organ accessible to examination. Local evidence includes information available from secretions (e.g. purulent secretions in bacterial inflammation), especially where the organ is less accessible, e.g. in the lung (sputum), renal tract (urine), gastro-intestinal tract (stool, vomitus). Examine secretions

closely, including microscopy for polymorphonuclear leucocytes, since neutrophils usually indicate (bacterial) inflammation, and eosinophils allergy or parasitic infection.

Other bodily fluids include blood and serous secretions which may give the clue to vascular or ulcerative pathological process, whether acute or chronic (e.g. ulcerative colitis).

Important information can be obtained from the full blood examination. As a general rule, where acute necrosis (tissue death) or bacterial inflammation is suspected, the blood neutrophil polymorphonuclear leucocyte count will be elevated, often with a "left shift" (i.e. a reduction in the average number of lobes per neutrophil nucleus indicating the release of more immature forms due to high neutrophil turnover). In cases of more severe bacterial inflammation such as septicaemia, "toxic changes" and "Doehle bodies" - cytoplasmic granulations and inclusions respectively - may also be present. Viral inflammations characteristically give rise to an increase in blood lymphocytes and/or monocytes. The erythrocyte sedimentation rate (ESR), or C-reactive protein (CRP) are useful indicators of subtle inflammation.

(c) Conditions continuing for weeks are referred to as SUB-ACUTE and those going on for months or more as CHRONIC. In both, evidence about inflammation should be sought. If present, this is often associated with weight loss. However if fever is absent, chronic progressive symptoms, especially when combined with weight loss, generally suggests a chronic neoplastic process. If, on the other hand, the patient's symptoms come and go over a period of months or years, determine whether you are dealing with a truly relapsing and remitting process entirely, or one where acute bouts are super-imposed on a slowly progressive chronic background of continuing disability. For example, so-called "chronic" duodenal ulcer is characterised by periods of pain for some weeks, with times in between, often months, when the patient is completely free of pain. Thus, this should really be called an acute relapsing process (recurrent healing and breakdown). Then, it becomes particularly important to ask about possible precipitating factors for each bout (relevant to Aetiological diagnosis).

(d) VERY CHRONIC conditions, which have gone on for years have, by and large, lasted too long for us to seriously consider neoplasia, at least malignant neoplasia, and belong more in the realm of chronic inflammatory, degenerative, hereditary, constitutional, auto-immune or immunological processes.

As with the Anatomical diagnosis, the history can be all-important in reaching the Pathological diagnosis in remote organs such as the brain, cardiovascular system and respiratory system. Actually with all systems, when there is an intermittent problem which you have not been lucky enough to observe at the time, the history is the *only* information you have to go on in making a pathological diagnosis. But with less evanescent problems in at least some systems, clinical examination can add powerfully to your assessment of the pathological process involved. This is most obvious in the skin and joints where, for example, the evidence of local inflammation (heat, redness, swelling pain and loss of function) as well as deformity etc. will sometimes be very obvious. The same holds true in some internal organs such as the abdomen, where masses may be palpable to the examining hand. Then, the consistency, shape, size, irregularity, presence of vascular murmurs, and the degree of

tenderness of the mass can add greatly to understanding the pathological nature of the process. For example, a grossly enlarged hard "knobbly" liver with an irregular surface and edge, would suggest infiltration by secondary carcinoma. A uniformly enlarged firm liver would suggest other some sort of chronic process (e.g. fibrous tissue infitration or cirrhosis).

In sorting such abdominal situations out, the presence or absence of a spleen is important (the spleen is rarely involved in secondary carcinoma). Other abdominal masses may be palpable too, and you should define not just their anatomical relationships, but their shape, consistency, extent of enlargement, irregularity, tenderness, and the presence or absence of vascular bruits, to help you better understand the nature of the general pathology involved. Rectal examination will give you essential information not only about the appearance of the stool, but the presence or absence of blood, altered or otherwise. As surgeons say: "If you don't put your finger in it, you'll put your foot in it!"

In the respiratory system, information about the nature of the pathological process is also often available from examination, and you should learn to distinguish the physical signs of consolidation, collapse, pleural effusion, pneumothorax, and chronic obstructive airways disease.

At the extreme, the central nervous system very rarely gives you any direct evidence on physical examination about the nature of the pathological process involved, so you are especially reliant on taking a good history.

Functional Diagnosis (HOW?)

This is the clinical physiological diagnosis, i.e. the disturbances of organ function. Again, this is particularly important in hidden organs such as the central nervous system, where the history and our knowledge of physiology can allow us to pinpoint within a few millimetres the anatomical site of the lesion - especially in the hind-brain.

Important to Functional Diagnosis is the *degree* of dysfunction (impairment, disability, handicap) of the organ system involved. Always state this (e.g. *moderately severe* left ventricular impairment secondary to acute myocardial infarction).

SEQUELAE and COMPLICATIONS arising from the condition are included under this heading (e.g. secondary atrial fibrillation from myocardial infarction, mitral valve incompetence from papillary muscle dysfunction, cardiac tamponade from cardiac rupture).

Cause and Effect

As we have seen, it can sometimes be difficult to separate Functional diagnoses from Anatomical/ Pathological ones, as in patients presenting with both hypertension and chronic renal failure, it is possible that either might be a consequence of the other. In this respect, you need to define which

symptom came first in time, because this will almost certainly be the cause, and the other its effect.An example arises in acute myocardial ischaemia associated with paroxysmal atrial tachycardia. There, the two possibilities are that the myocardial ischaemia caused an altered function of the normal cardiac pacemaker to induce the dysrhythmia; or on the other hand, that a primary atrial tachycardia caused such a rapid ventricular rate that the cardiac output (and with it blood pressure), fell to levels where diastolic perfusion of the coronary arteries became too low to sustain the increased myocardial demand for oxygen, so resulting in angina.

Always define the **EXTENT of the functional disability** under this heading, because this has just as important a bearing on treatment as on diagnosis. Don't talk of organ failure, but of the degree of impairment of function.

Aetiological Diagnosis (WHY?)

Having gained some idea of the general anatomico-pathological diagnostic axis, we next have to delve into the background and ask, "Why did this particular patient get this condition?". This is to determine the long-term background to the problem, for example high blood pressure and/or cigarette smoking as background "risk markers" predisposing to acute myocardial infarction.

Background aetiological factors include: environmental (climate, physical, social, psychological, nutritional, drugs, toxins, etc.), bacterial, viral, parasitic, immunological, allergic, genetic, and - more often than not - unknown ('idiopathic').

Consideration of background aetiological factors suggests that we might usefully expand our number of four "honest serving men" to Kipling's six, by including a further two questions in the aetiological category as follows:

"**WHEN**?" By this I mean, "Why did this particular patient get this condition WHEN he did?" This question is especially useful in unearthing any factors which might have precipitated an acute clinical episode, particularly in acute-on-chronic conditions such as myocardial ischaemia/infarction, where consideration about background aetiology usually relates to the more long-term (atheroma) "risk factors" (high blood pressure, high plasma cholesterol, etc.) and to the inquiry about what could have precipitated such a dramatic-onset clinical event. If no precipitating factors become evident to you, ask our patient what he/she thinks might have provoked the condition. The answers can be difficult to interpret, but they are important for us to know, and are sometimes revealing.

"**WHO**?" By this I mean "WHO has the disease?" To quote Francis Scott Smythe: "To know what kind of a person has a disease is as essential as to know what kind of disease a person has". This is an important question, not only because the personality/character strengths and weaknesses of patients will be important in determining their response to any disease process, but also because an individual patient's reaction to life events and stressors may be crucial as an initiating factor

precipitating his/her clinical condition. For example, acute episodes of bronchial asthma are undoubtedly precipitated in some patients by psychological stress.

"What is spoken of as a 'clinical picture' is not just a photograph of a man sick in bed; it is an impressionistic painting of the patient surrounded by his home, his work, his relations, his friends, his joys, sorrows, hopes and fears." (F.W.Peabody)

There is another important aspect to this Aetiological category of diagnosis, because it can also help allow us to question whether the *clinical setting* in which the diagnosis has been made is reasonable. For example, no matter how strongly the clinical information may point to a general diagnosis of acute myocardial infarction, you should hesitate before applying that label to a 15 year old boy with no obvious predisposing risk markers. The pain may be arising from the cardiovascular system right enough, and the hyper-acute onset of the episode may be well-established, but is it perhaps the pericardium rather than the myocardium that is involved anatomically, so does he perhaps have acute pericarditis?

As far as the structure of our inquiry is concerned, clinical information relevant to the Aetiological diagnosis may not be forthcoming until after the initial history-taking and physical examination have been completed; i.e. it will not be until we have gained a general idea of the Anatomico-Pathological Diagnosis that we will be able to delve more appropriately into its background (Aetiological) cause.

The way we have just seen the Aetiological diagnosis feed-back on, and even challenge the general diagnosis, leads to the next point, namely:

Diagnostic Category Overlap.
Each of the four broad diagnostic categories may interact so as to question or strengthen each other. We have already seen how the Functional diagnosis may interact strongly with the Anatomical one, particularly in organs remote from the direct reach of our examining senses. Also, there are some combinations of Anatomical and Pathological diagnoses which do not fit well together. Thus, damage to peripheral nerves by toxic poisoning would not fit well with a solely unilateral nerve lesion occurring in, say, one limb. Along similar lines, a knowledge about Pathology will sometimes favour one particular Anatomical diagnosis over another, such as in the predilection for cerebral haemorrhage to occur in the internal capsule of the brain rather than in the cortex. Then, if we obtain a history of rapid onset of relentlessly progressive cerebral symptoms suggesting cerebral haemorrhage as the Pathological diagnosis, then an internal capsular Anatomical site for a lesion causing hemiparesis is likely.

Another example of **category interaction**. In a patient presenting with sudden onset dysphasia and weakness in the right lower face and upper limb, now improving, we can say that this is a hyperacute event (the What? of diagnosis) involving the left prefrontal cortex (the Where?). Moreover, it is likely due to a blockage or rupture somewhere within the system. And since it is improving, it is more likely a blockage than rupture. Of course, in the abdomen, where there are many hollow tubes, this may not help much, but in the brain the only important hollow tubes are vascular ones. That means that we can now look at the anatomical diagnosis not just in neuroanatomical terms, but in terms of

neurovascular anatomy. From that, it is a simple step for students to realize that the territory involved is the left middle cerebral artery, and that we are dealing with some sort of vascular obstructive episode, either local thrombosis or embolic from somewhere else. In this way, we have honed down on the clinical anatomical and pathological diagnoses without going too far: we still need information on the degree of dysfunction (the How? of diagnosis), as well as the underlying cause (the Why?: is there any proximal left heart or vascular problem as a potential site for thrombo-embolism).

Once the four diagnostic categories have been satisfactorily answered, it remains to fit them together. Often this process is simple and straightforward, e.g. acute(Path.) myocardial(Anat.) infarction (Path.) with secondary ventricular dysrhythmia and left heart failure(Funct.), on a longterm background of severe hypertension(Aetiol.). But on occasions, the separate diagnostic categories themselves and the way they seem to fit best together, can cause some surprises and even lead to unanticipated conclusions.

This approach will not always give the total diagnosis in particular patients; nor will it always be necessary in making the diagnosis, particularly where the pattern of the disease is clearly recognizable. But in learning the diagnostic process as a student and in dealing with difficult diagnostic problems as a physician, it is extremely helpful. Importantly, it enables us to gradually corner the diagnosis, i.e. to narrow it down to some level of refinement within each of the four diagnostic categories, so as to allow a better approach to the problem overall.

Hierarchic Concept of Diagnosis

This is an important concept to grasp if you are to make physiology and other pre-clinical sciences work for you in building clinical diagnoses. Always define the BROAD areas of the problem within each diagnostic category first, before trying to delineate levels of detail. What you must do here is adopt a HIERARCHICAL approach which allows you to CORNER the diagnosis at each stage. You can do this by asking a number of questions which go from the general to the particular along a series of descending hierarchic levels, not unlike the game of "TWENTY QUESTIONS". There, the efficient player uses a minimum of questions, because he asks the broadest ones first (e.g. animal, vegetable or mineral; alive or dead; to do with mankind etc.) before focusing down on questions of detail. It is no use there guessing that the article in question is a knife, saucer, drawer, or curtain, if it is alive!

Example 1: If a patient has chest pain which is central, we can be fairly certain that it involves some mediastinal organ, and then by questioning about aggravating, relieving factors, etc. and by physical examination of the relevant organs, we should be able to gradually define further and further whether the mediastinal organ involved is the oesophagus, trachea, aorta, heart, pericardium etc.

Example 2: In a patient with low blood pressure, history and examination should determine the broad level of the problem as follows:

(i) Is the low pressure due to a poor pump output, or a drop in the peripheral circulatory resistance, or a decreased circulating blood volume?

(ii) Then, if the answer is a low cardiac output, is this due to decreased heart rate or stroke volume?

(iii) If due to a decreased stroke volume, is it that the heart CAN'T (e.g. valvular obstruction, severe hypertension) or WON'T (e.g. primary heart muscle disease) pump?

(iv) And if there is valvular obstruction, what is its site, nature, and severity?

By doing this we can eventually arrive at the closest approximation to a clinical diagnosis in each of the categories - anatomical, pathological, functional and aetiological.

Example 3: A patient with OEDEMA. The broadest question here must be:

(i) Is the oedema generalized or localized?

(ii) If generalized, is there any evidence of circulatory volume overload. If so, is this due to impairment of renal salt and water excretion from, for example, cardiac failure (reduced renal blood flow and therefore GFR) or from primary renal failure; or is it due to a primary retention of sodium and water.

(iii). If there is no evidence of circulatory volume overload at all, is there ANY evidence of a low plasma oncotic pressure (low plasma albumin) from ANY CAUSE? (Starling's law of capillary filtration?)

(iv). If so, is the trouble that the simple biochemical "building blocks" for protein production are not being taken in (anorexia), not being digested (maldigestion), or not being absorbed (malabsorption); or is the (liver) factory diseased and unable to produce albumin despite good protein intake and amino-acid absorption. Alternatively, is protein being lost somewhere in excessive quantities (perhaps in the urine as in nephrotic syndrome; or in the bowel from weeping chronic ulcerative or inflammatory lesions); or is it being metabolised excessively (e.g. severe fever/infections).

Getting this approach to work for you depends on several important features.

1. Asking general questions before the particular.

Thus, following on from the above, there is no point in wasting time considering a valvular obstruction as a cause of low blood pressure if the cardiac output is normal; and no point in thinking about nephrotic syndrome as a cause of oedema in the absence of ANY evidence of hypoproteinaemia.

2. Keeping your questions and concepts of physiology simple and down to earth. Using a lot of medical jargon may make you sound clever, but if in the process you lose the ability to see things simply, you will have lost your chance to SOLVE the clinical problem from the data in the individual patient. In any event, the use of jargon will obscure your patient's understanding of your questions, and his answers will be correspondingly confused, so that the very clinical 'data-base' from which you draw your conclusions will suffer.

Both of the above require you to understand the BROAD characteristics of organ function. Being able to ask simple questions of the patient and the clinical data in reaching a clinical diagnosis requires being able to see each organ function in its simplest form. For example, the tasks of the gastro-intestinal tract, broadly speaking, are to ingest, digest, and absorb foodstuffs, and for you to see this in its simplicity is as an absolute necessity to understanding the "malabsorption" syndromes. This may sound self-evident, but so many students become lost in the maze of detail here, unable to see the wood for the trees. In a similar vein, the broad tasks of the circulation are to maintain blood pressure and flow, especially to important areas of the body, and it does this by having a pump, and a variable vascular resistance to each organ which can open and close to allow variable blood flow.

Practising solving problems by the game of "Twenty Questions" will help you to go from the broadest to more detailed levels. Have another student give you a (limited) amount of clinical information about a real patient (e.g. with low blood pressure; with loss of consciousness; etc.) and see if you can build up the answer with a maximum of 20 questions.

When you play your game of "Twenty Questions" try not to use more. Again heed Kipling's advice:

> But different folks have different views;
> I know a person small -
> She keeps ten million serving men,
> Who get no rest at all!
> She sends 'em abroad on her own affairs,
> From the second she opens her eyes -
> One million Hows, Two million Wheres,
> And seven million Whys!'

<div align="right">From Kipling's 'Just So Stories'.</div>

It was Jerome Bruner, one of the wiser educationists, who said (of mathematics) that "discovery.......is a by-product of making things simpler". The same is true of clinical diagnosis. And you can simplify your approach to that both by analyzing the clinical information from individual patients within the clinical science subgroups and by seeing the clinical sciences (physiology, anatomy, etc.) for that essence which allows you to build up a diagnosis from the broadest to the most detailed level. If you can do that, you will experience the real joy of DISCOVERY in clinical diagnosis, and of being able to

question traditional wisdom. Thus sometimes, having defined the diagnosis in the four diagnostic categories, you may see that they can interact in unusual and interesting ways: e.g. why is epilepsy of such sudden onset, since the sudden onset events we know of are usually obstruction to hollow tubes, and the only hollow tubes in the brain are vascular ones; is epilepsy therefore due to some sudden reversible vascular occlusion in particular areas of the brain??

But when I talk of simplicity I do not in any way mean dogmatism, fixed rules, or instant judgements, for they should have no part in our method. All that we derive should be in the nature of WORKING HYPOTHESES, even the very guidelines by which those derivations are made.

If, as will happen with experience, you find yourself recognizing disease patterns in individual patients, make sure any label you apply to him/her is accurate and complete, i.e. contains comment in all four diagnostic categories - particularly the Functional and Aetiological ones. Acute (Path.) myocardial (Anat.) infarction (Path.), for example, is not a sufficient diagnosis in the individual patient.

Having established, through your clinical history-taking and physical examination, the clinical data-base, you should then formulate your CLINICAL DIAGNOSIS. Now, sometimes the information will be incomplete, but nonetheless you should try to make a clinical diagnosis. If you are uncertain, jot down a "CLINICAL IMPRESSION" - Provisional Clinical Diagnosis - but you then commit yourself to commenting in four categories. Thus, even a clinical impression should contain an Anatomico-Pathology axis, followed by a comment about Functional consequences (including the severity of the condition, and any sequelae or complications) and then a comment about predisposing and precipitating factors. An example of a complete diagnosis in the such a patient might be as follows:

 Acute (Path.), myocardial (Anat.), infarction (Path.), with the secondary (Functional) consequences/ complications of impairment of left heart function and atrial fibrillation on the predisposing long-term background (Aetiol) risk markers of high blood pressure and heavy cigarette smoking and recent (? precipitating) psychological stress. (Aetiol).

This diagnosis is long, but better long and all inclusive than brief with pieces missing. If you cannot make a complete clinical diagnosis or impression in all categories, at least this method will highlight where your diagnosis is deficient, and this will set the stage for further rational investigation. In this respect, the first investigation should be to take a further history, particularly to fill in background information relevant to the Aetiological diagnosis. When in doubt about this, always ask the patient!

Reference
G. W. Boyd. Education Debate: clinical diagnostic reasoning
Internal Medicine Journal, July 2011

CASE STUDY EXAMPLES

Tutorials

A brief example: A 65 year old recently-retired male hobby gardener (Aetiol. Dx - ?environmental exposure) presents with a one week history (Pathol. Dx - Acute process) of increasing (Pathol. Dx - progressive process) shortness of breath on exertion (Physiol. Dx - ?Dysfunction of CVS or Resp. system), to the point of being unable to walk up a single flight of stairs without stopping (Physiol Dx - severe dysfunction). Associated cough (Anat. Dx - more likely Resp. system); productive of yellowish sputum (Pathol. Dx = prob. inflammatory process); occasional blood streaks (Anat Dx - large bronchi inflamed); three days (Pathol Dx - progressive process) shivers and sweats (Pathol - elevated temp. = confirms inflammatory process) . . . .

So even with this limited amount of information, we can 'rule off' in our minds and say that this man, with background possible environmental exposure, has an acute progressively severe inflammatory condition affecting the respiratory system. Not bad for just a few phrases! Importantly, we have not gone beyond the available information, yet have begun to corner the diagnosis. And we are aware that some of the categories are less complete than others - for example we need to know more about the precise anatomical localisation of the respiratory problem (Where?) and about the background hobby (Why?)

In **summary**, the 'Tutorials' under the menu-bar aim to help you gradually synthesize a diagnosis through assigning each phrase of the case to an appropriate category as the history and exam findings unfold, and then seeing how the various observations, inferences and sub-conclusions interact. The nature of the WHERE, WHAT, HOW, AND WHY columns should now be clear. Remember that the **WHY** column includes the important psycho-social aspect of **WHO** has the illness, as well as the often-related question of why the condition occurred **WHEN** it did. (See Online Book - General Introduction).

The Online Book.

The initial chapters of this Online Book (see menubar) aim to help you make **clinical** diagnoses in the several categories of Anatomical, Pathological, Physiology, and Aetiological diagnoses. Later, we will consider the biochemical as well, particularly in areas where biochemistry rather than pure physiology gives the clue to the nature of functional disturbances. This is particularly true in disturbances of acid-base, in patients with electrolyte problems, and in delineating the site and nature of maldigestion/malabsorption disorders.
Cases are available **for 'four column' diagnostic problem solving** at the end of **each online book chapter**. Having grasped the essence of the the 'four column' approach to diagnosis from the online tutorials (see below), it is recommended that you **work through these problems yourself,** gradually building up inferences, and interim conclusions to an eventual overall four-columned diagnostic conclusion. **Graphic solutions** are then available for comparison.
But do the **'Tutorials' first**.

Using the Tutorials

1. The Quickstart guide outlines the method. Consult this before attempting the tutorials

2. The tutorials. Start with the 'Start Here' tutorial. The reason(s) for the author choosing a particular categories are especially well detailed in the initial paragraph of this particular tute.

3. 'Appropriate' Categories. There may be more than one category relevant to any one observation.

4. Nothing is absolute. The allocation of observations to categories is not meant to be defined by rigid rules. Therefore, you should feel free to differ from the author's suggestions at any stage. There is nothing absolute here. Indeed, if you have important suggestions, please email the author - see 'menu bar'. This will always be a work in progress.

5. Other methods. Along the same lines, the method here is not meant to be the only way of reaching a diagnosis. As you gain experience, you will sometimes recognizepatterns with parallels to previous cases, and so be able to diagnose by 'pattern recognition'. At other times, when you are stuck, it will help if you can find some analogy, maybe from life quite outside your medical experience, something the present condition is like. That can sometimes lead to real discovery, as in the way William Harvey was able to see the heart as a pump, rather than the previous furnace-like model of Galen from 1400 years before!

We now go on in the book to look at the clinical physiology of a number of clinical situations. Often, in order to begin to understand many diseases we have to discuss the physiology of named conditions. But we will do this merely to provide a background setting for what really matters to us in the final analysis, viz. making our knowledge of clinical physiology work for us in building a clinical diagnosis in our **individual** patients.