The Hands-on Guide to Clinical Reasoning in Medicine

Mujammil Irfan

with website

WILEY Blackwell

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The names used in this text are not intentional but only used as vehicles to promote learning

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Dedicated to my grand-parents
Syed Maqboolullah Sha Khadri
Zahedunnisa
Noorunnisa Begum

Contents

Foreword

Teaching Clinical Medicine is both an art and a science. In its simplest form it has several components: history taking or gathering information relevant to the patient's problem, clinical methods, or how to examine the patient, differential diagnosis, or what is the most likely condition affecting the patient, and, a management plan or what investigations one should undertake and what treatment to start. Arguably, history taking and clinical examination are two of the easiest to teach. One could even use tick-boxes for history and examination to teach and assess the students. The most difficult component of clinical medicine is teaching the art and science of arriving at a diagnosis and formulating an appropriate action plan, in other words, Clinical Reasoning.

Clinical experience and analytical thinking are both extremely important in making an accurate diagnosis but not yet available or developed in a medical student. Furthermore, uncertainties and complexities of clinical medicine make reliance on experience wholly inadequate. A more structured approach is required both by the teacher and by the student to employ clinical reasoning at every step of the process of making a diagnosis.

This book explores, with practical examples, the process of learning through reasoning. It enables the student (and his teacher) to approach the patient with an open mind. It helps him to collect relevant information, both positive and negative, whilst at the same time critically evaluating its relevance to clinical diagnosis.

Dr. Irfan has long had an interest in teaching and this book is, but one example of his commitment to teaching medical students. He should be commended for approaching this difficult aspect of clinical teaching in a unique and conversational way, using medical students, and, with complex medical scenarios that get resolved through critical analysis and reasoning. I would have carried this book and the accompanying booklet with me both as a final MB student and as a house officer. These new and exciting methods in clinical teaching enable one's mind to think so the eyes can see.

Dr B S Dwarak Sastry OBE
DL FRCPI FRCP

Preface

All through medical school we are taught to find one diagnosis that fits the clinical picture. We are taught to decipher clinical information in black and white, and investigate and initiate treatments evidenced by randomised controlled trials. We are trained to move in a linear fashion from data collection (history taking, clinical examination, investigations) to diagnosis, treatment, and prognosis. Armed with this knowledge and practice we start as doctors in the real world and quickly feel insecure in the face of uncertainty.

The real world as we know it is a bit more unforgiving! Clinicians are faced with incomplete data, uncertain circumstances; and difficult diagnostic, investigational, and therapeutic decisions on a daily basis. A pure subjective stance favouring astute clinicians who rely heavily on their past experience in solving the present diagnostic problem can be fatally flawed. Enter evidence based medicine (EBM) which aims to take away all that subjectivity and usher in the era of objective science based on statistical analyses and clinical practice rooted in guidelines and protocols. There is however a glitch: several clinical problems have no evidence, and when there is some it can be insufficient or even conflicting. Hence, rigid adherence to guidelines in these settings can lead to uncomfortable outcomes as at the end of the day we are dealing with people, not numbers (Sniderman et al. 2013).

Through the course of this book I shall aim to sow the seeds of a 'thinking doctor.' One who not only heeds best practice evidence and follows evidence based guidelines, but also remains attuned to human communication. One who knows where each of these attributes is to be used. For, you will soon be exposed to vague symptoms, complex histories and complex disease presentations. Circumstances where protocols and guidelines do not venture, where there are no randomised controlled trials that show you that 'a' is better than 'b.' Yet you are expected to give this person sitting in front of you an answer as to what is wrong with him or her and suggest a solution, all with an ever dwindling commodity in modern medicine, called time. Medicine is an inexact science. We have to learn to be comfortable with uncertainty.

Clinical reasoning is not only tested in all your exams including objective structured clinical examinations (OSCEs) but will be constantly tested throughout your careers as clinicians. You will eventually develop into a unique practicing clinician who will have their own world view, with all its accompanying fallacies and quirks. So long as you remember to consciously develop yourself every step of the way you are unlikely to go wrong and heaven forbid help someone who is ill!

I have used a unique conversational style in this book utilizing two imaginary students and addressing the reader directly. My humble attempt at teaching clinical reasoning is not the only way to learn this art. There will be many who will not agree with everything that I say and that is the beauty of clinical reasoning in medicine where dissent with dogma is the key to progress. The thrill of solving a clinical dilemma is unmatched. I would like you to enjoy every moment of it and thrive in those grey areas where few dare to tread!

Sniderman, A.D., LaChapelle, K.J., Rachon, N.A. et al. 2013. The necessity for clinical reasoning in the era of evidence-based medicine. *Mayo Clinic Proceedings* 88(10), pp. 1108–1114.

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Abbreviations

A&E	Accident and emergency unit
A-a	Alveolar-arterial gradient
ABG	Arterial blood gas
ACEi	Angiotensin converting enzyme inhibitor
ACPA	Anti-citrullinated protein antigen
ACR	Albumin-creatinine ratio
ACS	Acute coronary syndrome
ACTH	Adreno-corticotropin hormone
ADH	Anti-diuretic hormone
ADL	Activities of daily living
AF	Atrial fibrillation
AG	Anion gap
AIN	Acute interstitial nephritis
AKI	Acute kidney injury
ALP	Alkaline phosphatase
ALS	Advanced life support
ALT	Alanine transferase
AMT	Abbreviated mental test
ANA	Antinuclear antibody
ANCA	Anti-nuclear cytoplasmic antibody
APKD	Adult onset polycystic kidney disease
APTT	Anti-prothrombin clotting time
ARB	Angiotensin receptor blocker
ARDS	Acute respiratory distress syndrome
ARR	Absolute risk reduction
ATN	Acute tubular necrosis
AV	Atrioventricular
AVM	Arteriovenous malformation
AXR	Abdominal X-ray
BE	Base excess
BMI	Body mass index
BMs	Blood sugars
BP	Blood pressure
BPH	Benign prostatic hyperplasia
bpm	Beats per minute
BTS	British Thoracic Society
CABG	Coronary artery bypass graft
CAM	Confusion assessment method
CAP	Community acquired pneumonia
CCF	Congestive cardiac failure
CDT	Clock drawing test
CGA	Comprehensive geriatric assessment
CK	Creatinine kinase
CKD	Chronic kidney disease
CMV	Cytomegalovirus
CNS	Central nervous system
COPD	Chronic obstructive pulmonary disease
CRP	c-reactive protein
CSF	Cerebrospinal fluid
CT	Computerised tomography
CTD	Connective tissue disease
CTPA	Computerised tomography pulmonary angiogram
CVA	Cerebrovascular accident
CVS	Cardiovascular system
CXR	Chest x-ray
DI	Diabetes insipidus
DIP	Distal interphalangeal joint
DKA	Diabetic ketoacidosis
DM	Diabetes mellitus
DNAR	Do not attempt resuscitation
DVT	Deep vein thrombosis
EBM	Evidence based medicine
EBV	Epstein Barr virus
ECF	Extracellular fluid
ECG	Electrocardiogram
EF	Ejection fraction
eGFR	Estimated glomerular filtration rate
EMG	Electromyogram
ESR	Erythrocyte sedimentation rate
ESRD	End-stage renal disease
ET	Exercise tolerance
GBS	Guillain Barre syndrome
GCA	Giant cell arteritis
GCS	Glasgow coma scale
GERD	Gastro-esophageal reflux
GGT	Gamma glutamyl transpeptidase
GIB	Gastrointestinal bleed
GI	Gastrointestinal
GN	Glomerulonephritis
GORD	Gastro-oesophageal reflux
GP	General practitioner
GTCS	Generalised tonic clonic seizures
GTN	Nitroglycerine
H/O	History of
HAP	Hospital acquired pneumonia
Hb	Haemoglobin
HBV	Hepatitis B
HCM	Hypertrophic cardiomyopathy
HHS	Hyperosmolar hyperglycaemic state
HIV	Human immunodeficiency virus
HPA	Hypothalamo-pituitary-adrenal axis
HPOA	Hypertrophic pulmonary osteoarthropathy
HR	Heart rate
HRCT	High resolution computerised tomography
HSV	Herpes simplex virus
HT	Hypertension
IBD	Inflammatory bowel disease
IBS	Irritable bowel syndrome
ICD	Implantable cardioverter defibrillator
ICH	Intracranial haemorrhage
ICP	Intracranial pressure
ICS	Intercostal space
IGRA	Interferon gamma release assay
IHD	Ischaemic heart disease
IIH	Intracranial hypertension
ILD	Interstitial lung disease
INR	International normalised ratio
IPF	idiopathic pulmonary fibrosis
ITU	Intensive care unit
IV	Intravenous
JVP	Jugular venous pressure
LBBB	Left bundle branch block

LDH	Lactate dehydrogenase
LFT	Liver function test
LGIB	Lower gastrointestinal bleed
LHF	Left heart failure
LIF	Left iliac fossa
LMN	Lower motor neurone
LMWH	Low molecular weight heparin
LP	Lumbar puncture
LR	Likelihood ratio
LV	Left ventricle
LVF	Left ventricular failure
LVH	Left ventricular hypertrophy
MAP	Mean arterial pressure
MC&S	Microscopy, culture and sensitivity
MCI	Mild cognitive impairement
MCV	Mean corpuscular volume
MI	Myocardial infarction
MMSE	Mini mental state examination
MODS	Multi-organ dysfunction syndrome
MRI	magnetic resonance imaging
MRA	Magnetic resonance angiogram
NASH	Non-alcoholic steatohepatitis
NCS	Nerve conduction studies
NIV	Non-invasive ventillation
NMJ	Neuromuscular junction
NNT	Number needed to treat
NOAC	Newer oral anticoagulants
NPH	Normal pressure hydrocephalus
NSAIDs	Non-steroidal anti-inflammatory drugs
NSTEMI	Non-ST elevation MI
O/E	On examination
OA	Osteoarthritis
OCP	Oral contraceptive pill
OGD	Oesophagogastroduodenoscopy
OSCE	Objective structured clinical examination
PA	Per abdomen
P-A	Postero-anterior
PCKD	Polycystic kidney disease
PCP	Pneumocystis carinii pneumonia
PCR	Polymerase chain reaction
PE	Pulmonary embolism
PESI	Pulmonary embolism severity index
PFT	Pulmonary function test
PMH	Previous medical history
PMN	Polymorphonuclear cell count
PMR	Polymyalgia rheumatica
PND	Paroxysmal nocturnal dyspnoea
PNS	Peripheral nervous system
PPI	Proton pump inhibitor
PPM	Permanent pacemaker
PR	Per rectal
prn	As required
PSC	Primary sclerosing cholangitis
PT	Prothrombin time
PTH	Parathyroid hormone
PUD	Peptic ulcer disease
PVD	Peripheral vascular disease
py	Pack year
RAAS	Renin-angiotensin-aldosterone system
RF	Rheumatoid factor
RHF	Right heart failure
RR	Respiratory rate
RS	Respiratory system
RVH	Right ventricular hypertrophy
SAAG	Serum ascities albumin gradient
SAH	Subarachnoid haemorrhage
SARD	Systemic autoimmune rheumatic disease
sats	Oxygen saturations
SDH	Subdural haemorrhage
SHO	Senior house officer
SIADH	Syndrome of inappropriate ADH secretion
SIRS	Systemic inflammatory response syndrome
SLE	Systemic lupus erythematosis
SOB	Shortness of breath
SOBOE	Shortness of breath on exertion
SOL	Space occupying lesion
SpA	Spondylarthropathy
SBP	spontaneous bacterial peritonitis
SQs	Semantic qualifiers
STEMI	ST elevation MI
SVCO	Superior vena cava obstruction
TB	Tuberculosis
TFTs	Thyroid function tests
TIA	Transient ischaemic attack
TLOC	Transient loss of consciousness
TSH	Thyroid stimulating hormone
U&E	Urea and electrolytes
UACS	Upper airway cough syndrome
UGIB	Upper gastrointestinal bleed
UMN	Upper motor neurone
UOP	Urinary output
URTI	Upper respiratory tract infection
USG	Ultrasonography
UTI	Urinary tract infection
V/Q	Ventillation/perfusion
VSD	Ventricular septal defect
VTE	Venous thrombo-embolism
WCC	White cell count

Normal Reference Ranges

Biochemistry

Renal function	
Urea and electrolytes (U&Es)	
Sodium(Na^+)	135–145 mmol l^{-1}
Potassium (K^+)	3.5–4.5 mmol l^{-1}
Urea	2.5–6.7 mmol l^{-1}
Creatinine	53–106 µmol l^{-1}
Chloride (Cl^-)	95–105 mmol l^{-1}
Bicarbonate (HCO_3^-)	24–30 mmol l^{-1}

Liver function tests	
Bilirubin	3–17 µmol l^{-1}
Alanine aminotransferase (ALT)	5–35 IU l^{-1}
Aspartate aminotransferase (AST)	5–35 IU l^{-1}
Alkaline phosphatase (ALP)	30–150 IU l^{-1}
Albumin	35–50 g l^{-1}
Total protein	60–78 g l^{-1}
Globulin	18–36 g l^{-1}
Gamma-glutamyl transpeptidase (GGT)	
Male	11–58 IU l^{-1}
Female	7–33 IU l^{-1}
Alpha fetoprotein (AFP)	0–40 mcg l^{-1}

Bone profile	
Corrected calcium (Ca^{2+})	2.1–2.65 mmol l^{-1}
Phosphate (PO_4^{3-})	0.8–1.45 mmol l^{-1}
Alkaline phosphatase (ALP)	30–150 IU l^{-1}
Albumin	35–50 g l^{-1}

Miscellaneous	
Amylase	25–125 U l^{-1}
C-reactive protein	<10 mg l^{-1}
Creatine kinase (CK)	
Male	25–195 IU l^{-1}
Females	25–170 IU l^{-1}
Lactate dehydrogenase (LDH)	70–250 IU l^{-1}
Plasma osmolality	280–300 mosmol kg^{-1}
Troponin I	<0.1 µg l^{-1}
Troponin T	<0.03 µg l^{-1}
Urate	0.15–0.5 mmol l^{-1}

Drug levels	
Digoxin (6 h post dose)	0.8–2 nmol l^{-1}
Lithium	0.5–1.5 mmol l^{-1}

Endocrinology	
Free thyroxine (free T_4)	7.6–19.7 pmol l^{-1}
Total thyroxine (T_4)	70–140 nmol l^{-1}
Thyroid-stimulating hormone (TSH)	0.4–4.5 mU l^{-1}

Hematology

Full blood count (FBC)	
Hemoglobin (Hb)	
Males	135–180 g l^{-1}
Females	115–160 g l^{-1}
Mean cell volume (MCV)	76–96 fl
Red cell distribution width	12–15%
Packed cell volume (PCV) or hematocrit (Hct)	
Males	0.4–0.54
Females	0.36–0.46
Red cell count (RCC)	
Males	4.5–6.5 × 10^{12} l^{-1}
Females	3.8–5.8 × 10^{12} l^{-1}
White cell count (WCC)	4–11 × 10^9 l^{-1}
Differential cell count	
Neutrophils	2–7.5 × 10^9 l^{-1}
Lymphocytes	1.5–4 × 10^9 l^{-1}
Eosinophils	0.04–0.4 × 10^9 l^{-1}
Monocytes	0.2–0.8 × 10^9 l^{-1}
Basophils	0.0–0.1 × 10^9 l^{-1}
Platelets	150–400 × 10^9 l^{-1}
Reticulocytes	05–2.5% of red blood cells

Clotting profile	
Prothrombin time (PT)	12–16 s
Activated partial thromboplastin time (APTT)	35–45 s
Fibrinogen	2–4 g l^{-1}
D-dimer	<0.5 mg l^{-1}

Haematinics	
Iron studies	
Iron	11–32 mol l^{-1}
Total iron-binding capacity (TIBC)	42–80 mol l^{-1}
Ferritin	12–200 µg l^{-1}
Folate	>2 µg l^{-1}
Vitamin B_{12}	>150 ng l^{-1}

Miscellaneous

Cerebrospinal fluid (CSF)	
Total protein	<0.45 g l^{-1}
Glucose (2/3 of plasma glucose)	2.5–4.4 mmol l^{-1}
White cell count (WCC)	<5/mm^3
Red cell count (RCC)	0/mm^3

Urine	
Creatinine clearance (Ccr)	
Male	85–125 ml min^{-1}
Female	75–115 ml min^{-1}
Osmolality	250–1250 mosmol kg^{-1}
Protein	<0.2 g day^{-1}

Ascitic fluid	
Total protein	<0.45 g l^{-1}
Glucose (2/3 of plasma glucose)	2.5–4.4 mmol l^{-1}
White cell count (WCC)	<5/mm^3
Red cell count (RCC)	0/mm^3

ABG on air	
pH	7.35–7.45
$PaCO_2$	4.7–6 kpa
PaO_2	11–13 kpa
HCO_3–	7.35–7.45
Base excess (BE)	–2 to +2
Saturations	>94%

Icons Explained

Activity requiring written answers

Answer key

Activity requiring 'thinking'

Remember me!

Summary

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/irfan/clinicalreasoning

The website includes a reflective action guide.

1 Introduction: The Skeleton Laid Bare

▼
This chapter discusses the basic layout of this book
▲

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).

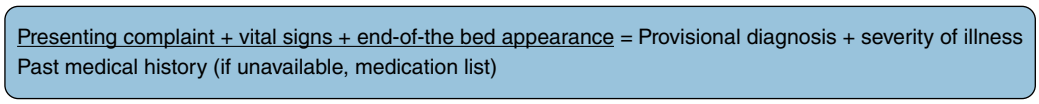

Figure 1.1

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.

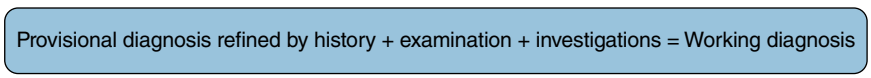

Figure 1.2

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).

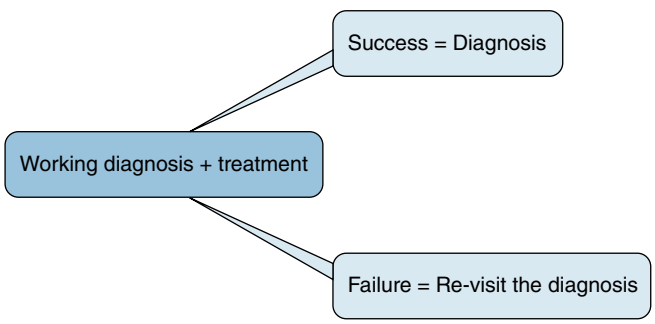

Figure 1.3