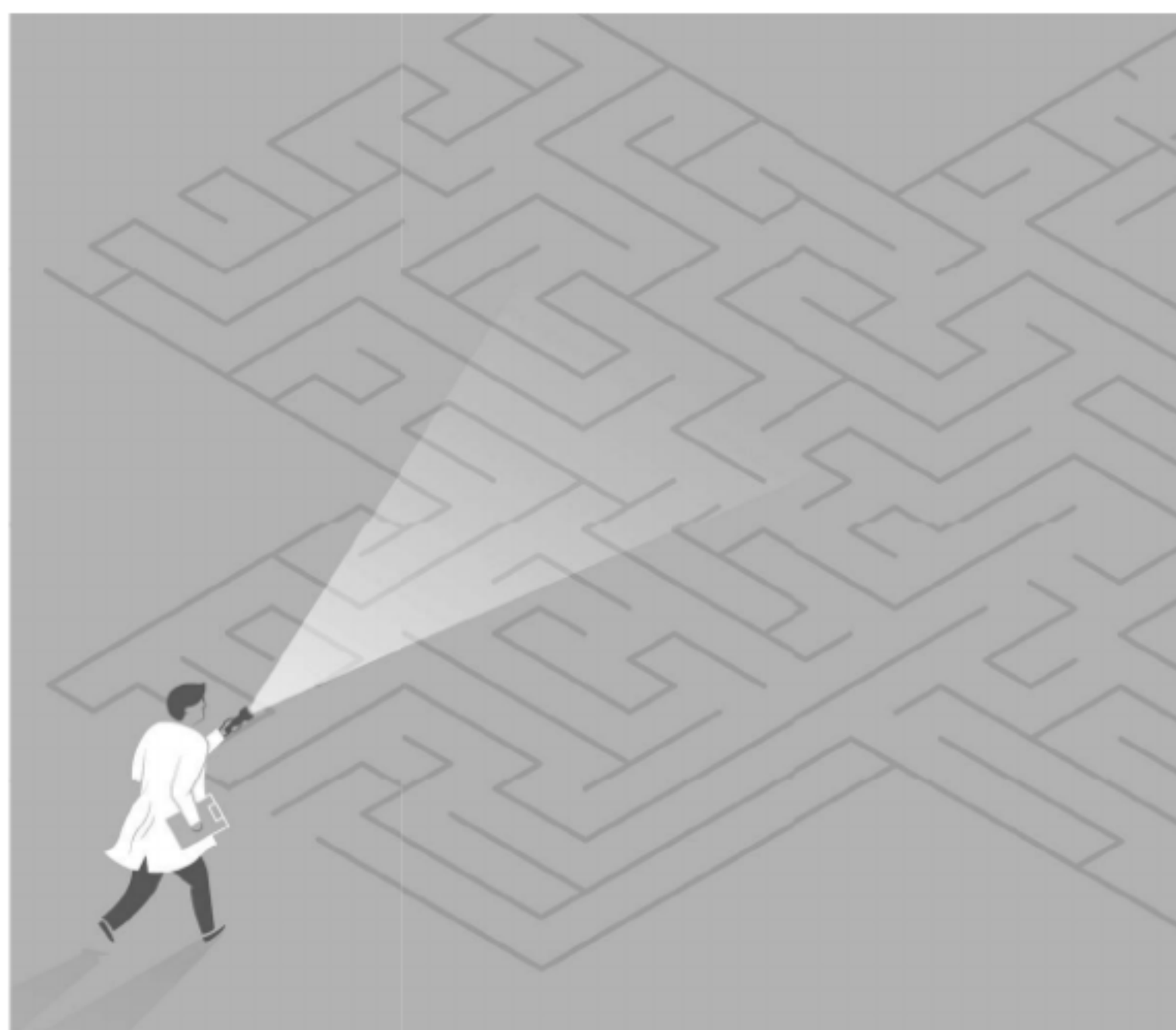



# Algorithms in Differential Diagnosis

How to Approach Common Presenting Complaints in Adult Patients,  
for Medical Students and Junior Doctors



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# Preface

This is a book for medical students and first-year doctors who wish to learn how to approach a patient's symptoms, and sharpen their skills of clinical reasoning and diagnosis.

Clinical medicine begins with the patient. Many of us learn one disease condition at a time, yet, patients present with symptoms and not a known disease. Many students know the correct evidence-based treatment of heart failure, yet are stumped when given a breathless patient on the ward, and told to 'figure out what is wrong'. Many can list the differentials for a symptom, but struggle to separate important diagnostic features from the irrelevant details that many patients throw at us. It is tempting to rely ever more heavily on our growing armamentarium of diagnostic tools. But testing without thinking not only confounds and misleads, it also costs our patients dear.

Having been a student myself not too long ago, I have experienced first-hand the struggles that a budding clinician faces in synthesising vast amounts of new information and applying it to real patients. In this book, I try to offer a toolkit to tackle this challenge.

Each chapter tackles one presenting complaint, identifying key differentials, providing a strategy to distinguish each differential from the other and setting out the thought process behind history, examination and initial investigations. Each approach cuts across different specialties, integrating approaches and conditions from various medical and surgical fields.

This book uses *algorithms* to aid diagnosis. This is a method of clinical reasoning that uses critical pieces of information as branch points to distinguish between groups of diagnoses. It complements (not replaces) clinical skills and knowledge of individual disease conditions. Junior diagnosticians particularly benefit from learning algorithms, because they not only provide a systematic and functional way to approach patients, but also serve as a scaffold to organise knowledge and learn the skills of clinical reasoning. I have written these algorithms to be usable for those who are just starting out, rather than theoretically complete but too complex to use.

Finally, always remember that if one hopes to develop clinical acumen, there is no substitute to seeing patients—they are our best teachers, inspiration and sources of creative inquiry.

**Nigel Fong**

2018

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Most of all, I thank my patients, who time and again have proven to be my best tutors, and who have given medicine its meaning.

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# Introduction

# Chapter 1 Learning How to Diagnose

## Clinical Case

*A final-year medical student has difficulty making clinical diagnoses. Although she is able to regurgitate a great deal about many conditions (having read the textbooks cover-to-cover), she has great difficulty pinpointing the correct diagnosis whenever she sees a patient on the ward. She says that she ‘does not know where to begin’, and ‘just can’t think of the right diagnosis’. What would you advise her?*

Welcome! We must begin by learning how to diagnose. These skills are central to a physician’s healing art—making an accurate and timely diagnosis allows the physician to administer appropriate treatment, which gets the patient better. Conversely, diagnostic errors harm patients.

This chapter opens with an exposition of how clinicians make diagnoses, and discusses the process of diagnostic reasoning, along with some other issues. Next, I offer some advice on how to use this book, and suggestions on how to learn the science and art of clinical diagnosis. We will use the clinical scenario (Box 1.1) as a starting point for discussion.

### Box 1.1. Diagnostic Reasoning During Ward Rounds

**Third-year student:** Our next patient is a 70-year-old gentleman who complains of jaundice. It started 1 month ago, and has been getting worse. He does not have fever, abdominal pain, nausea or vomiting. He has been feeling lethargic and has also lost 10 kg in the last 3 months. His bowel movements are normal. He is a non-smoker, and has no travel or contact history. A sexual history is unremarkable. He has no past medical history. On examination, his abdomen is soft and there is no palpable liver. Liver function tests show elevated bilirubin, AST, ALT and ALP.

**Consultant:** What do you think it could be?

**Third-year student:** Hmm... does he have cirrhosis? He has jaundice and abnormal liver enzymes. Hepatitis? Haemolysis? Gallstones?

**Final-year student:** I’ve clerked him too. He has no stigmata of chronic liver disease, so cirrhosis is unlikely. I don’t think he has haemolysis either, as his haemoglobin was normal. He has no abdominal pain, history of eating raw shellfish, unprotected sexual intercourse or hepatotoxic drug ingestion, so it’s probably not hepatitis. His liver function test is actually

(Cont’d)

showing higher ALP than AST and ALT, suggesting biliary obstruction, maybe by gallstones or a mass.

**Resident:** Yes, this is a gentleman with obstructive jaundice. The causes of obstructive jaundice can be divided into painful and painless. He is painless, which makes gallstones less likely, and extrinsic compression of the bile ducts more likely. I would order an ultrasound next, looking for dilated bile ducts. If ducts are dilated, then we need to find out if there is biliary tree compression by an extrinsic mass, or if he has a stricture. If ducts are not dilated, then we should send off antimitochondrial antibody for primary biliary cholangitis.

**Consultant:** Alright. This is an elderly gentleman with painless progressive obstructive jaundice. You are right that in the absence of pain, bile duct compression is more likely than gallstones. Strictures and primary biliary cholangitis are possible, but in this patient with clinically significant weight loss, I am most worried about a pancreatic tumour with bile duct compression. Let's skip ultrasound and go straight to CT. That will tell us whether there is any pancreatic mass.

## How Do Clinicians Diagnose?

Observe how each person in the ward round (Box 1.1) employed a different method of clinical problem solving. The third-year student was guessing, and had little chance of arriving at the correct diagnosis, except by fluke. The final-year student was testing one hypothesis at a time until he could find one to match the available information (*hypothetical-deductive* reasoning). The resident was using an *algorithm*, which divided the causes of jaundice based on key predictors (e.g., pre-hepatic, hepatic and obstructive jaundice; painful and painless). The expert clinician effortlessly *recognised this pattern of symptoms*, and synthesised his intuition with analytical decision rules (painful vs. painless). Understanding the diagnostic thought process is fruitful for all who wish to learn clinical diagnosis.

## Hypothetical-Deductive Reasoning

Hypothetical-deductive reasoning is about generating and evaluating differential diagnoses. Every medical consult is an iterative process of information gathering, data interpretation, hypothesis generation and testing. Hypotheses so generated prompt a search for additional information (further history, examination or investigation); in turn, additional information may confirm or refute existing hypotheses, or trigger the generation of new hypotheses.

- (a) **Hypothesis generation:** Do not simply take a history and examine the patient. Think! Think of: (i) what are the most likely diagnoses, and (ii) what are the most dangerous/important diagnoses that must not be missed (even if they are less likely).
- (b) **Evaluating differentials:** Critically examine the differentials generated (Table 1.1). For every hypothesis, ask: (i) what information fits? (ii) what does not fit? (iii) what did I expect but could not find? Then rank the diagnoses in order of likelihood. Next, ask: (iv) what additional information do I need to confirm or refute this

**Table 1.1. Hypothetical-Deductive Reasoning in a 60-year-old Presenting with Shortness of Breath and Wheeze**

Hypothesis	What information fits the hypothesis?	What does not fit?	What did I expect but not find?	What additional information do I need?
Asthma	Acute-onset wheeze	Unusual to have new-onset asthma at 60 years old	There is no precipitating trigger	Is there an atopic history?
COPD	Smoker	Should be chronic, not acute		Is the chest X-ray hyperinflated?
Heart failure	Pedal oedema can cause wheeze		He has no known heart disease	Are there q-waves on his ECG?

hypothesis? This prompts a search for additional information (whether further history, examination or investigation), which would further allow one to distinguish between the possible differentials.

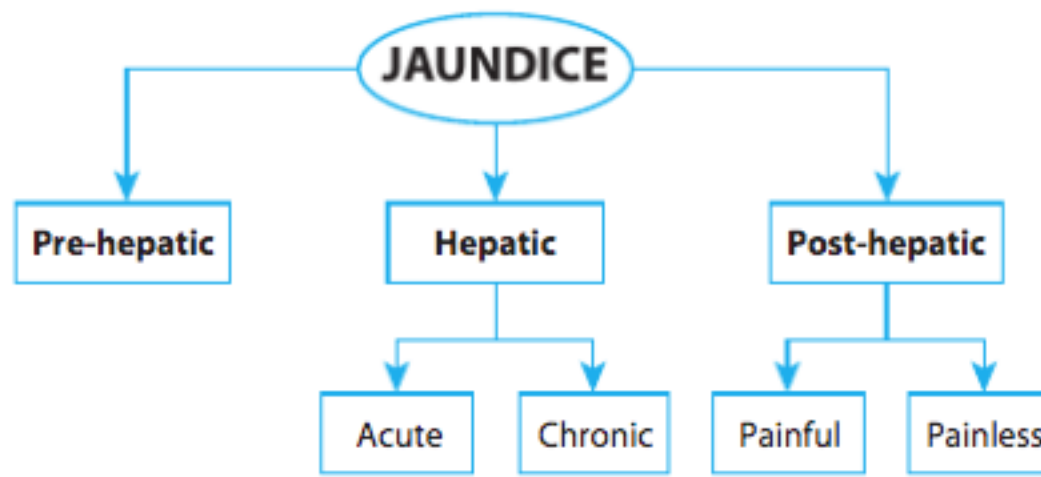
**How to use:** The ability to critically examine a list of differentials, rank them according to likelihood, and pick up ‘what does not fit’ is very important. *Drawing this table for every patient seen is a useful exercise in clinical reasoning.* Additionally, having a list of differentials guides physical examination (you can only find what you look for) and investigations.

**Caveats:** Generating differentials based simply on ‘what I can think of’ is a potential source of error, as the correct diagnosis may never be considered. Conversely, going through every possible differential for a certain symptom is inefficient and impractical. Therefore, while the hypothetical-deductive model is a helpful tool to evaluate differentials and identify ‘what does not fit’, it is best to augment this approach with a method to consistently generate the most important differentials.

## Algorithms

Algorithms employ differentiating pieces of information (history, physical signs or investigations) as key branch points to distinguish between groups of diagnoses. The clinical consult begins by identifying the appropriate algorithm to use. Thereafter, information gathering is guided by the algorithm (Figure 1.1), with particular emphasis on deciding between diagnostic groups as made explicit by the branch points. After several branching points, only a small number of possible differentials are left, and hypothetical-deductive reasoning may be used to rank the remaining options.

**How to use:** Algorithms systematically and rapidly identify likely differentials, helping to reduce cognitive load and the chance of not considering the correct diagnosis. They reflect an organised knowledge structure. This book is about helping you develop algorithms and organise your knowledge, so that you may be able to use algorithms in your diagnostic process.



**Figure 1.1. A simple algorithm for jaundice.**

See Chapter 10 for complete algorithm.

**Caveats:** Algorithms are best used in conjunction with the critical lens of hypothetical-deductive reasoning. Algorithms identify the *most likely* diagnoses; the most dangerous diagnoses (that have to be excluded) may need to be considered separately via a hypothetical-deductive approach, even if deemed less likely by the algorithm.

## Pattern Recognition

All of us can instantaneously recognise people we know, without conscious or effortful thought. In the same way, clinicians may instantly recognise a patient's diagnosis,<sup>1</sup> with little deliberate thought. This is an intuitive process involving matching the new patient's problem against similar ones solved previously. The ability to do so requires accumulation of 'illness scripts' (previous patients seen and mental prototypes) through clinical experience, as well as a well-organised knowledge structure so that correct matches can be retrieved rapidly based on salient cues.

**How to use:** To use pattern recognition, you must have accumulated a database of illness scripts through clinical experience, and organised your knowledge structure via deliberate reflection on patients seen. Furthermore, illness scripts are condition-specific (having seen thousands of fractures does not make one better able to diagnose chest pain) and context-specific (having seen 10 sick pneumonia inpatients does not help one diagnose and manage community-acquired pneumonia in a general practice setting). The accuracy of pattern recognition depends on the clinician's breadth and volume of condition-specific illness scripts; simply 'seeing many cases' is insufficient—these cases must encompass the variety of conditions encountered, and accurately represent the range of ways in which each condition presents. This is why developing pattern recognition takes time, and it is not something that can be taught in a book.

**Caveats:** Errors occur when the clinician is inexperienced, over-confident, distracted or fatigued. Patients who present atypically may be misdiagnosed, for example, acute coronary syndromes are more likely to be missed when the patient lacks chest pain.

<sup>1</sup> Referred to as 'pattern recognition', 'system one thinking' or 'non-analytical approach' in literature.

## Combining Methods

There is good evidence that combining intuitive pattern-recognition with analytical strategies (algorithms and hypothetical-deductive reasoning) improves diagnostic outcomes. Most clinicians with some experience employ pattern recognition as a default, as it is faster and less effortful. But when faced with an atypical patient, or a novel clinical scenario, one is unfamiliar with, it is wise to abandon pattern recognition approaches in favour of conscious analytical thinking. Even when pattern recognition is used, safe diagnosis requires constant vigilance for information which ‘does not fit’, in which case analytical reasoning must take over.

## The Process of Diagnostic Reasoning

Having understood the mental frameworks clinicians employ in making a diagnosis, this section delves further into the key elements of the diagnostic reasoning process: (i) information gathering, (ii) interpretation of the information to formulate a problem representation, (iii) making a diagnosis, (iv) searching for aetiologies and complications and (v) dealing with patients who have multiple issues.

### (a) Information Gathering

Some students find that after an initial phase of allowing the patient to talk freely, they are not sure what questions to ask, or default to a memorised list of questions for each symptom. It is helpful to form an early impression of the patient’s presenting complaint, and have some possible diagnoses in mind. Search for further information (history taking, examination and investigations) that would discriminate between the differentials. This is where an algorithm guides information gathering by specifying clinically important pieces of information that differentiate between diagnostic groups (i.e., the branch points). It is a useful maxim that *‘the eye does not see what the mind does not think’*—clinically significant details often have to be explicitly solicited, or may be missed.

Information is interpreted as it is gathered; this is an iterative process culminating in the promotion of one differential as the ‘most likely’ and the relegation of others (Table 1.1).

### (b) Problem Representation

Another early cognitive step is the creation of a one-sentence summary defining the case in specific terms. This may be explicitly articulated, or subconscious. Going back to our clinical scenario (Box 1.1), observes the consultant’s problem representation—*an elderly gentleman with painless progressive obstructive jaundice and clinically significant weight loss*.

Notice how the third-year student’s description of *‘jaundice ... getting worse ... no abdominal pain’* has become *‘painless progressive jaundice’*, and the final-year student’s comment *‘higher ALP than AST and ALT’* is now *‘obstructive jaundice’*. These are



abstract **semantic qualifiers**—painless, as opposed to painful; progressive, not time-limited. The consultant has interpreted the information gathered and recognised diagnostically significant details.

A sound problem representation using semantic qualifiers is associated with strong clinical reasoning. Conversely, failure to generate an accurate problem representation (whether explicit or subconscious) can lead to guessing possible diagnoses (as in the third-year student) based on isolated findings.

### (c) Making a Diagnosis

The mechanics of making a diagnosis has been discussed in the previous section. Ideally, a combination of pattern-recognition and analytical methods should be used; for the novice clinician to whom pattern recognition is unavailable, analytical methods remain useful.

### (d) Aetiologies and Complications

Don't stop at making a diagnosis. Many diseases have underlying aetiologies (although some are 'idiopathic'); keep asking *why*, *why*, and *why*—until no further *why* can be asked. Similarly, every disease causes complications—search for them (Table 1.2).

**Table 1.2. Asking 'Why' and 'So What' in Various Diseases**

Scenario	'Why'—search for aetiology	'So what'—search for complications
40-year-old with acute cerebral infarct	<i>Why</i> does he have a stroke (e.g., cardioembolic, atherosclerosis) <i>Why</i> is getting a stroke this young?	Is there mass effect? Is he able to swallow?
21-year-old with an asthma exacerbation	<i>Why</i> did he get an exacerbation? (e.g., infection, allergen exposure)	Is he in respiratory failure?
70-year-old with iron deficiency anaemia	<i>Why</i> is there iron deficiency? (e.g., occult gastrointestinal bleeding)	Is he symptomatic?

### (e) Patients with Multiple Issues

Patients can be complex. One disease leads to another, and multiple organ systems can be affected by ongoing disease processes. Add to that the potential for drug–disease and drug–drug interactions and things can get really confusing. Try to tease out cause–effect relationships and formulate a problem list to make sense of the madness.

For example, consider the scenario (Box 1.2).

**Box 1.2. A Patient with Multiple Issues**

A 75-year-old man suffers a fall while getting up from bed. He has been having diarrhoea and vomiting after eating some leftovers, and was given antibiotics by a general practitioner 2 days ago. When he got out of bed, he felt giddy and passed out momentarily. He recalls waking up on the floor, alert and in pain. Examination is normal except for marked postural hypotension and decreased skin turgor. ECG is normal. Initial blood tests show a creatinine level above his baseline, and an international normalised ratio (INR) of 5. A small subdural haemorrhage is seen on CT brain. He has a past medical history of diabetes, atrial fibrillation and heart failure, and is on metformin, bisoprolol, furosemide and warfarin.

A problem list might look as follows:

1. Subdural haemorrhage secondary to fall and over-anticoagulation.
2. Syncope secondary to postural hypotension.
3. Hypovolaemia due to gastroenteritis and diuretics, complicated by postural hypotension and acute kidney injury.
4. Over-anticoagulation? due to drug interaction (warfarin and antibiotic).

Formulating a problem list clarifies the issues on hand, and facilitates the creation of a management plan, which addresses all the problems.

## Further Issues

Some other issues in diagnostic reasoning deserve brief mention—the use of diagnostic tests, diagnostic uncertainty and diagnostic error.

## Using Diagnostic Tests

A few words about using diagnostic tests is in order. Unfortunately, the scenario in Box 1.3 is not uncommon. This young man has a very low clinical probability of prostate cancer, and prostate specific antigen (PSA) testing was inappropriate. The test is likely to be a false positive. Even with a positive test, he remains unlikely to have prostate cancer, but most clinicians (and patients) would consider themselves obliged to investigate further ‘to rule out’ cancer. On the other hand, in an 80-year-old gentleman with a hard nodular prostate, bony pain and weight loss, a raised PSA would only increase the clinician’s confidence in a diagnosis of prostate cancer.

**Box 1.3. The Fallacy of Prostate Specific Antigen Testing**

A 25-year-old man seeks a consult for a mildly elevated prostate specific antigen (PSA). He is asymptomatic. It turns out that his company offers ‘free’ yearly health screening, and PSA was included in the standard package. He is anxious about the raised PSA level and upset because his application for a new health insurance policy had been denied on the basis of elevated PSA.

Diagnostic tests help refine a list of differential diagnoses (Table 1.1) by providing additional information to increase the probability of certain diagnoses while decreasing others. For example, amylase/lipase levels are useful in a patient complaining of epigastric pain radiating to the back; a positive test makes pancreatitis likely, but a negative test would prompt a search for alternative diagnoses. Diagnostic tests may also help to guide management (e.g., deciding if a transfusion is necessary), and identify aetiologies (e.g., if pancreatitis, are there gallstones?) and complications of disease. As a rule of thumb, *before ordering a diagnostic test, know what you are looking for*—be sure you know how to interpret the test, how it will help you with diagnosis, and how it will change your management of the patient.

Every diagnostic test performs differently; each has a certain false negative and false positive rate. For example, a negative CT scan does not rule out subarachnoid haemorrhage in a patient with high pre-test probability (false negative rate), and a moderately high troponin does not always imply myocardial infarction in a patient on chronic dialysis (false positive rate). As illustrated, interpretation of test results depends on the pre-test probability, and one should be familiar with the test characteristic before ordering it. Generally, a sensitive test (low false negative rate) helps to ‘rule out’ disease, while a specific test (low false positive rate) helps to ‘rule in’ disease. Some awareness of the issues in diagnostic testing is important, but a detailed discussion of probabilistic reasoning is beyond the scope of this text (but see ‘GO FURTHER’ at the end of this chapter).

### Dealing with Diagnostic Uncertainty

Diagnostic uncertainty is a reality in clinical medicine. Information is limited and conflicting. Treatment decisions often have to be made before the results of clarifying investigations return. This unnerves many but every clinician must learn to be comfortable with diagnostic uncertainty. Some tools:

- Admit the uncertainty. Do not commit to one diagnosis and shut out all others, when this is not borne out by evidence. Rather, keep the differentials in mind.
- Look for a test that will increase the probability of one diagnosis and/or decrease the probability of another.
- When stakes are high and treatment has a favourable benefit/risk ratio, consider empirical treatment even if the diagnosis is uncertain. For example, patients with minor wounds often receive anti-tetanus toxoid even though the risk of tetanus is low, simply because the risk of treatment is smaller than the remote but catastrophic possibility of tetanus. Similarly, in acutely unwell patients with multiple competing differentials, consider treating both diagnoses in parallel if benefit/risk ratios are favourable. For example, in a patient presenting with fever, melena and hypotension; it will be prudent to treat for both septic shock and gastrointestinal bleeding.
- Reverse what is easily reversible. For example, if it is unclear whether a patient’s weakness is due to hypoglycaemia or stroke, administer dextrose and re-examine in 10 min; if weakness persists then it is no longer due to hypoglycaemia. Similarly, if it is unclear whether a patient’s breathlessness is due to pneumonia or progression

of lung cancer, it is reasonable to treat pneumonia (even if the diagnosis is not 'definite'), as the latter would be difficult to treat.

- Remember that diagnosis is a dynamic process. The patient's response to ongoing treatment is usually very informative. Reassessment over an appropriate time period allows us to use time as a diagnostic tool; be prepared to re-consider the initial diagnosis if the patient fails to respond to treatment for the assumed diagnosis.
- Finally, realise that in some situations, it is not necessary to know the exact diagnosis. These situations include when the treatment for both differentials is identical, when the risk of diagnostic testing outweighs the benefit of information gained (e.g., subjecting patients with multiple comorbidities to invasive investigations), or when there is no intention to initiate treatment even if a diagnosis is made (e.g., due to quality of life).

## Diagnostic Error

Every clinician commits diagnostic errors. Wise clinicians learn from their errors, and learn the pitfalls so that errors are minimised. There are three sources of diagnostic error: (i) knowledge deficits, (ii) attitude problems such as overconfidence and (iii) cognitive bias. Apart from a good dose of humility and diligent work to improve knowledge, being aware of cognitive biases (Table 1.3) helps one to avoid them.

## How to Use This Book

This book suggests sample diagnostic algorithms for several common presentations of disease. I hope that learning from these algorithms will help you to become a better diagnostician, and ultimately help patients. For maximal benefit, some advice is in order.

## Five Things This Book Is NOT

1. This is NOT a guide to history taking and clinical examination. Basic history taking and examination skills are prerequisites before higher-order thought processes can be learnt.
2. This is NOT a replacement for standard clinical textbooks. The focus is on differential diagnosis, not on the presentation, diagnosis and management of individual disease entities. Familiarity with individual diseases will help greatly in differential diagnosis—an algorithm makes little sense if the diseases it leads to are completely alien to you.
3. This is NOT a protocol to rote-learn and follow rigidly, *in lieu* of the fullness of clinical reasoning. Algorithms are but one tool in the diagnostic toolbox, and must be used in conjunction with the other tools discussed in this chapter.
4. This is NOT a substitute for seeing real patients. That would be a tragedy. Students with extensive textbook knowledge but inadequate patient contact often find

**Table 1.3. Common Cognitive Biases**

Example		Cognitive bias	Mechanism of bias
1.	A patient is admitted from the emergency department for 'pneumonia'. Although he has no fever, the inpatient team continues to treat 'pneumonia'. Two days later, he is intubated for respiratory failure. A repeat chest X-ray shows worsening fluid overload.	Diagnostic momentum	Accepting a previous diagnosis without sufficient scepticism.
2.	A patient with diabetes and ischaemic heart disease presents with acute crushing chest pain. He is given loading doses of aspirin and Ticagrelor, while awaiting troponin results. One hour later, he collapses and a post-mortem finds aortic dissection.	Anchoring	Locking onto a diagnosis too early, based on initial information.
		Premature closure	Accepting the first diagnosis without considering whether there is a better answer.
		Under-adjustment	Failure to revise the diagnosis when new information becomes available.
3.	A young lady presents with symmetrical small joint polyarthritis and morning stiffness lasting 2 hr. Her general practitioner declines to refer her to a rheumatologist because her rheumatoid factor was normal.	Blind obedience	Undue deference to a false-negative diagnostic test (or other authority).
4.	After missing a pulmonary embolism, a physician orders CT pulmonary angiography for the next five patients with breathlessness.	Availability bias	Diagnosis influenced by ease of recollection of possible diagnoses.
5.	A 'frequent flyer' with borderline personality disorder, who is known to waste emergency department resources, presents with hip pain and difficulty walking. She is sent home with analgesia, with no X-ray done. She is later found to have a hip fracture.	Framing effect	Decision making unduly biased by the way it is presented.
		Affective bias	Personal emotions affecting judgement.

themselves unable to apply their knowledge to a clinical situation. Rather, seeing patients goes hand-in-hand with learning knowledge and knowledge structure—remember, patients are a clinician's best teachers.

5. This is NOT comprehensive or absolute. My goal is simply to help beginner diagnosticians develop a functional diagnostic framework. Therefore, I focus on common presentations of common conditions, downplaying rare diseases and uncommon exceptions to general rules. This book's *algorithms are generally written for adult patients*, and will lead to error if applied in paediatric contexts without modification.

## Five Learning Aids to Help You

Apart from an algorithm to every symptom, and a discussion of the content of the algorithm, this book includes several elements to aid learning:

1. **Clinical case and case discussion:** Every chapter opens with a clinical vignette, and closes by applying the content of the chapter onto the clinical case. You are encouraged to pen down how you would approach the clinical vignette before reading the discussion.
2. **End-chapter summaries** of key learning points.
3. **Common pitfalls** highlighted at the end of each chapter.
4. **Illustrative ECGs and radiographs** are included in some chapters. For the sake of brevity, only a limited number of images can be included—looking up more examples can be helpful.
5. **Discussion questions** in several flavours are found at the end of each chapter.
  - (a) **REFLECT!** questions encourage reflection on clinical cases previously encountered, so as to reinforce ‘illness scripts’ and encourage reflective practice.
  - (b) **EXPLORE!** questions prompt reading up on topics related to the chapter, or prompt reinforcement and application of concepts discussed.
  - (c) **DISCUSS!** questions should be discussed in small groups. They include differing opinions up for debate, as well as mini-exercises to synthesise and apply content learnt in the chapter.
  - (d) **GO FURTHER!** questions suggest slightly more advanced concepts interested learners might wish to explore.

## Five Ways to Use This Book

1. **As a primer.** Before beginning a new clinical rotation, flip quickly through relevant chapters. Use the approach discussed as a roadmap to read up on what you are unfamiliar with. Try to hit the ground running and learn as much as possible from the first patient you see.
2. **As a just-in-time aid.** Bring this book to the bedside. If you ever get ‘stuck’ while clerking a patient, whip out the relevant chapter and see if it can give you some ideas.
3. **Self-practice.** Read the clinical case at the beginning of each chapter. Jot down how you will approach the patient. Then read the chapter; as you read, continually edit your draft approach.
4. **Deliberate practice with patients.** Clerk patients (without referring to this book or the case notes); list and rank the top differentials. Next, refer to the relevant book chapter, and see if there is anything else it might prompt you to consider (other differentials to consider, important differentiating questions you did not ask, etc.). Go back to the patient and refine your differential list. Finally, open the case notes and investigations, and check if your diagnosis is similar to that of the expert clinician’s (if it differs, ask why—the expert is not always right). *Repeated deliberate practice is essential in learning clinical diagnosis.*

5. **Critical reflection.** Examine the algorithms critically. Identify the key differentiators used (usually at branch points), and ask:
- What is the pathophysiologic basis of these key differentiators? Understanding the pathophysiologic mechanisms for the presentation of diseases aids understanding.
  - To what extent are they reliable? It is helpful to understand what are ‘hard signs’ with high specificity (e.g., upgoing plantars makes an upper motor neuron lesion *very* likely) and what are ‘soft signs’ with low specificity (e.g., a positive anti-nuclear antibody does not always mean that a patient has autoimmune disease).



Using what you have learnt, **pen down your approach** to the Clinical Case at the start of the chapter **BEFORE reading the discussion** below.

### Case Discussion

*This student is not alone in having good content knowledge but struggling with clinical reasoning and the diagnostic process. Very often the root of the problem lies in (i) poor study habits—rote learning without processing to improve knowledge structure, and (ii) inadequate patient contact. She should immediately:*

- Put down the textbook and hit the wards. Try to clerk as many typical patients as possible, and for every patient, list three top differentials and complete Table 1.1.*
- Use algorithms to jump-start her journey in clinical reasoning. This book can help but it is not another textbook to be learnt by rote; rather, it is a teach-book to be used alongside seeing patients (page 13).*

<b>Key Lessons</b>	<ol style="list-style-type: none"> <li>1. Diagnosis requires content knowledge and knowledge structure. Combining analytical reasoning (via hypothetical-deductive methods and algorithms) with intuitive pattern-recognition increases the chance of diagnostic success.</li> <li>2. An appropriate search for underlying aetiologies, appropriate use of diagnostic tests, and problem list formulation can assist in diagnostic reasoning.</li> <li>3. Hard work, humility and an awareness of cognitive biases are helpful in minimising diagnostic error.</li> </ol>
<b>Common Pitfalls</b>	<ol style="list-style-type: none"> <li>1. Trying to memorise this book is not going to work.</li> <li>2. Trying to learn medicine without seeing patients is doomed to failure.</li> </ol>
<b>Questions</b>	<ol style="list-style-type: none"> <li>1. <b>REFLECT!</b> Recall one diagnostic error that you have made (or witnessed someone make). What were the factors that led to the error? What could have been done differently to prevent the error from being made?</li> <li>2. <b>EXPLORE!</b> What is the difference between surface learning and deep learning? When might one favour either approach?</li> </ol>

3. **DISCUSS!** The explosion of medical knowledge in the past 10–20 years has made it difficult for any clinician (must less a beginner) to keep up. What are some strategies to cope with this volume of information and pace of progress?
  4. **GO FURTHER!** Read up on Bayesian conditional probability. How does the pre-test probability influence interpretation of a given diagnostic test?
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# Heart and Lungs

## Chapter 2 An Approach to Hypotension

### Clinical Case

*You are called to review a 64-year-old gentleman on the Renal ward for a blood pressure of 75/43. He has a history of end-stage renal failure on haemodialysis, diabetes, hypertension, ischaemic heart disease, atrial fibrillation and peripheral vascular disease with a left below-knee amputation. He was admitted 4 days ago for a thrombosed arteriovenous fistula. As thrombectomy of the fistula was unsuccessful, a tunnelled temporary dialysis catheter ('Permcath') was inserted in his right internal jugular vein 3 days ago. He is due for discharge tomorrow. How will you approach his hypotension?*

Hypotension is a medical emergency. It indicates that the patient is severely ill, with a disease process causing physiologic compromise exceeding the body's compensatory mechanisms. Pathophysiologically, hypotension reflects a failure in preload (cannot fill left ventricle due to volume loss, inflow obstruction or excessive heart rate), contractility (heart muscle dysfunction or outflow obstruction) or afterload (decrease in systemic vascular resistance due to peripheral vasodilation).

In addition to hypotension, look out for tachycardia, oliguria, abnormal mental status and clammy peripheries, which are likewise indicators of shock—a state of tissue hypoperfusion that leads to organ dysfunction. High lactate indicates anaerobic metabolism due to insufficient tissue perfusion; in sepsis, levels above 4 mmol/L, even with normal blood pressure, has been shown to have a mortality rate comparable to overt septic shock.

The causes of hypotension can be classified as hypovolaemic, cardiogenic, obstructive and distributive (Table 2.1).

### Initial Approach

Obtain a rapid clinical impression from history, examination and bedside investigations (including ECG and arterial blood gases). Focus initially on common causes of shock, and any cause that the patient may be particularly predisposed to. The temperature of the peripheries is a helpful differentiator—Warm peripheries indicate vasodilation, as mediated by bacterial endotoxin (septic shock), histamine (anaphylaxis) or loss of sympathetic tone (neurogenic shock and Addisonian crisis). Conversely, clammy peripheries indicate vasoconstriction, which is an appropriate response to hypovolaemic, cardiogenic and obstructive shock. Figure 2.1 provides an initial approach.

**Table 2.1. Causes of Shock**

	<b>Hypovolaemic</b>	<b>Cardiogenic</b>	<b>Obstructive</b>	<b>Distributive</b>
<b>Aetiology</b>	Haemorrhage— obvious or occult Dehydration Polyuria or over-diuresis	Acute myocardial infarct Arrhythmias Mechanical dysfunction e.g., acute valve rupture	Massive PE Tension pneumothorax Cardiac tamponade	Septic shock (common) Anaphylaxis Addisonian crisis Neurogenic shock Myxoedema crisis
<b>Clinical history</b>	Bleeding Abdominal pain Diarrhoea, vomiting	Chest pain Diaphoresis Dyspnoea Palpitations	Respiratory distress, pleuritic chest pain Unilateral swollen leg	Fever Angioedema, wheeze, new medication taken
<b>Peripheries</b>	Cold	Cold	Cold	Warm
<b>Fluid status</b>	Under-filled	Over-filled (usually) Under-filled (very acute)	Over-filled (tamponade) Euvolaemic (PE, pneumothorax)	Under-filled or euvolaemic
<b>Other exam findings</b>	Pallor Per-rectal bleeding Abdominal tenderness in occult bleeding	Arrhythmia Displaced apex beat Murmur	Calf swelling (PE) Hyper-resonant lung (pneumothorax) Beck's triad (tamponade)	Localising source of sepsis Angioedema, stridor (anaphylaxis)
<b>Investigations</b>	Haemoglobin + GXM Urine pregnancy test Consider abdominal imaging	ECG, cardiac enzymes Chest X-ray Consider echo	ECG Bedside echo CT pulmonary angiogram	Septic workup, lactate

GXM, group and cross-match; PE, pulmonary embolism.

Institute emergent management based on the bedside clinical impression; do not wait for blood tests to return. Return to review the patient, reviewing if the patient has responded to initial therapy, and if blood results support the initial diagnosis. Failure to respond to initial treatment should prompt reconsideration of the initial diagnosis, and escalation of initial treatment (e.g., more fluids and starting inotropes). Adjuncts to bedside clinical diagnosis, in particular bedside ultrasound scanning, can be helpful. Bear in mind that there may be multiple concurrent causes of shock, for example, sepsis precipitating an acute myocardial infarction.

The subsequent sections discuss individual classes of shock in greater detail.

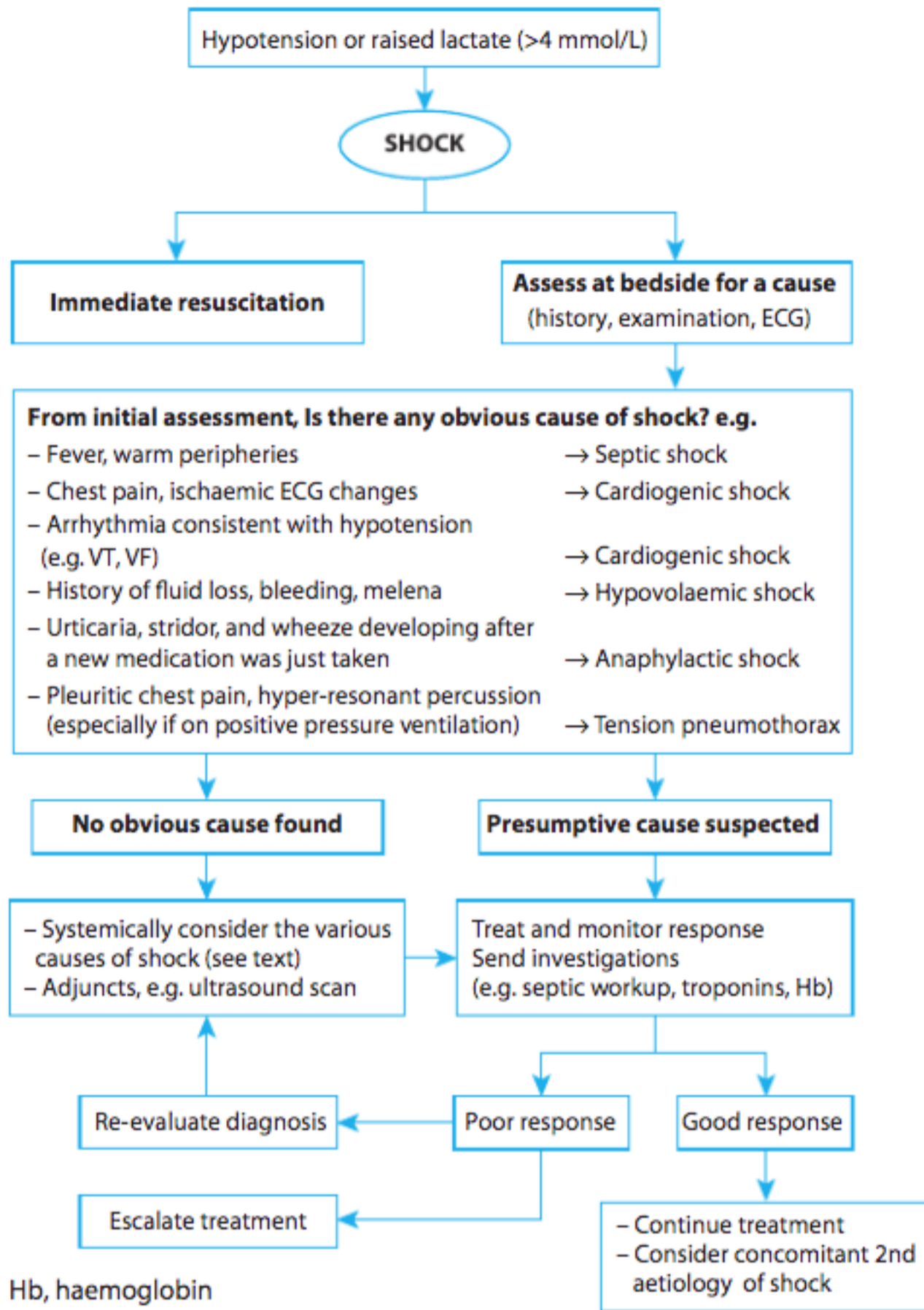


Figure 2.1. Initial clinical approach to hypotension.

## Hypovolaemic Shock

In hypovolaemic states, examination reveals under-filled peripheries (decreased skin turgor, dry mucous membranes, flat jugular venous pressure (JVP) and clear lungs). Hypovolaemia can arise from:

- Fluid loss:** There may be a clinical history of gastrointestinal losses (diarrhoea and vomiting), renal losses (polyuria e.g., in recovery phase of acute tubular necrosis, or hyperglycaemic emergencies). Victims of heat stroke and burns are also hypovolaemic.

Workup