1 Introduction: The Skeleton Laid Bare

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This chapter discusses the basic layout of this book
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1.1 THE BONES OF THE BOOK

Clinical reasoning is an enigma that has been the subject of research over the last few decades. It pertains to how physicians not only arrive at a diagnosis, but then use their clinical judgement to decide the next best course of action. This could be ordering another test, initiating treatment or the most curious course of just observing and not acting at all.

Current thinking revolves around the dual processing theory, which is an amalgamation of all the research thus far. It incorporates analytic and non-analytic strategies of clinical reasoning, which interact at different phases of the patient encounter and are called into play when needed. Non-analytic strategies (unconscious/reflexive) include pattern recognition, heuristics, illness scripts, and semantic qualifiers. Analytic strategies (conscious) include causal reasoning and probabilistic reasoning, where logic and critical thinking are given importance. Meta-cognition, an awareness of one's own thinking, overarches the analytic and non-analytic processes of cognition directing the clinician to the diagnosis.

An example in action:

An 82 year old lady presents with acute confusion. The doctor, using pattern recognition and heuristics (mental shortcuts) thinks this is likely to be a urinary tract infection (UTI), because he has seen this all too often. He notes the lady was on warfarin, so wonders if he is missing something (meta-cognition). He telephones her carers querying any recent falls with head injuries (analytic strategies). It turns out she had a head injury a week ago, following which she became increasingly confused and drowsy. This leads him to a working diagnosis of subdural haematoma, which gets confirmed on a CT scan.

If he had not been consciously aware of his own thinking (meta-cognition) he would have settled on the diagnosis of a UTI and ascribed a raised white cell count and low-grade temperature as confirmatory – thereby missing a significant diagnosis that carried a greater burden on the patient concerned.

You could argue that an experienced clinician would have got this diagnosis right first time. However, there are several contextual factors at play, which can easily mitigate in-depth analysis. Patient factors, such as an acutely confused person unable to give a clear story; environmental factors such as a busy A&E department and physician factors such as fatigue and sleep deprivation can all impact the decision-making process, leading to an unpleasant outcome for all concerned. Remember that experience does not equate with expertise.

Norman (2005) has suggested that clinical reasoning can only be imbibed by 'deliberate practice' wherein the learner encounters a plethora of examples, rather than just learning the strategies of clinical reasoning. In other words, practice, practice, and more practice will develop you into a skilful clinician. You can read this book to master the strategies of clinical reasoning, but unless you put them into practice, it will continue to remain an enigma.

The Hands-on Guide to Clinical Reasoning in Medicine, First Edition. Mujammil Irfan.

Companion website: www.wiley.com/go/irfan/clinicalreasoning

This book has been divided into sections relating to the clinical placements you may find yourselves in. This allows you to work with the book whilst on your placements, transferring knowledge into practice. The topics include those often felt to be poorly covered, and are a treasure trove of common conditions that you will encounter.

The book does not claim to be an exhaustive resource on clinical medicine, but rather a route map, showing the intricacies of clinical reasoning. I shall start with a personal perspective of some rules-of-thumb for diagnostic reasoning, followed by rules-of-thumb for decision making to guide investigations and treatments. This will be followed by a unique way of approaching patients that should make your life a lot easier.

If there is one thing I would like you to take from this book, it is to always be open to diagnostic possibilities, ensuring that the thinking process never stops.

Rules of Thumb for Diagnostic Reasoning – A Personal Perspective:

1. **Commit to a diagnosis**
   'Collapse query(?) cause' is a common colloquial term in UK practice amongst junior doctors and is touted as the diagnosis for someone presenting with collapse. This is not a diagnosis. All you are doing is elaborating the fact you do not know the cause of their collapse. The first step in learning to diagnose is to commit to a diagnosis. We all make mistakes along the way, but not committing to a diagnosis is cognitively far more dangerous than making one and learning from it – as long as it does not put a patient at risk. If in doubt, ask a senior clinician for help in making those mental connections, but make sure you at least have a working diagnosis. Occasionally, a diagnosis maybe elusive, in which case a plan of action still needs to be formulated whilst acknowledging uncertainty and ensuring follow-up. Often, diagnoses emerge in the fullness of time, hence adequate follow-up is essential.
2. **Link to the past medical history**
   When trying to make a diagnosis, remember that any presentation in medicine is usually linked to the past medical history or medication list. When that train of thought does not yield a diagnosis, a new diagnosis should be entertained. If someone is known to have ischemic heart disease, they are likely to be breathless because of that than due to say, 'Churg-Strauss syndrome.'
3. **Common things are common**
   Use disease prevalence as a yardstick to know what is common. Epidemiologically speaking, a middle-aged male smoker in the developed world is likely to have vascular risk factors such as hypertension, hypercholesterolemia, and diabetes mellitus, predisposing him to ischemic heart disease and strokes.
4. **Explain the symptoms**
   Patients seek help because they are having symptoms not because they have an abnormal electrocardiogram, test result, or radiograph. Hence always try to explain the symptom/-s, and you'll hit the diagnosis.
5. **Explain all the findings**
   Can you explain all the findings (history, clinical examination, and investigations) with the diagnosis you have made (Kassirer and Kopelman 1991)? If there are any unexplained findings, re-visit the diagnosis.
6. **Think of all the alternatives**
   Always pause just before you make the final diagnosis and think of all the alternatives that can present in a similar fashion. Rule them out consciously before accepting the favoured one.
7. **ABC buys you time**
   All treatment, from intravenous fluids and antibiotics, to intensive care, is a temporary holding measure to buy time and allow the body to recover. The way you do it is by stabilising the physiological parameters, thus buying time to make a diagnosis. The ABC (airway, breathing, circulation) of emergency medicine is just this.

Rules of Thumb for Management Plans – A Personal Perspective

1. **Risk vs. benefit ratio**
   This should form the basis for decisions regarding patient management, including investigations.
2. **Mortality and morbidity**
   Most interventions in medicine are designed to prolong life (improve mortality) or reduce suffering (morbidity). Hence, the best treatment (anchored on evidence-based medicine) should improve mortality, and the second best should reduce morbidity. Of course, quality-of-life issues and patient choice trump all of this, but again, symptom control (alleviating morbidity) plays a big role even here.
3. **Will it alter my management?**
   Before ordering any test, be it a blood test or an MRI scan, ask yourself 'Will it alter my management?' This will ensure you do not do unnecessary tests.
4. **Masterly inactivity**
   Not intervening can also be a part of your management, e.g. observing a patient to see how their disease evolves before invasive tests are ordered or treatments initiated. This skill requires expertise – hence the phrase 'masterly' inactivity.
5. **Patient autonomy**
   Patients' informed decisions of not having further tests or treatments are to be respected at all times – despite how bizarre they may sound.

1.2 HEURISTICS

Some of the points elaborated above are called mental shortcuts or heuristics. Physicians use these to develop hypotheses – especially when confronted with incomplete information. They form part of the non-analytic strategies at the discretion of a clinician. Knowing when to use them and when to avoid them is a skill we must develop. When heuristics lead you down the wrong diagnostic pathway, we label them cognitive errors or biases (Croskerry 2002, p. 1201). With experience you will develop your own heuristics, but make sure they are based on accurate clinical knowledge (e.g. use disease prevalence to know what is common) and not faulty reasoning. This will ensure they do not turn into cognitive biases.

1.3 CLINICAL REASONING IN ACTION

When a junior doctor is presenting someone with acute central chest pain to the Consultant Physician, the latter is paraphrasing the information into digestible chunks, and listening intently to elicit whether the pain is pleuritic, positional, or exertional. The junior doctor may well have got lost in the sea of information ascertained from the patient, but the Consultant just picks what is relevant. You too can learn to do this. The starting point is to paraphrase the presentation using precise medical terms. The chunks of relevant information that you paraphrase from the data are called semantic qualifiers (SQs).

Allow me to illustrate:

A 56 year old man presents with a one hour history of right-sided weakness. This developed suddenly whilst sitting in a chair. He is a 30 pack year smoker and drinks 40 units of alcohol per week. He has a history of hypertension, diabetes mellitus, and hypercholesterolemia. He takes ramipril 2.5 mg od, gliclazide 80 mg od, and simvastatin 40 mg od.

Semantic Qualifiers
Middle aged man + acute neurological deficit + vascular risk factors

A *middle aged* man presents with an *acute* onset right sided weakness on a background of *smoking* and *alcohol excess*. He has *vascular risk factors* including hypertension, diabetes mellitus, and hypercholesterolemia.

A middle aged man with vascular risk factors presenting with an acute (sudden onset) focal neurological deficit is very likely to have had a vascular event. I'm thinking he has had a stroke. This is one of several possibilities, but we have made a start (Figure 1.1).

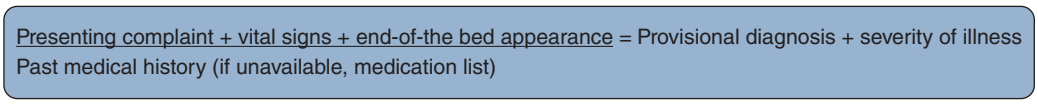


Figure 1.1

You see what I did there? Paraphrasing the data into chunks lets you pick the relevant details and thread them into a coherent line of thinking.

We use these chunks to create a working space or 'context' which in this case is 'a neurological problem.' This is then refined in light of further history, examination, and so on.

We shall be using this technique throughout this book and hopefully you will learn to incorporate it into your daily practice.

1.4 ARRIVING AT THE PROVISIONAL DIAGNOSIS

Having paraphrased the clinical problem into meaningful chunks I then use a combination of vital signs and end-of-the-bed appearance (a 'bed-o-gram' in common parlance) to give me a measure of physiological derangement and the rapidity with which I need to formulate a working diagnosis (Figure 1.1).

Using the example above, his vital signs read: HR 110 bpm, BP 180/90, Temperature 38 °C, RR 28 per minute and Saturations of 92% on air. To this I normally add blood sugars (BMs), which read 'low.' He has marked physiological derangement with a strikingly low blood sugar. I combine this marked physiological derangement with his end-of-the-bed appearance – he appears drowsy and confused, and conclude that he is 'very ill.' I need to act *quickly* (translation: 'rule out life-threatening diagnoses first'). Life-threatening diagnoses in this case would include a stroke, low blood sugars causing neurological symptoms (neuroglycopaenia) (McAulay et al. 2001) and subdural hematoma due to history of alcohol excess (although the acute onset makes this unlikely). Life-threatening conditions need timely treatment and a delay in diagnosis will put your patient on the slope of deterioration that can be fatal.

The astute amongst you may have noticed our initial suspicion of stroke is now being called into question with more data (Figure 1.2). This is a reflection of the real world. We must keep an open mind to all possibilities before we accept any particular diagnosis. Premature closure is something we should be wary of.

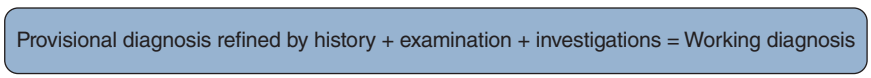


Figure 1.2

Obviously, correcting the hypoglycaemia would be the first step but I would not rule out a stroke just yet. If the symptoms resolve with a normal blood sugar then you have confirmed your diagnosis, if not you request a CT scan of his head to rule out a stroke. Remember to constantly re-visit your diagnosis and be prepared to change it if new data demands (Figure 1.3).

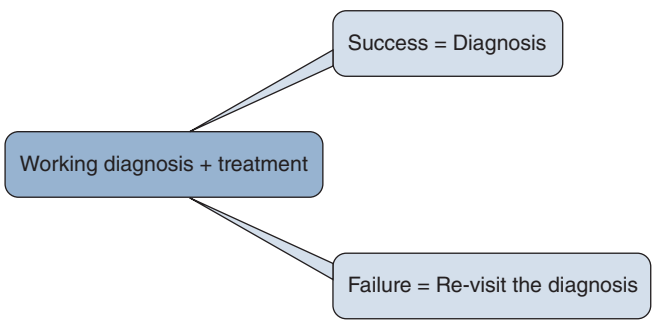


Figure 1.3

Our next task is to explain all the findings. I wonder why he has a low blood sugar. Why are his vital signs so deranged? Are we missing something? To recap: HR 110 bpm, BP 180/90, Temperature 38 °C, RR 28 per minute, Saturations 92% on air and BMs 'low.'

This can be overwhelming, but is truly very easy. I want you to now look at the beautiful orchestra of physiology, which is trying to tell you the diagnosis. His RR is high – there is something wrong physiologically; his saturations are low – there must be something wrong with his 'gas-exchange area' (the alveolar – capillary interface of the lung). I must ask if he has had any chest symptoms (breathlessness, chest pain, cough, etc.) and examine his heart and lungs. His blood pressure is raised at 180/90, with a pulse of 110 bpm indicating sympathetic activation. He must be sweaty and clammy too (the inner Sherlock speaking). This could just be a response to the hypoglycaemia, or to the neurological deficit. But, why is his temperature raised? Has he had any infective symptoms? Or is it secondary to the neurological problem?

It turns out he has had a cough with discoloured phlegm for the last four days with increasing breathlessness, lethargy, and poor oral intake. I seem to be localising a problem to his lungs. I listen to his chest – he has signs compatible with consolidation. A CXR confirms my suspicions. An ABG reveals hypoxia and metabolic acidosis – the latter secondary to sepsis and acute renal impairment detected on the bloods.

The sequence of events seems to be:
Pneumonia → acute kidney injury and sepsis → ↓BMs (secondary to poor oral intake + accumulation of gliclazide due to renal impairment) → new focal neurological deficit.

He improves with intravenous glucose and antibiotics and is discharged five days later.

Thus far we have discussed the six basic principles of clinical reasoning. To recap:

1. Frame a 'context' upon first contact with the data. Use semantic qualifiers to paraphrase the data and come up with a provisional diagnosis/-es.
2. Use vital signs and end-of-the-bed appearance to establish the severity of illness and elicit inconsistencies between the data and your provisional diagnosis.
3. Refine this with further data from history and clinical examination to arrive at a working diagnosis.
4. Outline further investigations and/or treatment based on this working diagnosis.
5. Re-visit the diagnosis in light of investigations and treatment responses.
6. Above all, do this consciously with a thought-provoking monologue so people around you can correct any faulty models of thinking.

1.5 ASSESSING SEVERITY OF ILLNESS

Over the years I have learnt that there are four kinds of patients: well, ill, very ill, and dying. There is a fifth kind – 'dead,' but really we are trying not to get there. How do I come to this conclusion? I'll give you a simple yet comprehensive way of assessing patients to tailor your history taking and examination and come up with a diagnosis or reasonable differential – all in a timely fashion. Oh, you are in for a treat.

The trick in assessing how ill a person is, is to look at the individual physiological parameters. The greater the deviation of the physiological parameters from the mean, the sicker the patient is. Then match this to the end-of-the-bed look of the person to get a fuller picture. This tells you if they are well, ill, very ill or dying.

Severity of illness	Time for assessment (rough guide) (min)
Well	30
Ill	20
Very ill	15
Dying	30

Picture a bleep from a staff nurse on the wards. She informs you that her patient has a heart rate of 150 bpm. This is grossly abnormal. However, on arrival to the ward if you find said patient sat reading a newspaper, he is 'well' (relatively speaking). You have time to find out why he is tachycardic, take a history, examine him, and get some tests. Conversely, if you find him feeling lightheaded with a slightly low BP (100/70) you class him as 'ill.' You will have to be a bit quicker here, but you still have time.

On the other hand, if you find him lying in bed looking grey and complaining of chest pain, he is 'very ill' and if you don't act quickly, will soon be 'dying.' Eliciting that he is

very ill gives you the severity of illness. It tells you to be very quick, take a focused history, do a targeted examination and get an urgent ECG *whilst* you are doing the aforementioned. So, you see vital signs or even history taken out of context can be misleading. Until and unless you have cast an eye on the patient, you cannot say how ill this person is.

A subtle component of this end-of-the-bed-o-gram is to assess the functional impact of the symptoms. This is most helpful when you do not have a set of observations guiding you – as in General Practice. Daily symptom burden impacting activities of daily living would class the person as ill or very ill depending on the degree of functional impairment. This is something I would like you to start doing. Try classing every patient you come across into either of these categories just by their end-of-the-bed appearance. This is a very subjective assessment, but comprehensive nonetheless. Used on its own it can be fatal, but if used in conjunction with other data, it is priceless.

Okay, now for some questions:

What's the first physiological parameter that goes off when someone is ill (I mean ill due to any reason)? Hazard a guess? No peeking….

Activity 1.1

(Allow few seconds)

Respiratory rate.

Two unique things about respiratory rate – it's the only sign under voluntary control and the only parameter which is measured manually, and most likely to be left out. So, all those calls from nurses which say 'I do not like the look of him,' may have an underlying sign of clinical instability where the respiratory rate is abnormal (normal range 8–18).

1.6 THE SCIENCE IN THE ART OF MEDICINE

A 60 year old man sees his GP in the UK with a one week history of fever and cough. The clinician wishes to exclude the possibility of community acquired pneumonia (CAP) and takes a history and performs a clinical examination to increase or decrease their suspicion of CAP. The prevalence of CAP in the general population in the UK is 0.035 (British Lung Foundation data 2012). In practical terms, if no clinical assessment is carried out, the disease prevalence is equivalent to the pre-test probability of the disease in question (Figure 1.4). However, it is important to remember that this varies with the context in which this patient is seen. In Primary Care, the pre-test probability is lower than in a hospital where the population has been filtered.

Positive LR = sensitivity/1 – specificity.
Negative LR = 1 – sensitivity/specificity.

During clinical assessment, each clinical finding (sign or symptom) increases or decreases the probability of CAP – some more so than others. The degree to which this suspicion is shifted can be measured quantitatively using the likelihood ratio (LR). LR is a measure of the diagnostic weight each finding carries. Disease prevalence does not affect sensitivity and specificity, but disease severity does (Parikh et al. 2009). Since LR is derived from sensitivity and specificity, it is unaffected by disease prevalence. Figure 1.4 conceptualises how LRs shift the post-test probability towards or away from CAP.

The 'test' in this case is a clinical sign.

1. Prevalence ~ pre-test probability prior to clinical assessment.
2. Pre-test odds = pre-test probability/(1 – pre-test probability) = 0.035/1 – 0.035 = 0.036.
3. Post-test odds = pre-test odds × LR_1 × LR_2 × LR_3 × ….LR_n = 0.036 × 2.4 = 0.087.
4. Post-test probability = post-test odds/(1 + post-test odds) = 0.087/1 + 0.087 = 0.08 which is 8%.

For instance, the positive LR for pyrexia is 2.4, raising the post-test probability of CAP to 8% (see opposite for calculation (Parikh et al. 2009) or use a nomogram (Fagan 1975)). If

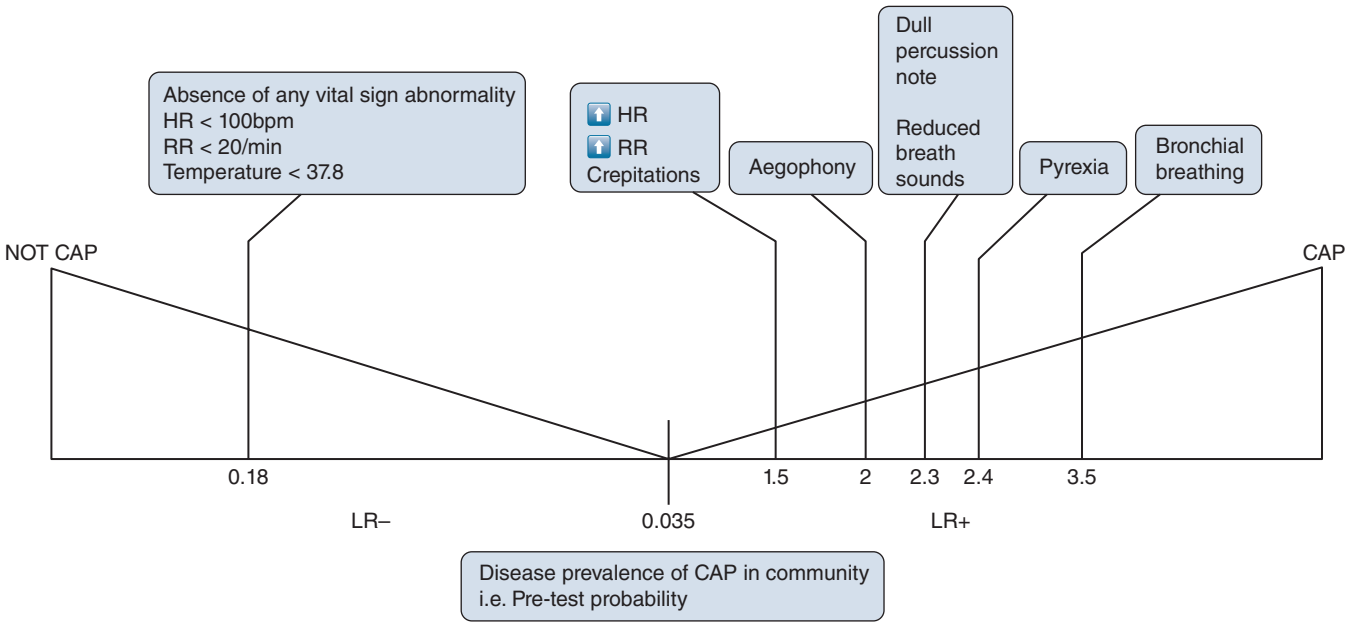


Figure 1.4 Likelihood ratios as diagnostic weights (not to scale)

the clinician gives undue importance to finding crackles alone (LR 1.6), the post-test probability then rises to 12%, which is still low to justify using antibiotics.

From literature, the LRs of all the clinical signs of CAP are as shown in Figure 1.4 (Metlay et al. 1997). Incorporating all these into the assessment will raise the post-test probability to 91.5%. In this scenario, even if the CXR does not show consolidation, we would go ahead and treat. Indeed, studies have shown that if the initial CXR is normal, consolidation can often 'blossom' following rehydration, becoming visible in the next day or two (Feldman 1999).

Let us say, the post-test probability following clinical assessment was 50%, the clinician can now choose to order a CXR. Once again, tests have sensitivity and specificity and therefore a LR. The LR for CXR is 6.21 (Self et al. 2013). If the CXR shows consolidation, the post-test probability rises to 86% from 50% helping us make treatment decisions. Remember, *tests do not make the diagnosis* – they merely increase or decrease the probability of the disease in a similar fashion to clinical assessment.

Conversely, if there is no abnormality in the vital signs, the post-test probability of CAP is <1% making it unnecessary to get a CXR (Gennis et al. 1989).

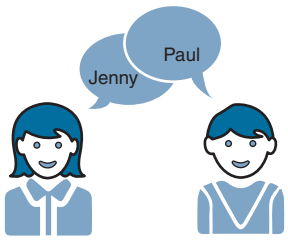
These calculations are individualised to the context in which the patient is encountered, providing truly personalised medicine. Seasoned clinicians often do all of the above intuitively without resorting to formal calculations, but even they can sometimes err in quantifying the true disease prevalence.

Cooper and Frain (2017) have simplified the approach to memorising the change in diagnostic probability according to the value of the LR. A LR of 1 implies no change in the diagnostic probability of the disease in question. The higher the LR, the greater is the probability of the disease. LRs of 2, 5, and 10 correspondingly increase the disease probability by 15%, 30%, and 45%. Similarly, by inverting the numbers 2, 5, and 10 we get LRs of 0.5, 0.2, and 0.1, which reduce the disease probability by 15%, 30%, and 45%. A positive LR implies that the clinical finding is present, whilst a negative LR implies it is absent. One limitation of a LR, is that it cannot be calculated unless the disease has a conclusive technological diagnostic standard (in this instance, a CXR).

There are several online databases showing prevalence of diseases, as well as giving LRs for common clinical findings/tests in various diagnoses. You can easily incorporate them into your practice using apps too. At the very least, they can be useful in grey cases where there is diagnostic dilemma.

Another concept to understand is the predictive value of a test. If a positive test is able to pick up everyone with disease, it has a high positive predictive value (PPV). Similarly if a negative test is able to exclude everyone without the disease, it has a high negative predictive value (NPV). However, these values vary with disease prevalence, e.g. the lower the disease prevalence, the lower the PPV. In other words, if the pre-test probability of disease is low, no matter how good a test, a positive result is less likely to rule in disease. This is why 'fishing tests' without knowing what the diagnosis is, do not give us the answer.

1.7 STRUCTURE OF THE BOOK



With my thinking cap on, I imagined various methods in which I could enable your learning. Everything seemed to be a drag but one. The best way of going about it was to imagine a couple of students in front of me to whom I would teach the art of clinical reasoning. Better still, what if I were to materialise two students right now? Meet Jenny and Paul, two fourth-year medical students henceforth known as J and P. They are going to be learning the basic principles of clinical reasoning, and you are most welcome to join us. Who knows, I might be able to help you and if I do I shall consider myself to be very privileged.

My aim is to paint as real a picture as possible, mirroring the real world. That includes all the inaccuracies, complexities, and fun that real life would present to us. I have assumed a fair amount of background knowledge in clinical diagnoses for you to be tackling the content of this book. If you fancy, you could have a quick revision in the Common Clinical Conditions section before tackling the rest of the book.

As we walk along, there will be plenty of space to record your thoughts and I hope you will avail of this. Feel free to use the margins in the text to record your reactions (clear, unclear, need more examples, diagrams, etc.) as you go along.

I shall begin by introducing new concepts through case based scenarios, showing my thought processes in brackets. My conversations with Jenny and Paul will be in 'CronosPro,' whilst they will converse in '*italicised CronosPro*.' The conversations with you (the reader) will be in 'Times New Roman.' I hope that the principles learnt here will be applied elsewhere in other specialties and not limited to Medicine.

The activity boxes are designed to give you a pause in your reading, allowing you to engage with the material. They are a resource in themselves and have a valid rationale. The time needed to complete them has been indicated, giving you the choice of doing them or not depending on how stretched you are. Some of these activities have no specific answers. This is done deliberately to make you think broadly, and is a reflection of clinical practice where there is often no single best answer, and entertaining several possibilities will ensure you do not miss diagnoses.

I can sense some of you are getting impatient and wondering if I'm ever going to start. I can't stop you from skipping parts of this book, and I don't intend to – you are an independent individual and are free to use this book the way you want. Once you have completed the book you can use the accompanying Reflective Action Guide. Print out the sections relevant to your placement and take them to the wards, hopefully transferring your learning into practice.

Enjoy!

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