THE ESSENTIAL GUIDE TO CLEAR AND SENSIBLE DIAGNOSIS

OXFORD HANDBOOK OF CLINICAL DIAGNOSIS

Huw Llewelyn | Hock Aun Ang
Keir Lewis | Anees Al-Abdullah

Describes the reasoning processes used to arrive at diagnosis and how to explain a rationale for students and practising doctors

Includes additional images to aid understanding

Provides information on initial and longer-term treatments after diagnosis

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Oxford Handbook of Clinical Diagnosis

Third edition

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Foreword to third edition

Last year, I celebrated my 30th year as a doctor and my son began his training as a (graduate entry) medical student. I have come to enjoy the intergenerational 'grand rounds' in which one of us describes a case in the time-honoured format—starting with a structured history, going on to the clinical examination and adding diagnostic tests that progress from the simple and non-invasive to all the wonders and dreads of modern technology—while the other tries to guess the diagnosis from as few clues as possible. Given that most medical knowledge now lies in the category 'forgotten by the mother and not yet encountered by the son', this book is likely to become well thumbed by both of us as we play our diagnostic game.

Much of this book reflects the fact that Huw Llewelyn is a mathematician and logician as well as a highly experienced physician. In many cases, diagnosis can and should be a process of deduction that begins with a 'diagnostic lead' (a single symptom or sign, such as 'right iliac fossa pain', that gets you started), the cause of which can be progressively narrowed and refined by incorporating factors such as age and gender; the timing and speed of onset; the pattern of associated symptoms, signs and pre-existing conditions; and the results of investigations. Frontal headache in a teenager who was well until yesterday is likely to have a different cause from frontal headache that has been present for many months in a 65-year-old with hypertension and depression. Evidence can often be collected in the history and clinical examination that is 'suggestive' or 'confirmatory' (use these terms with care—they are defined in the book) of particular diagnostic possibilities. More rarely, certain tests or combinations of tests can effectively 'rule in' or 'rule out' certain diagnostic options.

You probably knew all that already, so what will you learn from this book that goes beyond standard teaching on clinical diagnosis? For me, the added value was in the sophistication with which the principles of probability and decision science have been applied to the many and varied challenges of clinical practice. The book's (mainly implicit) message is that if you take a logical and step-wise approach, using your experience, history-taking skills, and clinical acumen to select the best diagnostic leads and add granularity to your decision tree, you will often render costly and unpleasant diagnostic tests redundant. Less commonly, you will justify the expense and inconvenience of such tests in selected patients.

The skilled diagnostician is not the one who rattles off a long list of differential diagnoses for every symptom, applies algorithms mechanically, ticks all the boxes on a blood request form or scans the head of every patient with blurred vision. Rather, the skilled diagnostician is the one who combines thoughtful history-taking, focused clinical examination, and judicious investigation so that each successive step contributes to an emerging picture of the problem and informs the selection of the next step. As the authors say (p.20), 'It is important to understand that clinical diagnosis is not a static classification system based on diagnostic criteria or their probable presence. It is a dynamic process.'

The bulk of the book is a treasure-trove of diagnostic puzzles from red throat to wasting of the small muscles of the hands, from which I predict hours of fun for students and seasoned clinicians alike. There are also sections on biochemical conundrums such as hyponatraemia, and radiological old chestnuts such as a round opacity on the chest X-ray. Reassuringly, theoretical sections such as 'Grappling with Probabilities' and 'Bayes' and other rules' are relegated to a final chapter that can be safely omitted by those whose interests are more clinical than mathematical.

Despite its emphasis on deductive logic, this book is by no means an uncritical offering to the gods of decision science. Llewelyn and his coauthors are careful to point out (as Dave Sackett and colleagues did back in the 1970s) that many diagnoses are made intuitively—for example via the pattern recognition that allows us to look at a patient and instantly think 'Down's syndrome' or 'chicken-pox'. They also remind us that mild symptoms are often both non-specific and self-limiting (hence may need no more active management than advising the patient to return if not improving), and they warn us of the dangers of over-diagnosis and that increasingly common problem in modern diagnostics, the 'incidentaloma'.

Like the birth of a third child, the publication of the third edition of a book is cause for much celebration: it tends to both reflect and build on significant success with earlier versions. Perhaps it is too early to encourage the authors of the *Oxford Handbook of Clinical Diagnosis* (3rd edition) to contemplate a companion volume to this magnum opus. But if they were open to such a suggestion, I would encourage them to team up with experts in public understanding of science and produce a version of the book aimed at patients and carers. After all, if your patients were reading the wisdom distilled in these pages, that would surely make for some interesting and productive conversations.

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2014

Preface

This book helps doctors and students to arrive at a diagnosis, and to explain and to justify their reasoning, especially when seeing patients with new problems that lie outside their personal range of experience. This will happen very frequently to students, frequently to house officers, but will still happen regularly to very experienced senior hospital doctors and general practitioners.

The book adopts the approach used by experienced diagnosticians, by focusing on the finding with the shortest differential diagnosis (i.e. the best diagnostic lead). It describes the differential diagnoses of such findings that may be encountered by a reader in the history, examination and usual preliminary tests and how the diagnoses can be confirmed. It describes what tactics to adopt in order to find better leads, while not losing sight of the patient's original concern. The probability and set theory of this process is explained in Chapter 13.

The entries on each page of the book resemble a traditional past medical history with multiple diagnoses. The reader scans down the page to see which of the diagnoses with its findings match the patient's findings so far. The compatible findings can then be used as evidence for the diagnosis and treatment, to be shared with the patient and other members of the multidisciplinary team, such as nurses, pharmacists, physiotherapists, and other professionals allied to medicine. It can be used to create high-quality discharge or handover summaries.

Patients or their carers may wish to share in the diagnostic and decision-making process. In order to do this, they need to know what problems have been identified and the tests and treatments being proposed. They will need to know which of these diagnoses explain each problem and treatment. They may also need to know which findings are being used to confirm each diagnosis, and to choose its treatments and to mark the outcome. The book describes how this information can be provided in writing. The patient or carer will then be in a position to explain all this to another doctor, if necessary.

In this third edition, there are sections on each page that show how the diagnosis may be finalized by the outcome of management. This replaces the section in the second edition that described the 'initial management' of the condition. The purpose of this is to show how the response of treatment, etc., affects the diagnostic process. Chest X-ray images have been added to illustrate the findings in Chapter 12. The appendix of the second edition has been replaced by Chapter 13 in this third edition and explains the basis of evidence-based differential diagnosis and diagnostic confirmation.

Huw Llewelyn
2014

Dedication

For Angela.

Contents

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Symbols and abbreviations

OHCD	*Oxford Handbook of Clinical Diagnosis*
➔	cross reference
↑	increased
↓	decreased
→	leading to
+ve	positive
–ve	negative
±	with or without
>	greater than
<	less than
≥	equal to or greater than
≤	equal to or less than
α	alpha
β	beta
®	registered
1°	primary
2°	secondary
ABG	arterial blood gas
AC	acromioclavicular
ACE	angiotensin-converting enzyme
ACL	anterior cruciate ligament
ACTH	adrenocorticotropin
ADH	antidiuretic hormone
AER	albumin excretion rate
AF	atrial fibrillation
AFB	acid-fast bacilli
ALT	alanine transaminase
ANA	anti-nuclear antibody
ANCA	anti-neutrophil cytoplasmic antibody
A–P	antero-posterior
APTT	activated partial thromboplastin time
ARB	angiotensin receptor blocker
ARDS	acute respiratory distress syndrome
5-ASA	5-aminosalicylic acid
ASOT	anti-streptolysin O titre
AST	aspartate transaminase

ATLS	advanced trauma life support
AXR	abdominal X-ray
bd	twice daily
BMI	body mass index
BP	blood pressure
Ca^{2+}	calcium
CCU	coronary care unit
CIN	cervical intraepithelial neoplasia
CNS	central nervous system
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
CPK	creatinine phosphokinase
CRP	C-reactive protein
CSF	cerebrospinal fluid
CT	computed tomography
CVP	central venous pressure
CXR	chest X-ray
d	day
DC	direct current
dL	decilitre
DIC	disseminated intravascular coagulation
DNA	deoxyribonucleic acid
DOB	date of birth
DU	duodenal ulcer
DVT	deep vein thrombosis
ECG	electrocardiogram
ECT	electroconvulsive therapy
EEG	electroencephalogram
EMG	electromyography
ELISA	enzyme-linked immunosorbent assay
ERCP	endoscopic retrograde cholangiopancreatography
ESR	erythrocyte sedimentation rate
FBC	full blood count
FEV_1	forced expiratory volume in 1 second
FFP	fresh frozen plasma
FH	family history
FSH	follicular stimulating hormone
FT3	free T3
FT4	free T4
FVC	forced vital capacity

GALS	gait, arms, legs, spine
GCS	Glasgow Coma Score
γGT	gamma glutamyl transpeptidase
GI	gastrointestinal
G6PD	glucose-6-pyruvate dehydrogenase
GnRH	gonadotropin-releasing hormone
GTN	glyceryl trinitrate
GTT	glucose tolerance test
GU	gastric ulcer
h	hour
Hb	haemoglobin
HBsAg	hepatitis B surface antigen
hCG	human chorionic gonadotropin
HCV	hepatitis C virus
HDU	high dependency unit
5-HIAA	5-hydroxyindole acetic acid
HIV	human immunodeficiency virus
HMMA	4 hydroxy-3-methoxymadelic acid
HPC	history of presenting complaint
HOCM	hypertrophic cardiomyopathy
HRT	hormone replacement therapy
IC	intercostals
IgM	immunoglobulin M
IHD	ischaemic heart disease
IM	intramuscular
IP	interphalangeal
ITU	intensive treatment unit
IUCD	intrauterine contraceptive device
IV	intravenous
IVC	inferior vena cava
IVU	intravenous urography
JVP	jugular venous pressure
K^+	potassium
kg	kilogram
L	litre
LFT	liver function test
LH	luteinizing hormone
LIF	left iliac fossa
LMW	low molecular weight
LP	lumbar puncture

LRLQ	localized right lower quadrant
LVF	left ventricular failure
MCP	metacarpophalangeal
mg	milligram
MI	myocardial infarction
min	minute
mL	millilitre
mmHg	millimetre of mercury
mmol	milllimole
MMSE	mini-mental state examination
mo	month
MR	magnetic resonance
MRCP	magnetic resonance cholangiopancreatography
MRI	magnetic resonance imaging
MS	multiple sclerosis
MSU	midstream urine
MTP	metatarsophalangeal
Na^+	sodium
NB	*nota bene*
NG	nasogastric
NIV	non-invasive ventilation
NNT	number needed to treat
NSAID	non-steroidal anti-inflammatory drug
NSAP	non-specific abdominal pain
NSTEMI	non-ST elevated myocardial infarction
O_2	oxygen
OBAS	observation, bracing, and surgery
od	*omni die* (once daily)
OGD	oesophagogastroduodenoscopy
P2	pulmonary component of 2nd heart sound
P–A	postero-anterior
PC	presenting complaint
PCL	posterior cruciate ligament
PCR	polymerase chain reaction
PE	pulmonary embolism
PEG	percutaneous endoscopic gastrostomy
PET	positron emission tomography
PEFR	peak expiratory flow rates
PMH	past medical history
PND	paroxysmal nocturnal dyspnoea

PO	*per os* (by mouth)
PPI	proton pump inhibitor
PR	*per rectum* (by rectum)
prn	as required
PSA	prostatic-specific antigen
PT	prothrombin time
PUVA	psoralen UVA
qds	*quater die sumendus* (four times daily)
RA	rheumatoid arthritis
RBC	red blood cell
RF	rheumatoid factor
RICE	rest, ice, compression, and elevation
RLQ	right lower quadrant
RNA	ribonucleic acid
RUQ	right upper quadrant
S2	2nd heart sound
SALT	speech and language therapy
SH	social history
SHBG	sex hormone-binding globulin
SLE	systemic lupus erythematosus
SSRI	selective serotonin reuptake inhibitor
SVC	superior vena cava
SVT	supraventricular tachycardia
T4	thyroxine
TB	tuberculosis
tds	*ter die sumendus* (three times daily)
TSH	thyroid stimulating hormone
TFT	thyroid function test
TURP	transurethral resection of prostate
U&E	urea and electrolytes
UTI	urinary tract infection
URTI	upper respiratory tract infection
US	ultrasound
UV	ultraviolet
V/Q	ventilation/perfusion
VMA	vanillylmandelic acid
WBC	white blood cell
WCC	white cell count
wk	week
y	year

Chapter 1

The diagnostic process

The purpose of this book

This book explains how to interpret symptoms, physical signs and test results during the diagnostic process. There are many books that provide lists of differential diagnoses. However, this one also explains how you should use these lists. Each section describes:

- The main differential diagnoses of a single diagnostic 'lead'
- How to 'differentiate' between these differential diagnoses
- How to confirm the diagnosis and also to 'finalize' it using the outcome of treatment (see ➔ 'Transparent' reasoning, p.5, ➔ Changing diagnostic leads, p.7 and ➔ Confirming and finalizing a diagnosis, p.8).

Making diagnostic reasoning and decisions transparent

The book explains how to outline your diagnostic reasoning on paper. It does this by showing you how to write a list of differential diagnoses and established diagnoses, each with its supportive evidence so far, which includes the result of management (see ➔ An evidence-based diagnosis and plan, p.13). This can be used in a draft management plan and later in a hospital hand-over or in a discharge summary. The differential diagnoses in the sections of this book, with their evidence and initial management, are described in the same format and can be used as example entries when writing out an outline of the diagnoses and evidence, which includes the result of the management for a patient.

Understanding the reasoning of others

This book helps you to understand the diagnostic reasoning and decisions of others. In order to do so, you (and patients, carers, nurses, and other health professionals) have to ask:

- What is the current management plan (the pieces of advice, treatments, tests, and follow-up arrangements)?
- For each of these items, what are the diagnoses (provisional, probable, definitive, and final)?
- What is the evidence for each diagnosis (how it presented, how it was confirmed, and its markers of progress or outcome)?

Look up the 'problem findings' and diagnoses in this book so that you know what type of answers to expect to these questions. You can write them out in a similar format (see ➔ An evidence-based diagnosis and plan, p.13). After hearing these answers, you may wish to make your own notes in response.

Checking a clinical impression and explicit reasoning

It is important to check all diagnoses and decisions. Reasoning alone using knowledge from a book of this kind is not enough. Such reasoning should be checked by discussing it with someone who is familiar with the situation from past experience and who can recognize if the reasoning makes sense. However, it is equally important to check that diagnoses and decisions made 'intuitively' make sense when compared with transparent reasoning of the type described in this book.

When and how to use this book

This book can be used:

- When assessing a patient, e.g. after the history of presenting complaint, after completing the full history, after completing the examination, and when the test results come back
- In the same way during problem-based learning with case histories
- During private study and revision to allow you to solve clinical problems later without having to refer to the book
- When asking someone else to explain a diagnosis and decision to you.

If the presenting complaint is severe (e.g. pain or breathlessness), disabling (e.g. inability to move a limb or speak), or unusual (e.g. coughing or vomiting blood), then it will tend to be good lead with a shorter differential diagnosis. The most useful diagnostic leads are described in this book—look at the 'Contents' list of each section so that you can recognize them.

Remember that many symptoms and other findings are due to self-limiting conditions that are transient or are corrected within hours or days by the body's own restorative mechanisms. Such self-limiting conditions always have to be considered as part of any differential diagnosis. If the finding is mild and has only been present for a short time and is not accompanied by other features, then it is more probable that it will resolve spontaneously without its cause being identified. However, it is important to review such patients to ensure that there is improvement or resolution, by asking the patient to return if the problem persists. The ability to deal with such self-limiting conditions is a very important skill that has to be learnt by experience. Severe and persistent findings will often turn out to have a cause that requires medical attention.

If the presenting complaint is not a good lead but has a long differential diagnosis, then consider what systems (e.g. cardiovascular or respiratory) it came from and ask 'direct questions' directed at this system to try to find better leads. Also, focus on that system first in your examination. Note the speed of onset; this will suggest the underlying disease process. Onset within seconds suggests an 'electrical' cause, e.g. a fit or rhythm abnormality; onset over seconds to minutes suggests an embolus, a trauma, or rupture; onset over minutes to hours suggests a thrombotic process, over hours to days an acute infection, over days to weeks a chronic infection, weeks to months a tumour, and months to years a degenerative process.

Read this book during private study or revision by covering the column of diagnoses on the left side of the table and testing your ability to recognize the diagnoses when you read the nature of the diagnostic lead associated with the table, and the suggestive and confirmatory findings on the right side of the table. If you are able to do this successfully, you will soon learn to take a history and examine a patient without having to use this book. Do it first with the symptoms and physical signs that are common in your current (and next) clinical attachment so that you are prepared.

'Intuitive' reasoning

Most of the time, experienced doctors use a non-transparent reasoning process. This seems to involve recognizing combinations or patterns of findings consciously or subconsciously, which suggest or confirm a diagnosis, or indicate that some treatment should be given. This is a skill that is improved by repetition. This book will encourage you to do this sooner. However, all doctors specialize and the information in this book will be of help to experienced doctors with patients outside their specialty.

If you were told that a patient had suffered sudden onset of sharp chest pain over seconds to minutes, then this 'diagnostic lead' will make you think consciously or subconsciously of a pneumothorax, pulmonary infarction, etc. If another patient has suddenly started coughing up blood, then this lead would suggest acute bronchitis, pulmonary infarction, bronchial carcinoma, pulmonary tuberculosis, etc. However, if both happened in the same patient, your mental links would 'intersect' on pulmonary infarction and it would surface to consciousness.

If you were to come across this combination of features and had read in this book during private study that they 'suggested' pulmonary infarction, then you might think of this diagnosis directly. If you came across these findings many times and a diagnosis of pulmonary infarction was usually confirmed on CT-pulmonary angiogram, then you would soon recognize that the combination of findings as suggesting pulmonary infarction (like recognizing someone's face). The psychological process that leads to such recognition is sometimes described as 'Gestalt' (German for an overall impression). Instead of writing 'diagnosis' many doctors will write 'Impression:' to indicate this.

If the findings so far do not point to a single diagnosis with certainty, then you will have to consider a number of other possibilities. It may then be reasonably certain that the diagnosis will turn out to be one of these. A device for doing this is not to specify a list of diagnostic possibilities, but to write down a term that represents a group of diagnoses, e.g. 'pulmonary lesion' or 'autoimmune process'.

If a diagnosis or small number of differential diagnoses do not come to mind readily in one of these ways, then it is important to turn to the 'transparent' reasoning process. You will always come across unfamiliar situations, however experienced you become, so the 'transparent' approach will always be important.

'Transparent' reasoning

Diagnostic reasoning is transparent if the findings used to arrive at a diagnosis are specified clearly and if the interventions resulting from that diagnosis are also specified. The combination of findings used might have been recognized by the diagnostician at the outset. However, in many cases, the combination of findings would have been assembled by a reasoning process of elimination (see ➔ Diagnostic leads and differentiators, p.6).

A diagnosis will only be certain or 'definite' if the findings so far are 'sufficient' or 'definitive' by an agreed convention. For example, two fasting blood sugars of at least 7mmol/L on different days by convention provide a 'sufficient' criterion for confirming diabetes mellitus. There are other 'sufficient' criteria, e.g. two random sugars over 11mmol/L. All the different sufficient criteria collectively make up the 'definitive' criteria. This means that it is 'necessary' to have at least one of these various criteria. At least one fasting glucose of at least 7mmol/L is also 'necessary' (but not 'sufficient') to confirm the diagnosis, so if the first of a pair of fasting blood sugars is below 7mmol/L, the diagnosis is logically 'eliminated' because they both can no longer be over 7mmol/L.

If the first of two fasting sugars is 7.1mmol/L, then this makes diabetes mellitus more probable than not. The differential diagnosis will also include 'impaired fasting glucose' (if the next result is less than 7mmol/L). Medical conditions change and even though a diagnosis is 'eliminated', any borderline tests may be repeated quite soon. In reality, few diagnoses are defined precisely in this way and a doctor may 'confirm' a diagnosis if the probability of benefit from its advice or treatment is judged to be high and cite in a transparent way the findings on which this confirmation is based.

'Over-diagnosis' is said to occur if patients are labelled with a diagnosis when a high proportion show little prospect of benefiting from any advice or treatment directed at that diagnosis. For example, 'diabetic albuminuria' is said to be present if the urinary albumin excretion rate (AER) is between 20 and 200 micrograms/min on at least two out of three collections, provided that other findings indicate that there is no other cause of albuminuria present. However, there is no difference in those developing diabetic nephropathy within 2 years between those taking placebo or active treatment for the 1/3 of patients with an AER between 20 and 40 micrograms/min, suggesting that there is 'over-diagnosis' as this group of patients do not benefit. Diagnostic criteria need to be based closely on treatment outcomes to avoid this.

A diagnosis becomes final when all the findings that led to the diagnosis being considered can be 'explained' by that diagnosis. For example, if a patient complained of persistent fatigue and this did not respond to the treatments and advice for diabetes, then an additional diagnosis has to be considered. The diagnosis of diabetes mellitus may have been confirmed definitively, but the diagnostic process will not be finalized until other reasons for the fatigue have been confirmed or excluded. It is only then that the process stops. The 'final diagnosis' is then a 'theory' and no longer a hypothesis to be tested further, at least for the time being.

Diagnostic leads and differentiators

A combination of features that identifies a group of patients within which the frequency of those with a diagnosis is high (or even 100%) might well be recognized at the outset. If not, a combination of findings can be assembled 'logically' by using reasoning by elimination. This would be done by first considering the possible causes of a single finding, called a 'diagnostic lead' (e.g. localized right lower quadrant abdominal pain). The possible diagnostic explanations for this 'lead' are then considered, one is chosen (e.g. appendicitis) and findings looked for that occur commonly in that chosen possibility and less commonly (ideally rarely or never) in at least one other possibility.

If a finding (e.g. being male) occurs often in a diagnosis being pursued (e.g. appendicitis) but cannot happen in a differential diagnosis (e.g. ectopic pregnancy), then that diagnosis can be ruled out, being female being a 'necessary' condition for suffering an ectopic pregnancy! However, if a finding such as guarding occurs commonly in the diagnosis being chased (e.g. appendicitis) and less frequently in another diagnosis (e.g. non-specific abdominal pain—NSAP) then NSAP will become less probable, not ruled out.

The 'lead' and the new finding will form a combination within which the frequency of the diagnosis being chased (e.g. appendicitis) becomes more frequent and the diagnosis in which the finding occurs less often becomes less frequent in that combination of findings.

The frequency with which a finding occurs in a diagnosis is often described as its 'sensitivity' by epidemiologists, i.e. the frequency with which the finding 'detects' the diagnosis when screening a population. Statisticians also call the 'sensitivity' the 'likelihood' of the finding being discovered when the patient is known to have the diagnosis. If the finding is 'likely' to occur in a diagnosis being chased and is 'unlikely' to occur in one of its differential diagnoses, then the ratio of the two likelihoods represents the finding's ability to differentiate between those two diagnoses. This makes one more probable and the other less probable. This book describes such findings under the headings of 'Suggested by' and 'Confirmed by'. It is findings that cannot occur by definition in other diagnoses that 'confirm' a diagnosis—'definitely'.

Eddy and Clanton analysed the thought processes of senior doctors participating in the Clinico-Pathological Conferences at the Massachusetts General Hospital[1]. They pointed out that choosing a diagnostic lead, e.g. localized right lower quadrant abdominal pain (which they called a 'pivot') was central to these experienced doctors' explanations when solving diagnostic problems. They also noted that during diagnostic reasoning, other findings (e.g. guarding) were used to 'prune' some of the differential diagnoses (e.g. pruning away NSAP).

There has been a re-awakening of interest in all this as 'stratified' or 'personalized' medical research. The aim is to have more differential diagnostic sub-divisions so that each predicts treatment response more accurately.

Changing diagnostic leads

A patient presenting with breathlessness will have a long list of differential diagnoses. A diagnostician might suspect a 'cardiac' or 'respiratory' reason and after asking for cardiovascular and respiratory symptoms and looking for physical signs, might ask for a chest X-ray (CXR) in the hope of getting a better diagnostic lead. A circular shadow on a CXR will have a much shorter list of differential diagnoses and a CT scan showing a lesion contiguous with a bronchus an even shorter one. A biopsy might provide a diagnostic criterion for a bronchial carcinoma. However, this may only be a working diagnosis even if it is confirmed or definite. All the diagnoses applicable to that patient will not become final until the patient's symptoms have been cured, stabilized, or predicted correctly and no follow-up or other action needs to be taken.

If we come across a powerful finding or combination of findings (e.g. a dense, round shadow on a CXR), this will form a stronger lead with a shorter list of differential diagnoses. It is easier to make a fresh start with such a powerful new finding than to try to work out which of a long list of original diagnostic possibilities (e.g. breathlessness) are being made more probable or less probable by the new finding. Therefore, another measure of a powerful finding is the number of differential diagnoses required to explain, say 99% of patients with that finding. The better the lead, the fewer the differential diagnoses.

Care has to be taken to consider spurious and self-limiting causes for any lead (e.g. a CXR appearance), especially if the differential diagnoses of that lead finding cannot explain any of the patient's symptoms. The same consideration applies when a screening test is performed, e.g. a mammogram. If the patient is asymptomatic, then it is important to consider the possibility that a new finding might be due to a self-limiting condition that might resolve spontaneously without medical assistance. One option would be to repeat the test after a short interval to see if there has been regression. Asymptomatic conditions that are detected incidentally are often labelled wryly as 'incidentalomas'. In many cases they are investigated aggressively and the patient sometimes subjected to potential harm (e.g. radical surgery) with adverse consequences only to find out that the lesion was innocent after all. This is sometimes described as 'over-diagnosis' and 'over-treatment'.

Confirming and finalizing a diagnosis

A diagnosis can be confirmed in different ways. The different confirming (or 'sufficient') criteria taken together form the 'definitive criteria' of the diagnosis. The definitive criteria thus identify all those and only those with the diagnosis. Such criteria can be based on symptoms, signs, and test results (and, in some cases, on the initial result of treatment). However, few patients with a diagnosis will require all the advice or treatments suggested by that diagnosis (e.g. not all patients with diabetes mellitus will need insulin). Further findings may have to be looked for called 'treatment indications', which often form sub-diagnoses. For example, the presence of a very high blood sugar, weight loss, and persistent ketones in the urine would be one such 'indication' for giving insulin; that patient might also be diagnosed as having 'Type 1 Diabetes Mellitus or Type 2 Diabetes Mellitus with severe insulin deficiency'.

In many cases, a diagnostician will start treatment when a diagnosis is probable or suspected without waiting for formal criteria to be fulfilled (e.g. a treatment given on suspicion of meningitis). In such a situation, the diagnostician might imagine the existence of a large number of identical patients who were randomized into different treatment limbs of a randomized clinical trial. The treatment chosen would be the one 'imagined' (i.e. 'predicted', ideally with a known track record of success) to produce the best outcome, bearing in mind the benefits and adverse effects. If the patient improves on treatment, then this may also be regarded as confirmation of the diagnosis, if patients with no other diagnosis could have improved in that way. However, if the patient and diagnostician were satisfied that nothing else needed to be done, then the diagnosis would become 'final'. This could happen even if the diagnosis was only probable, e.g. if a severe headache had been suspected of being meningitis, had resolved on antibiotics but no bacteria had grown in the laboratory, then the final diagnosis would be 'probable bacterial meningitis'.

There may be no formal criteria that are suitable for use in day-to-day clinical care. One approach is to provide a trial of therapy, and if the patient improves, to regard this as a confirmatory result and no other explanation is looked for. The confirmatory findings in this book are based on all of the approaches outlined here. They reflect typical approaches used in the authors' experience. However, none of these approaches are ideal; future medical research may improve matters.

Some patients with a diagnosis have mild conditions so that treatment is not necessary; others may be so severe that it is too late to treat, while others are treatable—this subdivision is known as 'triage' in emergency settings. The group with a diagnosis may also contain subgroups with causes and complications that also require treatment. Therefore, diagnoses (probable or confirmed) may be thought as 'envelopes' that enclose subgroups of patients with other diagnoses for which different actions are indicated. The way in which evidence can be sought to form diagnostic indications and sub-diagnoses is described in Chapter 13.

Evidence that 'suggests' a diagnosis

It is important to remember what 'evidence' means. Evidence is made up of facts, which are records of observations and actions that took place at a place and time. A 'fact' becomes 'evidence' when it is used to make a prediction—in the context of this book, about the presence of a diagnosis (which leads to other predictions that include what could be done to improve matters). A diagnosis is the title to what we picture or predict is happening now, has happened in the past, and what will happen to a patient in the future. This will include causes and complications of the diagnosis. Some of this may be pictured with certainty (i.e. what has been observed already) or with different degrees of probability, depending on the available evidence.

Evidence may be based on facts such as symptoms, signs, and test results recorded in a particular patient. This is 'particular' evidence from a particular patient, which is a 'particular' proposition in logic. In contrast to this, 'general' evidence will be based on facts related to groups of patients such as the result of a clinical trial, which is a 'general' proposition in logic. In order to practice evidence-based medicine, we have to relate the 'particular' evidence from a particular patient to 'general' evidence about groups of similar patients that we have observed and documented carefully or published by others in the medical literature.

The predictions based on 'particular' evidence are diagnoses with different degrees of probability about what is wrong with a patient and what to do. If the listener is going to accept such an opinion on the basis of the evidence, there has to be agreement as to what is acceptable as evidence, which includes how the evidence was obtained. This book contains typical evidence that is used to 'suggest' probable diagnoses and to 'confirm' diagnoses according to definitive criteria that are accepted at present by most doctors in their day-to-day work. These conventions will no doubt change as more 'general' scientific evidence is published.

Each differential diagnosis in every section is followed by the evidence that 'suggests' the probable presence of the diagnosis. The diagnosis is considered to be 'definite' when the confirmatory 'sufficient' criteria are present. In each section, the confirmatory evidence for each diagnosis is provided under another subheading.

For example, localized right lower quadrant abdominal pain with guarding 'suggests' that the diagnosis will probably be appendicitis (see ➔ Localized tenderness in left or right lower quadrant p.363). The diagnosis of appendicitis is 'confirmed' by the appearances at laparotomy and by the resulting definitive histological examination. It is important to note that not all the available findings from the patient have to be used in the reasoning process to confirm a diagnosis. The findings selected may be called the 'central' evidence'. For example, a patient with a large number of findings that includes localized right lower quadrant (LRLQ) pain and guarding can be regarded as a member of a set of such patients with LRLQ and guarding within which the frequency of appendicitis is high (see ➔ Picturing probabilities, p.618).

Confirmatory findings based on general evidence

A confirmatory finding identifies a group or set of patients that 'envelopes' all those with indications for treatment 'explained' by the diagnosis. If new treatment indications are discovered that are explained by the diagnostic theory, then 'the envelope' may need to be expanded. For example, it was discovered some years ago that many patients with features of diabetic retinopathy requiring treatment had blood sugars outside the criteria for diabetes mellitus. Because of this, the World Health Organization and the American Diabetes Association suggested that the 'envelope' for diabetes should be expanded by lowering the diagnostic cut-off point of fasting blood glucose.

It is also possible that new tests may be discovered in the future that select patients more efficiently for treatment. If these new treatable patients lie outside the diagnostic group that was previously considered for treatment, then it might be appropriate to use the new test to identify patients who should be deemed to have the diagnosis. So if 'confirmatory' tests are to be chosen in an evidence-based way, then they should be shown to be superior to rival tests by including more patients who respond to the advice or treatments directed at the diagnosis and excluding more patients with no prospect of responding.

Many diagnoses are based on test results that are 'abnormal', i.e. above or below two standard deviations of the test result in the general population. This means that the 2.5% of patients above and 2.5% of those below these two standard deviations could be regarded as 'abnormal'. The use of two standard deviations is arbitrary and not 'evidence-based'. For example, patients with diabetes mellitus are 'diagnosed' as having 'diabetic microalbuminuria' if their AER are above two standard deviations of the mean (i.e. >20 micrograms/min).

However, in a clinical trial on patients with type 2 diabetes mellitus where their blood pressures had been controlled, there was no difference between those on treatment and placebo in the proportion of patients developing nephropathy within two years if they had an AER between 20 and 40 micrograms/min.[2] This suggests that the cut-off point should be 40 micrograms/min. However, before changing the definition, it would be important to ensure that the patients inside the envelope with an AER between 20 and 40 micrograms/min might not benefit in other ways, e.g. by some being prevented from developing peripheral or coronary artery disease.

Ruling diagnoses in and out

A diagnosis is 'ruled in' if at least one of its confirming (or sufficient) criteria is present. A diagnosis is 'ruled out' if it can be shown that the patient lies outside the diagnostic envelope by showing that one of its 'necessary' criteria is absent. Another way of doing this is to show that not one of the possible confirming (or sufficient) features is present. Another way is to show that a single necessary feature is absent, which must occur in those with the diagnosis, e.g. that the patient is not female and, therefore, cannot have an ectopic pregnancy. Such a constant diagnostic finding is called a 'necessary' criterion, of course.

Findings that suggest diagnoses based on general evidence

The best findings for 'suggesting' probable diagnoses are those which, when used alone or in combination with others, predict the presence of 'confirmatory' test results with the highest frequency of success. The general evidence for the ability of findings to do this during population screening is usually offered in the form of indices such as sensitivity, specificity, and likelihood ratios (the use of such indices can be misleading, however; see ➔ Things that affect 'differential' and 'overall' likelihood ratios, p.627). However, in order to assess the usefulness of tests during the differential diagnostic process, other indices have to be used. One index is the number of diagnoses required to explain most (e.g. 99%) of the differential diagnoses of a diagnostic lead—the fewer the better.

Another index is the ability of a test to differentiate between pairs of diagnoses in such a lead. If a test result occurs commonly in patients with confirmatory findings of one diagnosis and uncommonly in patients with another diagnosis, then that test will help to differentiate between them. The difference in these frequencies of occurrence can be measured by their ratio.

Statisticians describe the frequency of a finding that occurs in those known to have a diagnosis as the 'likelihood' of it occurring (the 'likelihood' is also known to epidemiologists as the 'sensitivity'). The difference between these 'likelihoods' for two different diagnoses can be represented by the ratio of the two likelihoods. As this ratio refers to a pair of differential diagnoses, we can call it a 'differential likelihood ratio'. This is different to the 'overall likelihood ratio', which is the frequency of a finding in patients with a confirmed diagnosis divided by the frequency of the same finding in *all* those confirmed *not* to have that diagnosis. This 'non-differential' or 'overall' likelihood ratio is more useful when screening populations by using one test to detect one diagnosis. The 'overall' likelihood ratio is not as helpful for differential diagnoses (see ➔ Evidence for a finding's role in reasoning by elimination, p.625 for a discussion about likelihood ratios).

Explaining a diagnostic thought process

You may well have arrived at differential diagnoses by using intuitive, non-transparent, pattern recognition and not considered in an explicit way how it was done. Alternatively, you may have recorded your team's consensus opinion. However, you may be asked by a patient, student, nurse, or doctor to explain your thinking. In fairness, the way that your own mind (let alone someone else's mind) has actually worked subconsciously may be impossible to explain.

The first step is to write a summary of the positive findings, diagnoses, evidence, and management, as shown in ➔ An evidence-based diagnosis and plan, p.13. The original evidence for established diagnoses (e.g. type 2 diabetes mellitus) may not be available. However, for new diagnoses, choose from the evidence the best lead with the shortest differential diagnosis. Use the other findings to show that the one (or some) diagnoses are more probable or confirmed, and others less probable or ruled out.

If these conclusions of the non-transparent and transparent thought processes are not the same, you may wish to revise your opinion and list of differential diagnoses. By doing this, you will be checking diagnoses by using a different mental process in the same way as you would check the answer to arithmetic addition by adding up the list of numbers in a different order.

In order to avoid overlooking diagnoses, jog your memory by using 'sieves' to use 'recognition' to and help 'recall' by listing the possible broad anatomical and physiological explanations (see ➔ Medical and surgical sieves, p.14).

An evidence-based diagnosis and plan

Positive findings summary

Central chest pain for 4h with jaw discomfort, sweating, and nausea (1/10/13). PMH of hypertension for 10y. History of mild jaundice during febrile illnesses for years. BP 146/88 on admission (1/10/13). ECG: T wave inversion S2, AvF, V4, and V5. Latest HbA1c=8.7% (5/8/13).

Assessment and plan

?Unstable angina

?Non-ST elevated myocardial infarction (NSTEMI)
Outline evidence: central chest pain for 4h with jaw discomfort, sweating and nausea (1/10/13). ECG: T wave inversion S2, AvF, V4, and V5.
Plan: for troponin I immediately and 12h after onset of pain. Aspirin 300mg stat, bisoprolol 5mg od, isosorbide mononitrate 10mg bd.

?Gilbert's disease

?Cholelithiasis
Outline evidence: jaundiced sclera, history of mild jaundice during febrile illnesses for years, none of liver disease (1/10/13).
Plan: check bilirubin, urobilinogen, AST, γGT.

Other active diagnoses

Essential hypertension
Outline evidence: history of raised BP for 10y. Current BP 146/88 on admission (1/10/13).
Plan: continue bendroflumethiazide 2.5mg od, perindopril 2mg od.

Type 2 diabetes mellitus
Outline evidence: latest HbA1c = 8.7% (5/8/13).
Plan: stop gliclazide 160mg bd. Start insulin sliding scale.

Medical and surgical sieves

Check that you have not forgotten something by using a 'medical sieve'. For example:

- Social system and environment
- Locomotor system
- Nervous system
- Cardiovascular system
- Respiratory system
- Alimentary system
- Renal and urinary tract
- Reproductive system
- Endocrine and autonomic system
- Haematological and immune system.

Consider each of these systems by using the 'surgical sieve'. Is there a problem that is congenital, infective, traumatic, neoplastic, or degenerative?

There are many such 'sieves' in use; choose the ones that appeal to you.

The information in the pages of the OHCD is also set out in the same format as the Assessment and Plan (compare diagnoses of 'unstable angina' and 'NSTEMI' with those in ➔ Chest pain—alarming and increasing over minutes to hours, p.174). The section on chest pain gives some differential diagnoses with typical suggestive and confirmatory evidence that could also be added to those in ➔ An evidence-based diagnosis and plan, p.13. You may refer to these as examples when writing your own assessments and plans.

Diagnoses, hypotheses, and theories

Although the findings used to confirm a diagnosis can be observed, all things pictured or imagined under the title of the diagnosis cannot be confirmed by observation, e.g. molecular changes in damaged tissue or what would have happened in a particular patient if a treatment had not been given. Not only does this apply to hypotheses for individual patients, it also applies to what is imagined about populations of patients in scientific hypotheses and theories. It is thus possible that something else will be imagined or pictured in future that is also compatible with findings previously explained by another theory.

This is why the philosopher of science, Karl Popper, argued that general hypotheses and theories cannot be proven or confirmed in their entirety (see also ➔ Reasoning with hypotheses, p.637). However, if a new observation is inconsistent with one aspect of the hypothesis, it will have been 'falsified'. It will thus have to be changed to some degree (perhaps completely or slightly) to take the new observation into account.

Raised ST segments on an ECG in someone with severe central chest pain were formerly part of the criteria for confirming 'myocardial infarction', which suggested that a part of the myocardium was dead. However, one aspect of this theory has been 'falsified' because it has been discovered that some (or all) of the 'infarcted' myocardium is salvageable. With our new understanding, we use the same findings to 'confirm' an 'ST elevated myocardial 'infarction'. (It would be more accurate to say 'ST elevated acute myocardial ischaemia'.) We have modified the theory and now think that the process of 'infarction' is not complete and that the 'ischaemia' can be stopped with treatment, with reversal of many changes.

However, it is important to assess the reliability of the 'falsifying' fact. This is done by estimating the probability of the 'falsifying' observation being *replicated* by other scientists (or another doctor if the hypothesis is a diagnosis about an individual patient based on particular evidence). If the probability of replication of the evidence is high about a 'general' observation, then the observation may be accepted by the scientific community (but many may go to the trouble of repeating the study to make sure). If the *P* value is low or the 95% confidence intervals are narrow, then the probability of non-replication due to chance observations alone will be low. However, before we can conclude that the probability of replication is high, we must also be satisfied that the probability of non-replication due to other reasons is low (e.g. non-replication because of the presence of contradictory results in other studies, poor or idiosyncratic methodology, dishonesty, etc.). This is discussed further in ➔ Estimating the probability of replication with reasoning by elimination, p.636.

Imagining an ideal clinical trial

The findings used to define a 'diagnostic envelope' should enclose the best treatment indication criteria. These criteria should be chosen ideally from a number of candidate criteria. The chosen treatment criterion should be the one that produces the clearest outcome difference between the treatment and control in a comparative trial when all patients with some prospect of benefit are included. For example, when method A for measuring microalbumin in urine chose patients for a trial, 15.3% developed nephropathy on placebo and 7.7% developed nephropathy on treatment, the proportion benefiting being 7.6% (NNT=13.1). However, with method B, 25.9% developed nephropathy on placebo and 11.1% developed it on treatment, the proportion benefiting being 14.8% (NNT=6.9). This would suggest that method A was not identifying patients who benefited so well and would be inferior to method B. This is discussed in detail in ➔ Analysing clinical trials to 'stratify' diagnostic and treatment criteria, p.633; ➔ How to improve treatments by better selection or 'stratification' of patients, p.634; ➔ Studies to establish treatment indication and diagnostic cut-off points, p.635.

In the absence of detailed trial data, a doctor may have to guess whether a patient's findings would identify a group of patients who would benefit from the treatment more than a placebo, bearing in mind side-effects, costs, etc. If, on balance, this would be the case, the doctor could apply a diagnostic term that would summarize his theoretical explanation as to why giving that treatment to a patient with that combination of findings would be better than not doing so.

Decision analysis

Decision analysis is a discipline that models mathematically what would happen if a detailed clinical trial were performed to compare the treatment options being considered for a particular patient. A 'decision tree' is constructed first to show all the possible diagnoses. The tree is extended to show the possible interventional limbs into which the patient could be randomized, followed by all the possible outcomes of each treatment. The branches would end with the effect that each outcome would have on the overall well-being of the patient.

An estimate is then made of the proportions of patients with each diagnosis, the proportions opting for each treatment and the proportions of those experiencing various degrees of well-being. These proportions are then multiplied together to estimate the average degree of well-being experienced by patients sharing each treatment outcome. Each of these average degrees of benefit is regarded as the 'expected' degree of well-being that would be experienced by an individual patient with each outcome. This is regarded as a representation of what an experienced doctor would do when he or she estimates the effect on the patient of the different interventions available.[3, 4]

Medical science aims to provide diagnostic criteria, treatment indication criteria, and treatments that, when used together, will predict with a highest possible degree of certainty which treatment will work best for each patient (or would not help at all). This old aim is also the aim of 'stratified' or 'personalized' medicine. Such well-designed diagnostic systems would make it easier to choose the best option and to justify it using evidence in the form of data. This will not be possible without a clear understanding of the diagnostic process and criteria for confirming diagnoses that also indicate the best treatment for that patient as discussed in Chapter 13 (see ➔ Evidence-based diagnosis and decisions, p.616).

Diagnostic classifications, pathways, and tables

A diagnostic pathway or algorithm is a way of representing diagnostic reasoning processes or a diagnostic classification (see Fig. 1.1). The same reasoning processes can be displayed using a table of the kind shown in Table 1.1. This is also how information in this book is displayed. It is flexible and also allows findings to be shown that do not form part of the diagnostic criteria. The reader can scan down such a table to find the diagnoses that are compatible with the findings so far. The entry can then be copied into a table in the patient's records as a draft entry for that diagnostic possibility.

Table 1.1 Diagnostic table for the differential diagnoses of jaundice

Carotinaemia (not 'real' jaundice)	*Suggested by:* onset over months. Skin yellow with white sclerae, normal stools, and normal urine. Diet rich in yellow vegetables/fruits. *Confirmed by:* no bilirubin, no **urobilinogen** in the urine, and normal **serum bilirubin**. Normal **liver function tests (LFT)**. Response to diet change.
'Pre-hepatic' jaundice due to haemolysis	*Suggested by:* jaundice and anaemia (the combination seen as 'lemon' or pale yellow). Normal dark stools and normal-looking urine. *Confirmed by:* ↑(unconjugated and thus insoluble) **serum bilirubin**, but normal (conjugated and soluble) bilirubin and thus no ↑bilirubin in urine. ↑**urobilinogen in urine** and ↓**serum haptoglobin**. Normal LFT. ↑**reticulocyte count**.
'Hepatic' jaundice due to congenital enzyme defect	*Suggested by:* jaundice. Normal-looking stools and normal-looking urine. Jaundice worse during febrile illnesses. *Confirmed by:* ↑**serum bilirubin** (unconjugated), but no (conjugated) bilirubin in urine. No **urobilinogen in urine** and **normal haptoglobin**. Normal **LFT**.
'Hepatocellular' jaundice ('hepatic' with some 'obstructive' jaundice)	*Suggested by:* onset of jaundice over days or weeks, pale stools but dark urine. *Confirmed by:* ↑serum (conjugated) **bilirubin** and thus ↑**urine bilirubin**. Normal **urine urobilinogen**. **LFT** all abnormal, especially ↑↑**ALT**.
'Obstructive' jaundice	*Suggested by:* onset of jaundice over days or weeks with pale stools and dark urine. Bilirubin (i.e. conjugated and thus soluble) in urine. *Confirmed by:* ↑ **serum conjugated bilirubin** and **urine bilirubin**, but no ↑**urobilinogen** in urine. Markedly (↑↑) **alkaline phosphatase**, but less abnormal (↑) **LFT** and ↑γ**GT**.

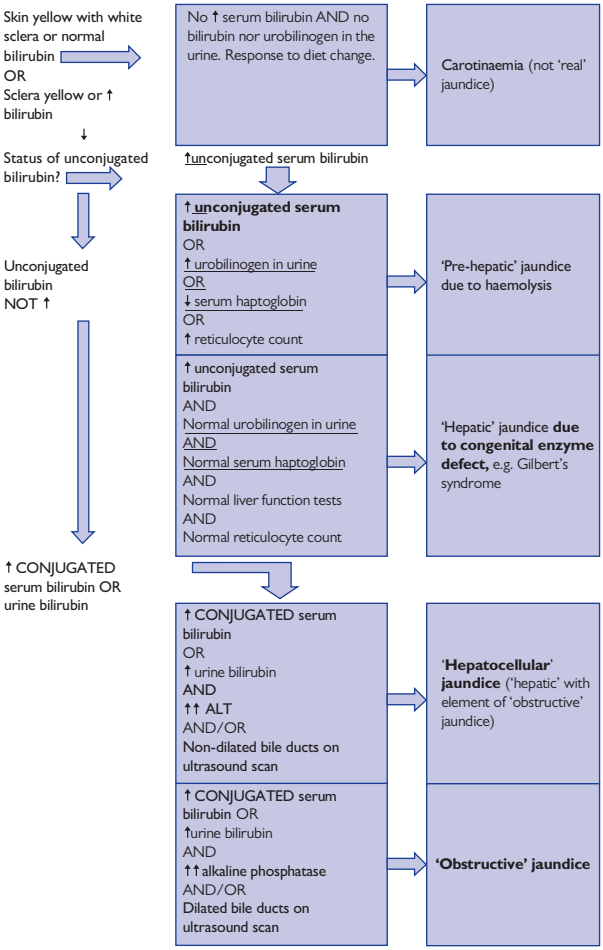

Fig. 1.1 A diagnostic pathway for jaundice.

Dynamic diagnoses

It is important to understand that clinical diagnosis is not a static classification system based on diagnostic criteria or their probable presence. It is a dynamic process. Diagnostic algorithms 'classify' patients by following a logical pathway based mainly on diagnostic criteria. Other systems predict the probable presence of diagnostic criteria. All these methods can be regarded as 'diagnosing' a snap-shot of what is happening at a particular time.

The diagnostician has to imagine the presence of a dynamic process that changes with time. There may be several processes taking place at the same time, some progressing over years (e.g. atheromatous changes), some over minutes to hours (e.g. a thrombosis in a coronary artery), some over minutes or seconds (e.g. ventricular tachycardia), and others instantaneously (e.g. a cardiac arrest).

A diagnostic process leading to treatment may have to happen in stages and for a number of diagnoses at the same time. It might be more appropriate to think of the process as one of 'feedback' control. In this way, the doctor would be acting as an external control mechanism support the patient's failing mechanisms. After the initial history and examination, the feedback information may come from electronic monitoring, nursing observations, ward rounds, hospital clinic, or primary care follow-up.

There are three types of mechanisms of interest to the diagnostician:

- Those that control the 'internal milieu' by keeping temperature, tissue perfusion, blood gases, and biochemistry constant.
- Those that control the body's structure by effecting repair in response to any damage.
- Those that control the 'external milieu' of day-to-day living.

These are all interdependent. If one mechanism fails, then it may unmask other weaknesses by causing other failures. It may not be enough to treat the main failure. It is often necessary also to treat the causes and consequences, as they may be unable to recover on their own. For example, a coronary thrombosis may be treated with stenting of the coronary artery, but any resulting rhythm abnormalities may need to be treated and also the causative risk factors (e.g. smoking) that could result in recurrence. So when we explain our diagnostic thought processes, it helps to think of each diagnosis as a subheading with its own evidence and decision.

The whole patient

A 'diagnosis' does not imply that only one solution needs to be discovered. The complete diagnosis (or diagnostic formulation) may have to include various causes, consequences, interactions, and other independent processes. As well as internal medical processes, it has to include external factors, such as circumstances at home and the effects on self-care, employment, and leisure.

There may be many diagnoses that have been confirmed previously and for which the patient is on established treatment. Therefore, the diagnostician must imagine what is happening to the 'whole patient'. This requires a broad medical education that allows a range of phenomena to be pictured, from molecular events to events in the home and outside world.

Explaining diagnoses to patients

The patient may already be imagining with some trepidation what might be happening. It is important to find out what the patient is imagining and to use this as a starting point for your own explanation. The patient's own views are usually sought and documented at the end of the history of the presenting complaint.

Patients and relatives usually ask questions spontaneously or request an appointment for time to be set aside to do this. Some may be too shy and need encouragement to do so, in which case this important aspect of care will be omitted. Others may be too ill to listen and may prefer relatives or carers to ask on their behalf. If questions are not asked spontaneously, it is best to ask patients tactfully if they or anyone else with their consent have any questions.

Although patients and relatives may understand explanations and other answers to questions at the time they are given to them, even the most intelligent may forget unfamiliar technical terms and their meaning within a short time, especially if they are ill. Therefore, it is important to provide a written reminder of such terms and how they are related. This can be done by giving the patient a printed summary similar to that in An evidence-based diagnosis and plan, p.13. This can also allow the patient to ask further questions if they wish.

Informed consent is also based on similar questions and discussion. The process is more effective if the patient is able to ask the questions (i.e. if the process is 'patient-centred'). Such a process may be facilitated if they refer to a summary such as that shown in An evidence-based diagnosis and plan, p.13.

Ideally, patients should know the presenting complaint for their latest problems, the primary diagnosis or differential diagnoses, and what actions are being taken in terms of tests and treatments. They should also be aware of their past medical history: the various diagnoses, how they presented and were confirmed, their treatments, follow-up arrangements, and markers of progress. Again, the relevant technical terms and how they are linked can be summarized for them as shown in An evidence-based diagnosis and plan, p.13.

Informed consent

In order for a patient to consent to treatment, he or she must understand what has been said and be able to retain that explanation. A basic understanding means the patient must know what actions have been agreed and the possible diagnoses in each case. In order to understand each diagnosis, it is essential to know which symptoms it explains and how these symptoms or some other markers are progressing. Few patients are able to retain all of this, especially if there are many technical terms that are unfamiliar to them. Therefore, it would be a sensible policy to provide the patient with a typed explanation setting out these basic relationships as shown in An evidence-based diagnosis and plan, p.13. This would then become the next 'past medical history' when the patient is asked to provide it by another doctor or nurse. It would thus allow patients to ask a doctor or nurse to remind them of the meanings of the various terms.

Minimizing diagnostic errors

The diagnostic and decision-making process usually takes place in busy clinics, wards, operating theatres, and emergency rooms. Therefore, most diagnoses have to take place by some rapid conscious or subconscious pattern recognition, and there is usually little time for reflection. Mistakes are kept to a minimum by good training, especially listening carefully and writing out what has been observed, thought, and done.

Another important principle to bear in mind is that even the most expert and well-founded diagnoses and decisions can only be successful in a proportion of cases. Therefore, there must be a strategy to monitor their outcome and to change diagnoses and decisions, if possible.

Diagnostic errors can be classified in terms of cognitive psychology[5] into:

- Faulty triggering
- Faulty context information
- Faulty verification
- No fault errors
- Faulty information gathering and processing.

Faulty triggering

This is a failure to consider appropriate diagnostic possibilities, often attributed to a weakness of medical education, which focuses on disease processes instead of the diagnostic processes. This type of error can be kept to a minimum by using the suggestions in the sections from ➔ 'Transparent' reasoning, p.5 to ➔ An evidence-based diagnosis and plan, p.13, and by referring to the differential diagnoses in the other sections. Finally, this error can be reduced by not only writing down the differential diagnoses, but also by writing down the findings from which were chosen the leads that 'triggered' them as shown in ➔ An evidence-based diagnosis and plan, p.13. This can be given to the patient to be shown to other doctors who might also spot any omissions.

Faulty context information

This is focusing on one diagnosis and failing to consider others that may also be present. It involves jumping to conclusions. This can be avoided by using the sieves in ➔ Medical and surgical sieves, p.14, referring to the appropriate section in this book, and writing out an overall plan as shown in ➔ An evidence-based diagnosis and plan, p.13, so that other doctors might spot any errors. Again, this can be given to the patient (to show to other doctors who might spot any errors).

Faulty verification

This is failure to ensure that the patient's presenting symptom and other markers of poor health have been controlled or stabilized as well as possible. This is discussed in ➔ Confirming and finalizing a diagnosis, p.8. It also helps to set out each diagnosis with its evidence as shown in ➔ An evidence-based diagnosis and plan, p.13, which includes the markers being followed and their latest results. Again, this summary can be given to the patient to be shown to other doctors who might spot such omissions.

No fault errors

Even the most expert and well-founded diagnoses and decisions can only be successful in a proportion of cases. This is why diagnoses and decisions are qualified with probabilities. Therefore, there must be a strategy to monitor the outcome of all diagnoses and decisions and to change them, if possible. If a summary of the kind shown in ➔ An evidence-based diagnosis and plan, p.13 is given to the patient to be shown to other doctors, they will be able to understand the basis of previous decisions and take appropriate action.

Faulty information gathering and processing

This is poor use of leads and differentiators in appropriate settings. This book focuses on this process. It is important to know the differential diagnoses of leads and the frequency with which they occur in different clinical settings. It is also important to know the frequency with which findings occur in pairs of diagnoses. At present, this is gained from personal experience. Little research is done into diagnostic leads, differential likelihood ratios, optimizing treatment indication, and diagnostic criteria because the main focus of research is currently on sensitivity, specificity, and overall likelihood ratios. The way in which the situation can be improved is outlined in Chapter 13 (see ➔ Evidence-based diagnosis and decisions, p.616).

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Chapter 2

Interpreting the history and examination

Thoughtful history taking

The aim of the diagnostic process is to build up a picture of what is happening to the patient. 'Diagnosis' is derived from the Greek 'to perceive through knowledge' (i.e. to predict from experience what is beyond the history, examination, etc.).

The diagnosis (or diagnostic formulation) may have to include prediction about past, present, and future, causes, consequences, interactions, and other independent processes. As well as internal medical processes, it has to include external factors such as circumstances at home and the effects on self-care, employment, and leisure.

It is important to establish very clearly why the patient has sought help. This is known as the presenting complaint. Ask about its severity and duration. Be prepared to act immediately to give symptomatic relief (e.g. for pain) if the patient is distressed.

In some cases, the presenting complaint may not explain the decision to seek help. The patient may be too ill, shy, guilty, or embarrassed to describe what is happening accurately. In other cases, it may be someone else who is unduly worried. Be alert to the real reason.

Having established the presenting complaint(s), establish the factual details of 'place and time'. It is the ability to give a place and time that establishes the complaints as 'facts' as opposed to vague 'anecdote'.

Listen without prompting first but, if necessary, ask where they were and what they were doing when the problem was first noticed. This will help the patient's recall and help your diagnostic thought process.

Establish the speed of initial onset and subsequent change in severity with time. Onset within seconds suggests a fit or heart rhythm abnormality, over minutes a bleed or clotting process, hours to days an acute infection, days to weeks a chronic infection, weeks to months a tumour, and months to years a degenerative process.

If there are other complaints, note the same details. Ask about other associated, aggravating, and relieving factors, especially as a result of the patient's own actions and other professional care.

Ask what the patient thinks is going on and is afraid of. This will be the starting point for your own explanation and suggestions to the patient later about what is to be done.

The history also allows patients and supporters to identify the issues that they want addressed in terms of discomfort, loss of function, and difficulties with day-to-day existence. Final diagnoses are based on the initial history because they have to explain it completely. If the diagnoses arrived at cannot explain the entire history and the effects of various treatments, then the diagnoses will be incomplete—others will have to be considered.

Write out your history in a systematic way, e.g. as shown in the next section, and go over it with the patient, if possible, to check that it is right.

This is a lot to remember, especially if you are trying to put it into practice in a busy, noisy environment. However, writing out your findings according to a plan each time will help you to remember.

The plan in Box 2.1 is an example—make up your own.

Box 2.1 A plan for writing out the history

History taker's name: *Date of assessment:*

Patient's name: *DOB:* *Age:* *Occupation:*
Patient's address:

Admitted as an emergency/from the waiting list on (date) at (time)

Presenting complaints (PC)
1st symptom—duration
2nd symptom—duration
etc.

History of each presenting complaint (HPC)
1. Nature of complaint (e.g. pain in chest), circumstances, and speed of onset, progression (change with time—picture a graph), aggravating and relieving factors, associated symptoms (describe under 2, etc.)
2. Next associated symptom, etc. described as in (1).

Add response to direct questions from chasing up some diagnostic possibilities that come to mind as the history is taken.

Add the patient's opinion or fear about what may be happening.

Past medical history (PMH)
1st diagnosis and when—evidence—treatment—name of doctor
2nd

Drug history (DH)
Name, dose, and frequency—diagnostic indication—evidence—prescriber
Next drug etc.
Alcohol and tobacco consumption, other 'recreational' drugs
Drug sensitivities and allergies

Developmental history
(especially in paediatrics and psychiatry): pregnancy, infancy, childhood, puberty, adulthood

Family history (FH)
Age Illnesses
(Arrange around 'family tree', if preferred)
Mention especially:

Parents	*Tuberculosis?*
Siblings	*Asthma? Eczema?*
Children	*Diabetes? Epilepsy?*
Spouse	*Hypertension?*

Social history (SH)
Home and domestic activity support—job and financial security—travel and leisure. (Consider the effect of all these on the illness and the effect of the illness on these.)

History by A N Other 3.00 p.m. 19 October 2013
Miss AM (DOB: 28/2/85) Aged 29 Secretary
23, Smith Square, Old Town
Emergency admission 19 October 2013 at 2.00 p.m.

PC:

1. Severe sore throat, sweats, and malaise for 2 days

2. Sudden loss of consciousness in A&E at 2.00 p.m.

***HPC**: The patient was well until last Friday afternoon 18 October when she developed a **sore throat** at work. It was relieved that day by warm drinks and paracetamol, but when she woke this Saturday morning, it was very severe. She remembered that she had been told to report sore throats because she was taking carbimazole and to get a white cell count. She was worried that she might have developed a low white cell count because of this drug. She came to A&E because it was a Saturday. When she got up from her seat in the waiting room after being called, she felt dizzy, blacked out, and fell to the floor, striking her head. She recovered consciousness within a minute.*

Interpreting the case history

There are two striking symptoms: (1) a severe sore throat that is getting worse and (2) the sudden loss of consciousness. Both are examples of findings with short lists of causes: good diagnostic 'leads' or 'pivots'.

Most readers will have experienced a sore throat and will be aware that it is usually due to a viral pharyngitis, bacterial tonsillitis (e.g. due to a haemolytic *streptococcus*), or glandular fever. It could also be due to bone marrow dysfunction (e.g. due to drug effect) or something else in a small proportion of cases (see Sore throat, p.31). The onset over days is compatible with all these possibilities. A white cell count might give results that would differentiate between these possible causes (see Table 2.1).

The sudden loss of consciousness with rapid recovery is known as 'syncope'. It is also a good lead with a well-defined differential diagnosis. It can be due to a vasovagal attack, cough, micturition or carotid sinus syncope, postural hypotension, transient cerebral ischaemia, a Stokes–Adams attack, aortic stenosis, hypertrophic cardiomyopathy (HOCM), hypoglycaemia, or epilepsy (see Table 2.1). The fact that it happened after the patient got up from a chair suggests postural hypotension (because this always occurs in this condition, but rarely, if ever, in the others). Postural hypotension may be due to fever and dehydration so although the two leads have common causes, postural hypotension could be a consequence of any infection. Therefore, the syncope does not differentiate between any of the causes of a sore throat. The patient has expressed a fear that the sore throat could be drug-induced because she has been warned about this.

These thoughts can be summarized in the problem-structuring note in Table 2.1. You can write this on a sheet of paper, perhaps in pencil for easy editing, on a computer, or on a black or white board when discussing a case with colleagues. Such thoughts are usually considered mentally without writing them down, which is why the diagnostic thought process can be difficult to learn from senior colleagues.

Outline findings

After outlining your thoughts in the problem-structuring note as shown in Table 2.1, turn to the appropriate page in this book by looking up 'sore throat'. Check that you have not forgotten to include something. The entry in this book for 'sore throat' is shown in ➔ Sore throat, p.31 and in ➔ Sore throat, p.320. You may wish to read this before moving on to the next step.

Table 2.1 Female. Aged 29. Severe sore throat for 2 days, getting worse. Taking carbimazole for 6 months. Sudden loss of consciousness after getting up from chair, recovery within a minute.

Diagnoses	Outline evidence	Management
Viral pharyngitis?	Severe sore throat for 2 days, getting worse. (19/10/13)	Paracetamol 500mg 6 hourly PRN. Examine throat. Request **WCC**: ↓neutrophils, ↑lymphocytes?
Acute bacterial (or follicular) tonsillitis (mainly streptococcal)?	Severe sore throat for 2 days, getting worse. (19/10/13)	Paracetamol 500mg 6 hourly PRN. Examine throat. Request **WCC**: ↑neutrophils?
Glandular fever (infectious mononucleosis due to Epstein–Barr virus)?	Severe sore throat for 2 days, getting worse. (19/10/13)	Paracetamol 500mg 6 hourly PRN. Examine throat. Request **WCC:** lymphocytes atypical? Paul–Bunnell or Monospot® +ve?
Drug-induced agranulocytosis? (this is what the patient fears)	Severe sore throat for 2 days, getting worse (19/10/13). Taking carbimazole.	Paracetamol 500mg 6 hourly PRN. Examine throat. Request **WCC**: ↓granulocytes (neutrophils, eosinphils, basophils)?
Postural hypotension syncope? Due to dehydration?	Sudden loss of consciousness after getting up from chair, recovery within a minute (19/10/13). Evidence of acute infection.	Look for fall in BP when standing. Request U&E. Consider fluids IV to rehydrate.
Thyrotoxicosis now controlled?	Taking carbimazole.	Examine for tremor, etc. Carbimazole 5mg od. FT4 and TSH normal?

Sore throat

Initial investigations (other tests in bold): FBC, U&E, throat swab, Paul–Bunnell test (Table 2.2).

Table 2.2 Main differential diagnoses and typical outline evidence, etc.

Viral pharyngitis	*Suggested by:* sore throat, pain on swallowing, fever, cervical lymphadenopathy, and injected fauces. WCC: ↑lymphocytes, leucocytes normal. *Confirmed by:* negative **throat swab** for bacterial culture, self-limiting: resolution within days. *Initial management:* analgesics, e.g. paracetamol.
Acute follicular tonsillitis (streptococcal)	*Suggested by:* severe sore throat, pain on swallowing, fever, enlarged tonsils with white patches (like strawberries and creamy lines). Cervical lymphadenopathy, especially in angle of jaw. Fever, WCC: ↑leucocytes. *Confirmed by:* **throat swab** for culture and sensitivities of organisms. *Initial management:* analgesics, antibiotics, e.g. benzyl-penicillin IM or Penicillin V orally; if no allergy, good fluid intake.
Infectious mononucleosis (glandular fever) due to Epstein–Barr virus	*Suggested by:* very severe throat pain with enlarged tonsils covered with grey mucoid membrane. Petechiae on palate. Profound malaise. Generalized lymphadenopathy, splenomegaly. **WCC**: ↑atypical lymphocytes. *Confirmed by:* **Paul–Bunnell or Monospot® test** +ve. **Viral titres**: ↑Epstein–Barr. *Initial management:* analgesia, no antibiotics (amoxicillin may cause skin rash).
Candidiasis of buccal or oesophageal mucosa	*Suggested by:* painful dysphagia, white plaque, history of immunosuppression/diabetes/recent antibiotics. *Confirmed by:* **oesophagoscopy** showing erythema and plaques, **brush cytology:** spores and hyphae. *Initial management:* local antifungal agents, e.g. miconazole oral gel or nystatin oral suspension. Parenteral administration if systemic involvement.
Agranulocytosis	*Suggested by:* sore throat, background history of taking a drug, or contact with noxious substance. *Confirmed by:* ↓ or absent neutrophil count. *Initial management:* stop potential causative drugs, antibiotic cover until resolved.

The systems enquiry

The systems enquiry may take place at various points in the history. The questions given here are detailed. They can also be asked as broad prompts (e.g. do you have any chest, abdominal, bladder symptoms, etc.?). Some may prefer to perform the systems enquiry immediately after the history of presenting complaint because they would not have enough knowledge to ask the questions to differentiate between the initial differential diagnoses (e.g. asking about generalized lymph node enlargement that might differentiate between glandular fever and the other causes of a sore throat). If the patient said 'yes' to a question during the systems enquiry, it could be added to the problem-structuring note and looked up later in this book.

If a direct question turns up a positive response, it has to be treated with caution. It may be a 'false-positive' response to a leading question. A positive response has to be treated as an extra presenting complaint, added to the original list and explored carefully with the history of presenting complaint. They can also be looked up in the pages of this book.

If there is a negative response to a direct question, this is more reliable (unless the patient is very forgetful or is purposely withholding information). The absence of all symptoms under a heading indicates that it is less probable that there is an abnormality in that system.

Systems enquiry

Locomotor symptoms

- No pain and stiffness in the neck, shoulder, elbow, wrist, hand, or back
- No pain and stiffness in the hip, knee, or foot
- No pain or stiffness in any joints and muscles.

Negative responses make locomotor abnormalities less probable. If any are positive, then a 'GALS' examination screen is performed under the headings of Gait, Arms, Legs, Spine. Care can be taken with painfully inflamed or damaged joints.

Skin, lymph nodes, and endocrine

No heat or cold intolerance (e.g. wanting to open or close windows when others are comfortable).

Sweats and shivering for 2 days

- No drenching night sweats
- No episodes of rigors
- No rashes and itching.

No skin lumps or lumps elsewhere

No heat or cold intolerance makes an abnormality of thyroid metabolism less probable (suggesting that the carbimazole is probably controlling the thyrotoxicosis). Positive findings (e.g. sweats and shivering for 2 days) can be looked up in this book—they will be found to be poor leads and differentiators (because they occur often in each condition), and not very helpful in differentiating between the causes of a sore throat.

No further information is gained with the following responses. However, they provide an opportunity to reflect on the function of each system.

Cardiovascular symptoms

No tiredness and breathlessness on exertion (non-specific)
Syncope after rising from chair in A&E—see HPC
No leg pain on walking

Negative responses make cardiac output and peripheral vascular disease less probable.

No ankle swelling

A negative response makes a right-sided venous return abnormality less probable.

No exertional dyspnoea
No orthopnoea
No paroxysmal nocturnal dyspnoea

Negative responses make a left-sided venous return abnormality less probable.

No palpitations
No central chest pain on exertion or at rest

Negative responses make a cardiac abnormality less likely.

Respiratory symptoms

No chronic breathlessness
No acute breathlessness

Negative responses make abnormality of overall respiratory and blood gas abnormality less probable.

No hoarseness
No cough, sputum, haemoptysis
No wheeze

Negative responses make airway disease less probable.

No pleuritic chest pain

A negative response makes pleural reactions and chest wall disease less probable.

Alimentary symptoms

No loss of appetite (non-specific)
No weight loss (non-specific)
No jaundice, dark urine, pale stools
Negative responses make metabolic gut and liver disease less probable.
No nausea or vomiting (non-specific)
No haematemesis or melaena
No dysphagia but ***sore throat—see HPC***
No indigestion
No abdominal pain
No diarrhoea or constipation
No recent change in bowel habit
No rectal bleeding ± mucus

Negative responses make gastrointestinal disease less probable.

Genitourinary symptoms

Menstrual history—date of menarche, duration of cycle, and flow normal
Volume of flow and associated pain normal
Any pregnancy outcomes normal
No dyspareunia and vaginal bleeding
No vaginal discharge

Negative responses make gynaecological disease less probable.

No haematuria or other odd colour
No urgency or incontinence
No dysuria
No polyuria or nocturia
No loin pain or lower abdominal pain

Negative responses make urological disease less probable.

No impotence or loss of libido
No urethral discharge

Negative responses make male urological disease less probable.

Nervous system symptoms

No vision loss, blurring, or double vision
No hearing loss or tinnitus
No loss of smell and taste
No numbness, pins and needles, or other disturbance of sensation
No disturbance of speech
No weakness of limbs
No imbalance
No headache
No sudden headache and loss of consciousness

Dizziness and blackouts in A&E—see HPC
No vertigo
No 'fit'
No transient neurological deficit

Negative responses make neurological disease less probable.

Psychiatric symptoms

No fatigue, not tired all the time
No mood change
No odd voices or odd visual effects
No anxiety and sleep disturbance
No loss of self-confidence
No new strong beliefs
No phobias, no compulsions, or avoidance of actions
No use of recreational drugs

Patients may hide or forget many symptoms. There is a school of thought that regards symptom reviews as being of little value, and that only symptoms that are volunteered are worthwhile investigating. Many doctors do not conduct systemic reviews and only ask these questions if other symptoms have been volunteered already in that system.

The past medical history

The past medical history (PMH) in this case has three components: the diagnosis, the evidence, and the management. The management may be omitted if it is mentioned elsewhere, e.g. if carbimazole is in the drug history together with its indication of thyrotoxicosis.

PMH

Thyrotoxicosis discovered 6 months ago

Outline evidence: anxiety, weight loss, abnormal thyroid function tests in Osler Hospital by Dr Miller.

Management: taking carbimazole, 5mg daily.

'Anxiety, weight loss, abnormal thyroid function tests' outlines the evidence for the thyrotoxicosis. Knowing the doctor responsible and the institution would allow the details to be checked, if necessary. In many cases, patients are not able to provide these details and they would have to be extracted from the records, in which case it is helpful to name the hospital or primary care centre or doctor responsible.

A comprehensive past medical history in this format could be written immediately after any consultation, in hospital or primary care with results and dates given to the patient. This would be more reliable than the next doctor having to do so, but this is not customary. This information can be added to the problem-structuring note No. 2. This can be set out in different formats; in this case, it is set out in subheading style, which is in effect a draft of the 'next' past medical history. This problem-structuring approach can also be used to draft discharge summaries on a hospital computer network, which can be updated during the patient's stay and printed out when the patient leaves hospital.[1]

The family history

The family history (FH) (Table 2.3) rarely contains features that form powerful leads. In general, there will be risk factors in the FH. For example, the fact that the patient's mother had type 2 diabetes mellitus means that there is an increased risk of the patient developing type 2 diabetes mellitus. This may have no immediate bearing on the current problems (but she should be checked for diabetes if only to exclude its presence so far). The patient could also be reminded to adopt a healthy diet and lifestyle. The new additions to the problem-structuring note (Box 2.2) are in **bold**.

Table 2.3 FH

Father		Aged 56—hypertension
Mother		Aged 55—diabetes (onset at 50)
Siblings	male	Aged 34—alive and well
		Aged 26—alive and well
	female	Aged 30—alive and well
Children		None

Box 2.2 Problem-structuring note No. 2

Outline findings: female. Aged 29. Severe sore throat for 2 days, getting worse. Taking carbimazole for 6 months. Sudden loss of consciousness after getting up from chair, recovery within a minute. PMH of thyrotoxicosis (anxiety, weight loss, abnormal thyroid function tests in Osler Hospital) treated with carbimazole. **FH of type 2 diabetes mellitus**.

Viral pharyngitis?
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13).

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↓neutrophils, ↑lymphocytes?

Acute bacterial (or follicular) tonsillitis? (mainly streptococcal)
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13).

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↑neutrophils?

Glandular fever (infectious mononucleosis due to Epstein–Barr virus)?
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13). No skin lumps or lumps elsewhere.

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: lymphocytes atypical? Paul–Bunnell or Monospot® +ve?

Drug-induced agranulocytosis? (this is what the patient fears)
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13). Taking carbimazole. Bruising on forehead.

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↓granulocytes (neutrophils, eosinophils, basophils)?

Postural hypotension syncope? Due to dehydration from infection?
Outline evidence: sudden loss of consciousness after getting up from chair, recovery within a minute (19/10/13). Evidence of acute infection.

Management: look for fall in BP when standing. Request U&E. Consider fluids IV to rehydrate.

Thyrotoxicosis now controlled?
Outline evidence: **anxiety, weight loss, abnormal thyroid function tests in April 2008**. No heat or cold intolerance currently.

Management: examine for tremor, etc. Carbimazole 5mg od. FT4 and TSH normal?

Increased risk of type 2 diabetes mellitus
Outline evidence: **FH of type 2 diabetes mellitus.**
Management: **test urine for sugar. Fasting glucose.**

The version of the problem-structuring note in Box 2.2 is in the same format as the 'textbook' page on 'Sore throat' (see ➔ Sore throat, p.31 and ➔ Sore throat, p.320). This makes comparison easier and allows the 'textbook' entries to be used as templates that can be copied into the problem-structuring note.

The drug history

The drug history (DH) is often placed near the end of the history. If the patient is on medication, then it indicates that this is for an active medical condition as opposed to a PMH. Therefore, there is something to be said for documenting the drug history immediately after the PMH so and current conditions can be thought about together.

Drug history
Paracetamol 1g 6 hourly (for ?viral pharyngitis, etc.)
Carbimazole 5mg daily for thyrotoxicosis (see PMH for evidence)
Alcohol 10 units per week
Non-smoker
No other recreational drugs

There is nothing to add to the problem-structuring note from the drug history.

The social history

The social history (SH) is always relevant. The activities of daily living can be considered under the heading of domestic, work, and leisure. Imagine what any person has to do from waking up in the morning to going to sleep at night, and consider whether the patient needs support with any of these activities. Fit young adults who are expected to recover completely may miss school, college, or work, and the timing of their return will have to be considered. Patients who are more dependent on others, such as children and the elderly, may need special provisions. Patients with permanent disabilities may need help with most, if not all, activities of daily living.

SH
Alone in a flat at present (flatmate on holiday for another week)
Parents live 200 miles away
Works as secretary for insurance firm

The patient has little domestic support and it would be sensible to admit her to be rehydrated until she is in no danger of fainting on discharge. This has been added to the problem-structuring note.

When the history is complete

The findings that will differentiate between the causes of a sore throat (see ➔ Sore throat, p.31) are the appearance of the throat and the white cell count. Generalized lymphadenopathy, splenomegaly, and petechiae on the palate would also occur commonly in glandular fever and uncommonly in the other differential diagnoses.

➔ Postural fall in blood pressure, p.212 shows that a fall in BP on standing would support postural hypotension because it occurs commonly in patients with this diagnosis and rarely in the other causes of syncope. A raised creatinine and urea would support dehydration because this

happens often in dehydration, but infrequently in the other causes of postural hypotension.

The diagnostic thoughts so far are represented in the problem-structuring note in Table 2.4.

Table 2.4 Problem-structuring note No. 3

Outline findings: female. Aged 29. Severe sore throat for 2 days, getting worse. Taking carbimazole for 6 months. Sudden loss of consciousness after getting up from chair, recovery within a minute. PMH of thyrotoxicosis (anxiety, weight loss, abnormal thyroid function tests). FH of type 2 diabetes mellitus.		
Viral pharyngitis?	Severe sore throat for 2 days, getting worse. (19/10/13)	Paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↓neutrophils, ↑lymphocytes?
Acute bacterial (or follicular) tonsillitis? (mainly streptococcal)	Severe sore throat for 2 days, getting worse. (19/10/13)	Paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↑neutrophils?
Glandular fever (infectious mononucleosis due to Epstein–Barr virus)?	Severe sore throat for 2 days, getting worse. (19/10/13)	Paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: lymphocytes atypical? Paul–Bunnell or Monospot® +ve?
Drug-induced agranulocytosis? (this is what the patient fears)	Severe sore throat for 2 days, getting worse (19/10/13). Taking carbimazole.	Paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↓granulocytes (neutrophils, eosinophils, basophils)?
Postural hypotension syncope? Due to dehydration from infection?	Sudden loss of consciousness after getting up from chair, recovery within a minute (19/10/13). Evidence of acute infection.	Look for fall in BP when standing. Request U&E. Consider fluids IV to rehydrate.
Thyrotoxicosis now controlled?	Anxiety, weight loss, abnormal thyroid function tests in April 2008. No heat or cold intolerance currently.	Examine for tremor etc. Carbimazole 5mg od. FT4 and TSH normal?
Increased risk of type 2 diabetes mellitus	FH of type 2 diabetes mellitus.	Test urine for sugar. Fasting glucose.
No domestic support	**Alone in flat at present.**	**Consider admission for initial care.**

Interpreting the physical examination

The physical examination tends to be focused. The 'open mind' approach, where findings are discovered and their meaning looked up later, is described at the end of this section. If this book is referred to before the examination, the reader could focus on the appearance of the throat and palpation of the neck to look for findings that may differentiate between the four differential diagnoses suggested by the history. The reader should also focus on the BP to see if there is a postural fall, and tremor and lid lag for inadequately treated thyrotoxicosis.

General

Looks unwell, flushed
No tremor or lid lag
Temperature 38.5°C
Bilaterally swollen tonsils, red with linear creamy patches
Bilateral, tender, multiple lymph node enlargement in neck. No lymph node swelling in axillae or groins

CVS

Pulse 110/min, regular, low volume
BP 110/70 reclining, 90/50 standing
Heart sounds normal
No murmurs

RS

Chest shape and movement normal
Breath sounds normal

AS

Not jaundiced
Liver—1 finger breadth below costal margin
Spleen not palpable

CNS

Conscious and alert
No neck stiffness
Hand and leg coordination normal
Reflexes all normal and symmetrical

The presence of linear patches of creamy pus in fissures on the surface of enlarged tonsils occurs commonly in patients with bacterial tonsillitis, but less commonly in agranulocytosis, viral pharyngitis, and glandular fever (where there is usually a grey mucoid film). This finding changes the order of the differential diagnoses, but they all remain possible. A high temperature and lymph node enlargement around the jaw occur in all the differential diagnoses of a sore throat and is a poor differentiator. There was no tremor and lid lag to suggest inadequately treated thyrotoxicosis.

The fall in BP when the patient stands up always occurs at some point in postural hypotension and uncommonly in its other differential diagnoses.

Therefore, the order of the possible diagnoses has changed; this is shown in the problem-structuring note in Box 2.3. The format has also changed again from a three-column chart to heading and subheadings.

Box 2.3 Problem-structuring note No. 4

Outline findings: female. Aged 29. Severe sore throat for 2 days, getting worse. Taking carbimazole. Sudden loss of consciousness after getting up from chair, recovery within a minute. PMH of thyrotoxicosis. FH of type 2 diabetes mellitus. No tremor, no lid lag, reflexes normal. Large red tonsils, **linear creamy patches**. Fall in BP on standing.

Acute bacterial (or follicular) tonsillitis? (mainly streptococcal)
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13). **Large red tonsils with linear creamy patches.**

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↑neutrophils?

Glandular fever (infectious mononucleosis due to Epstein–Barr virus)??
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13). **Large red tonsils with linear creamy patches.**

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: lymphocytes atypical? Paul–Bunnell or Monospot® +ve?

Viral pharyngitis?? (less probable)
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13). **Large red tonsils with linear creamy patches.**

Management: paracetamol 500mg 6 hourly PRN. Examine throat. Request WCC: ↓neutrophils, ↑lymphocytes?

Drug induced agranulocytosis? (less probable)
Outline evidence: severe sore throat for 2 days, getting worse (19/10/13). On carbimazole. **Large red tonsils with linear creamy patches.**

Management: paracetamol 500mg 6 hourly PRN. Request WCC: ↓granulocytes (neutrophils, eosinophils, basophils)?

Postural hypotension syncope? Due to dehydration from infection?
Outline evidence: sudden loss of consciousness after getting up from chair, recovery within a minute (19/10/13). **Fall in BP on standing**. Evidence of acute infection.

Management: request U&E. Consider fluids IV to rehydrate.

Thyrotoxicosis now controlled?
Outline evidence: anxiety, weight loss, abnormal thyroid tests in April 2008. No heat or cold intolerance. No tremor, no lid lag, reflexes normal.

Management: carbimazole 5mg od. FT4 and TSH normal?

Increased risk of type 2 diabetes mellitus
Outline evidence: FH of type 2 diabetes mellitus.

Management: test urine for sugar. Fasting glucose.

No domestic support
Outline evidence: alone in a flat at present. Parents 200 miles away.
Management: consider admission for initial care.

Interpreting the investigations

Investigations tend to be performed in a focused way like the physical examination. This means that they are done in order to differentiate between diagnostic possibilities created by the history and examination. However, urine testing, full blood count, urea and electrolytes (U&E), and CXR are often done routinely in the same way as aspects of the physical examination, such as the pulse, temperature, and BP. These are done in case that they will reveal a result that is a good diagnostic lead. This is a form of screening, but if the result is abnormal, then it is investigated in the same way as a presenting complaint. In this case, all the tests, except the CXR, were done in order to differentiate between the diagnostic possibilities, and most of the results were helpful.

Investigations

Urine testing: + glucose, no protein, no blood, no ketones

FBC: Hb 12.4g/dL
WCC 19.3×10^9/L, neutrophils 90%
No atypical lymphocytes present

Lab blood glucose 8.4mmol/L
Na^+ 141mmol/L, K^+ 4.3mmol/L, urea 10.1mmol/L, creatinine 112micromol/L
TSH, T4—results awaited

Monospot® test—result awaited
Throat swab—result awaited

CXR normal

The presence of glucose in the urine and the random glucose of 8.4mmol/L is suspicious of diabetes mellitus. The WCC of 19.3×10^9/L with 90% neutrophils occurs commonly in bacterial tonsillitis, but never (by definition) in agranulocytosis. This is also very rare in viral pharyngitis and glandular fever so that all these diagnoses drop out of contention. The raised creatinine and urea are common in dehydration and less common in other causes of postural hypotension.

The problem-structuring note in Table 2.5 shows how the diagnostic opinions and management have changed in the light of these test results.

Medical and surgical sieves

At this point, you can pause and use the medical and surgical sieves from ➔ Medical and surgical sieves, p.14. You can consider whether you have omitted a diagnosis in the social background or environment, the locomotor, nervous, cardiovascular, respiratory, and alimentary systems, the renal system and urinary tract, and the reproductive, endocrine, autonomic, haematological, and immune systems. Within each of these systems, you can consider whether you have forgotten a congenital, infective, traumatic, neoplastic, or degenerative process. If not, you can move on.

Table 2.5 Problem-structuring note No. 5

Outline findings: female. Aged 29. Severe sore throat for 2 days, getting worse. Taking carbimazole. Sudden loss of consciousness after getting up from chair, recovery within a minute. PMH of thyrotoxicosis. FH of type 2 diabetes mellitus. No tremor, no lid lag. Large red tonsils with linear creamy patches. Fall in BP on standing. Urine testing: +ve glucose. Hb 12.4g/dL, WCC 19.310^9/L, neutrophils 90%, no atypical lymphocytes present. Lab blood glucose 8.4mmol/L. Urea 10.1mmol/L. Creatinine 112micromol/L.

Acute bacterial (or follicular) tonsillitis (causing systemic effects, e.g. dehydration)	Severe sore throat for 2 days, getting worse (19/10/13). Large red tonsils with linear creamy patches. **WCC of 19.3x10^9/L with 90% neutrophils.**	Paracetamol 500mg 6 hourly PRN. Begin phenoxymethylpenicillin 500mg qds.
Probably not glandular fever (infectious mononucleosis due to Epstein–Barr virus)?	Severe sore throat for 2 days, getting worse (19/10/13). Large red tonsils with linear creamy patches. **WCC of 19.3x10^9/L with 90% neutrophils.**	Paracetamol 500mg 6 hourly PRN. Examine throat. Await Monospot® result.
Postural hypotension syncope? Due to dehydration from infection?	Sudden loss of consciousness after getting up from chair, recovery within a minute (19/10/13). Fall in BP on standing. Evidence of acute infection.	Fall in BP when standing. Request U&E. Consider fluids IV to rehydrate.
Dehydration from infection?	Fall in BP on standing. Evidence of acute infection. Urea 10.1mmol/L. Creatinine 112**micromol**/L.	Admit. Encourage oral fluids. For fluids IV if unable to drink 2L in 12h.
Thyrotoxicosis now controlled?	Anxiety, weight loss, abnormal thyroid function tests in April 2008. No heat or cold intolerance. No tremor or lid lag. Reflexes normal.	Carbimazole 5mg od. Await result of FT4 and TSH.
Probable type 2 diabetes mellitus	FH of type 2 diabetes mellitus. Urine glucose +ve. No ketones. Random glucose 8.4mmol/L.	Monitor blood sugar before and 2h after meals. Plan glucose tolerance test.
No domestic support	Alone in a flat at present. Parents 200 miles away.	Admit for initial care.

Writing the diagnosis and management

The positive finding summary could be written out as follows:

Female. Aged 29. Severe sore throat for 2 days, getting worse. Taking carbimazole for 6 months. Sudden loss of consciousness after getting up from chair, recovery within a minute. PMH of thyrotoxicosis (anxiety, weight loss, abnormal thyroid function tests). FH of type 2 diabetes mellitus. Large red tonsils with linear creamy patches. Fall in BP on standing. Urine testing: +ve glucose. Hb 12.4g/dL, WCC 19.3×10⁹/L, neutrophils 90%, no atypical lymphocytes present. Lab blood glucose 8.4mmol/L, urea 10.1mmol/L, creatinine 112 micromol/L.

The primary diagnosis (that explains the symptoms that led the patient to seek help) can be written as:

Primary diagnosis:
- *Probable acute bacterial (or follicular) tonsillitis (causing systemic effects)*

The other diagnoses can be written as:

Other diagnoses:
- *Postural hypotension syncope due to dehydration from infection*
- *Thyrotoxicosis probably now controlled*
- *Probable type 2 diabetes mellitus*
- *No domestic support currently*

The initial plan can be written as:

Plan:
- *Reassure patient that there is no agranulocytosis and explain other diagnoses*
- *Start phenoxymethylpenicillin 500mg qds (because of systemic effects)*
- *Continue paracetamol 1g qds*
- *Continue carbimazole 5mg od*
- *Encourage oral fluids (e.g. 2L in 16h)*
- *Monitor blood glucose before and 2h after meals*
- *Help patient to contact family*

It should be noted that this traditional way of writing out the findings does not give the reader an indication of the writer's thought process. It does not provide the particular evidence for each diagnosis or specify at which diagnosis each aspect of the management is directed. This is the approach mostly used in discharge summaries when patients are discharged from hospital. In contrast to this, the problem-structuring notes used here do provide this information.

Case presentations

If you are asked to give a case presentation, then in addition to the positive findings, you should mention negative features. These will imply that you have considered other diagnoses, but were unable to find the supportive features (i.e. that you considered those negative findings to differentiate between your probable diagnosis and those you consider improbable). The information that you require for your case presentation will be found in the 'evidence' column of the latest version of your problem-structuring notes. It is customary to give the history of presenting complaint in some detail, as follows in Box 2.4.

Box 2.4 Case presentation of Ms AM

Ms AM is a 29-year old secretary who came to the A&E department with a severe sore throat, sweats and malaise for 2 days. She also lost consciousness briefly in A&E 30 minutes after arriving.

She was well until last Friday afternoon 18 October when she developed a ***sore throat*** *at work. It was relieved that day by warm drinks and paracetamol, but when she woke this morning, it was very severe. She remembered that she had been told to report sore throats because she was taking carbimazole and to get a white cell count. She was worried that she might have developed a low white cell count because of this drug. She came to A&E because it was a Saturday morning. When she got up from her seat in the waiting room after being called, she felt dizzy, 'blacked out', and fell to the floor, striking her head. She recovered consciousness within a minute.*

There is a past medical history of thyrotoxicosis (as evidenced last April by anxiety, weight loss, abnormal thyroid function tests). There is a family history of type 2 diabetes mellitus. She shares a flat with a friend who is away at present.

On examination, she looked tired and unwell. Her temperature was 38.5. Her pharynx was red with enlarged tonsils, which showed linear creamy patches. There was lymph node enlargement in the angles of the jaw but not elsewhere. Her pulse was 110/min and regular. The BP was 110/70 reclining and 90/50 standing. The heart sounds were normal and there were no murmurs. The chest was clear. The abdomen was soft and there was no splenic enlargement.

Urine testing showed one plus of glucose but no ketones. The white cell count was 19.3×10^9/L, the neutrophils being 90%. There were no atypical lymphocytes present. The laboratory random blood glucose was 8.4mmol/L, the urea was 10.1mmol/L and the creatinine 112micromol/L.

Clinical opinions

After giving a case presentation, you will be asked to give a clinical opinion and expected to provide the (particular) evidence for your diagnoses. You may be asked if you do not volunteer this information first. The opinion could be based on the latest problem-structuring note.

Clinical opinion on Ms AM

The probable diagnosis is acute follicular tonsillitis (causing systemic effects, e.g. dehydration). This is because she has had a severe sore throat for 2d, there were large red tonsils with linear creamy patches and a white cell count of 19.3×10^9/L with 90% neutrophils. This should be treated with benzyl-penicillin IM or Penicillin V orally because of the systemic effects and the symptoms treated with paracetamol.

There is probably no infectious mononucleosis or agranulocytosis because of the raised neutrophils and absence of atypical lymphocytes. She should be reassured about this.

She has suffered postural hypotension syncope because of the sudden loss of consciousness after getting up from chair with recovery within a minute and the fall in BP on standing. She should not be discharged home until this problem has resolved with rehydration.

She is probably dehydrated from infection because of the pulse of 110/min, fall in BP on standing, urea of 10.1mmol/L, and creatinine of 110micromol/L. Fluids need to be encouraged.

The thyrotoxicosis appears to be controlled. The original anxiety and weight loss have resolved and there was no heat or cold intolerance. There was no tremor or lid lag. She should continue on carbimazole 5mg od, pending the result of T4 and TSH measurements.

She probably has type 2 diabetes mellitus because of the FH of this and the random blood sugar of 8.4mmol/L with no urine ketones. She is to have two fasting blood sugars, and her blood sugars monitored before and 2 hours after meals during the admission. A glucose tolerance test will be done if the fasting sugar is not less than 5.6mmol/L or not more than 7.0mmol/L on two occasions.

She has little domestic support because she lives alone in her flat this weekend and her parents live 200 miles away. She will be admitted and kept in hospital until she is well enough to self-care.

The 'open mind' approach

The preceding paragraphs described how diagnostic hypotheses were generated as soon as the presenting complaints had been heard. These were displayed in the problem-structuring notes. This approach requires the history taker to have the knowledge to identify the best leads and to know which items of information will differentiate between the possible diagnoses. Alternatively, it depends on the history taker looking up the information in the OHCD at different stages in the history and examination and when the test results become available.

The other option is to take the history and to examine the patient in a mechanical way, without interpreting the findings as they are discovered. The abnormal findings can then be listed at the end and then looked up in the OHCD. The thought process would then follow the same pattern as that described in the problem-structuring notes.

As the history and examination is being performed and the results become known, differential diagnoses may also occur to the assessor consciously or subconsciously in a passive way. This will depend on the assessor's knowledge, which can be helped by reading this book during private study. This can be done by covering the list of diagnoses, looking at the diagnostic lead above the list, and then reading the suggestive and confirmatory findings. The reader should then try to guess the hidden diagnosis and then see if he or she was correct.

The plan of the remainder of this book

An example of a systems enquiry has been given already in ➔ The systems enquiry, p.32. The following shows a typical example of the routine physical examination on which the remainder of this book is based.

The 'routine' physical examination

Note the patient's attire, presence of nebulizer masks, sputum pots, medication packets, etc. The general examination is directed mainly at assessing the skin and reticulo-endothelial system (lymph nodes), and the related matters of temperature control and metabolic rate. During the history, the order of questioning could be decided entirely by thought processes (e.g. probing indirectly for a symptom to chase up a diagnostic possibility that comes to mind), but the physical examination is different. It is more efficient to adopt a routine that is smooth and quick, and not to jump about looking for physical signs that might support the diagnostic idea of that moment.

You have already been looking at the patient's face, general appearance, and immediate vicinity (e.g. walking stick, medication packets, etc.) when taking the history. So for the general examination, begin with the hands and work your way up by inspecting (and, when appropriate, palpating) the arms to the shoulders, examine the scalp, ears, eyes, cheeks, nose, lips, take the temperature, examine inside the mouth, then the neck, breasts, axillae, and then the skin of the abdomen, legs, and feet.

Plan of the general examination

Hands, arms, and shoulders

- *Fingernails*
- *Clubbing*
- *Finger nodules*
- *Finger joint deformity*
- *Rashes*
- *Pain and stiffness in the elbow, shoulder, neck.*

Head and neck

- *Neck stiffness*
- *Patchy hair loss*
- *Eardrum redness*
- *Perforated eardrum.*

Eyes, face, and neck

- *Facial redness, general appearance*
- *Red eye*
- *Iritis*
- *Conjunctival pallor*
- *Temperature—high or low*
- *Mouth lesions*
- *Lumps in the:*
  - *Face*
  - *Submandibular region*
  - *Anterior neck*
  - *Anterior triangle of neck*
  - *Posterior triangle*
  - *Supraclavicular region.*

Trunk

- *Breast discharge*
- *Nipple eczema*
- *Breast lumps*
- *Gynaecomastia in male*
- *Axillary lymphadenopathy*
- *Sparse body hair*
- *Hirsutism*
- *Scar pigmentation*
- *Abdominal striae.*

Legs

- *Inguinal and generalized lymphadenopathy*
- *Sacral, leg, and heel sores.*

Cardiovascular system

Think first of cardiac output, and inspect and feel the hands for warmth or coldness. Feel the radial pulse, take the BP, and check the other pulses in the arms and neck. Next think of venous return and look at the jugular venous pressure (JVP). Then examine the heart itself (palpate, percuss, and then listen to it). Finally, examine output and venous return in the legs by feeling skin temperature, pulses, and looking for oedema of the legs, liver, and lungs.

Cardiac output

- *Peripheral cyanosis*
- *Radial pulse*
  - *Rate*
  - *Rhythm (compare cardiac apex rate, if irregular)*
  - *Amplitude*
  - *Vessel wall*
- *Compare pulses for volume and synchrony*
  Radial, brachial, carotid, (femoral, popliteal, posterior, and anterior tibials after the examining the heart)
- *BP standing and lying in right arm, repeat on left.*

Venous return

- *JVP*

The heart

- *Trachea displaced?*
- *Apex beat displaced?*
- *Parasternal heave*
- *Palpable thrill*
- *Auscultation*
  - *Extra heart sounds*
    —Systolic murmurs
    —Diastolic murmurs.

Cardiac output and venous return in the legs

- *Skin temperature*
- *Posterior and anterior tibials, popliteal, femoral*
- *Venous skin changes*
- *Vein abnormalities*
- *Calf swelling*
- *Leg oedema*
- *Sacral oedema*
- *Liver enlargement*
- *Basal lung crackles.*

Respiratory system

Think of general respiratory structure and function. *Inspect* and think of oxygen and carbon dioxide levels, then the ventilation process, which depends on the chest wall and its movement. *Palpate* by feeling for tactile vocal fremitus. *Percuss* and then *auscultate*. Finally, listen for wheezes, thus assessing airways, from small (high-pitched) to large (low-pitched).

General inspection

- *Tremor and muscle twitching*
- *Cyanosis of the tongue and lips*
- *Clubbing.*

Chest inspection

- *Respiratory rate*
- *Distorted chest wall*
- *Poor expansion*
- *Paradoxical movement.*

Palpation

- *Mediastinum*
  - *Position of trachea*
  - *Position of apex beat.*

Tactile vocal fremitus

- *Present or absent (or increased).*

Percussion

- *Hyper-resonant, resonant, normal, dull, or stony dull.*

Auscultation

- *Diminished breath sounds*
- *Bronchial breathing*
- *Crackles*
- *Rubs*
- *Wheezes, high- or low-pitched, or polyphonic during inspiration and expiration.*

Alimentary and genitourinary systems

Think first of metabolic issues related to general nutrition (obese, normal, thin, cachexia) and ensure that the patient is weighed. Check the mucous membranes, e.g. for signs of vitamin deficiency. Look for skin and eye signs of low fluid volume, and then liver disease. Next, turn your mind to anatomical aspects of the gastrointestinal and genitourinary systems together by inspecting, palpating, and auscultating. Finally, perform examinations (when indicated) that need special equipment, and do the urine tests.

Inspection

- *Obesity*
- *Cachexia*
- *Oral lesions*
- *Jaundice*
- *Hepatic skin stigmata*
- *Loss of skin turgor*
- *Low eye tension.*

Palpation

- *Supraclavicular nodes.*

Inspection of the abdomen

- *Abdominal scars*
- *Veins*
- *General distension*
- *Visible peristalsis*
- *Poor movement.*

Palpation

- *General tenderness*
- *Localized tenderness*
- *Hepatic enlargement*
- *Splenic enlargement*
- *Renal enlargement*
- *Abdominal masses.*

Percussion

- *Dull or resonant*
- *Shifting dullness.*

Auscultation

- *Silent abdomen*
- *Tinkling bowel sounds*
- *Bruits.*