

CLINICAL MEDICINE FOR THE MRCP PACES

*Volume 2: history-taking,
communication and ethics*

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OXFORD

UNIVERSITY PRESS

Great Clarendon Street, Oxford OX2 6DP

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Published in the United States
by Oxford University Press Inc., New York

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First published 2010

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British Library Cataloguing in Publication Data
Data available

Library of Congress Cataloguing in Publication Data
Data available

Typeset by Glyph International, Bangalore, India
Printed in Great Britain
by Ashford Colour Press Ltd., Gosport, Hampshire

ISBN 978-0-19-955749-3

10 9 8 7 6 5 4 3 2 1

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List of Abbreviations

↑	raised
↓	lowered
A&E	Accident and Emergency Department
AAFB	acid-alcohol fast bacilli
ABCD ₂	<u>A</u> ge, <u>B</u> lood pressure, <u>C</u> linical features, <u>D</u> uration of symptoms and Diabetes mellitus
A&E	Accident and Emergency Department
ABPA	allergic bronchopulmonary aspergillosis
ABVD	adriamycin, bleomycin, vinblastine, dacarbazine
AC	air conduction
ACC	American College of Cardiology
ACE	angiotensin converting enzyme
ACR	American College of Rheumatology
ACTH	adrenocorticotrophic hormone
ADA	Adenosine deaminase activity
ADH	antidiuretic hormone
ADPKD	autosomal dominant polycystic kidney disease
AF	atrial fibrillation
AFLP	acute fatty liver of pregnancy
AFP	alph-fetoprotein
AHA	American Heart Association
AICA	anterior inferior cerebellar artery infarction
AIDP	acute inflammatory demyelinating polyradiculoneuropathy
AIDS	acquired immunodeficiency syndrome
AIP	acute interstitial pneumonia
ALP	alkaline phosphatase
ALT	alanine aminotransferase
AML	acute myeloid leukaemia
AMPA	α-amino-hydroxy-5-methylisoxazole-4 propionic acid
AMTS	abbreviated mental test score
ANA	antinuclear autoantibody
ANCA	anti-neutrophil cytoplasmic antibody
anti-CCP	Anti-cyclic citrullinated peptide
anti-GBM	anti-glomerular basement membrane antibody
anti-RNP	anti-ribonucleoprotein antibody
AP	antero-posterior
APC	adenomatous polyposis coli
APTT	activated partial thromboplastin time
APUD	amine precursor uptake and decarboxylation
ARDS	acute respiratory distress syndrome
A-R-P	<u>A</u> ccommodation <u>R</u> eflex <u>P</u> reserved
ARPKD	Autosomal recessive polycystic kidney disease
ASA	atrial septal aneurysm
ASD	atrial septal defect
ATS	American Thoracic Society
AVM	arteriovenous malformations
AVNRT	atrioventricular nodal re-entry tachycardia

AVRT	atrioventricular re-entry tachycardia
BC	bone conduction
BCC	basal cell carcinoma
BEACOPP	bleomycin, etoposide, doxorubicin, cyclophosphamide, Oncovin (vincristine), prednisolone, procarbazine
BMD	Becker's muscular dystrophy
BMI	body mass index
BNF	British National Formulary
BNP	brain natriuretic peptide
BP	blood pressure
bpm	beats per minute
BPPV	benign paroxysmal positional vertigo
CAB	Citizens Advice Bureau
CABG	coronary artery bypass graft
CADASIL	cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy
CAM	Confusion Assessment Method
cAMP	cyclic adenosine monophosphate
c-ANCA	cytoplasmic anti-neutrophil cytoplasmic antibody
CAT	computerized axial tomography
CHD	coronary heart disease
CIDP	chronic inflammatory demyelinating polyneuropathy
CINCA	<u>C</u> hronic <u>I</u> nfantile, <u>N</u> eurologic, <u>C</u> utaneous, and <u>A</u> rticular
CJD	Creutzfeldt-Jakob disease
CK	creatine kinase
CML	chronic myeloid leukaemia
CMV	cytomegalovirus
CNS	central nervous system
COMT	catechol-O-methyl transferase
COP	cryptogenic organizing pneumonia
COPD	chronic obstructive pulmonary disease
COPP	cyclophosphamide, Oncovin (vincristine), prednisolone, procarbazine
CPA	cerebellopontine angle
CPEO	chronic progressive external ophthalmoplegia
CPR	cardiopulmonary resuscitation
CRC	colorectal carcinoma
CRH	corticotrophin releasing hormone
CRP	C-reactive protein
CSF	cerebrospinal fluid
CT	computed tomography
CT KUB	CT— kidneys, ureters, bladder
CXR	chest X-ray
D-AB	<u>D</u> orsal- <u>A</u> Bduct
DAFNE	Dose Adjustment for Normal Eating programme
DCCT	Diabetes Control and Complications Trial
DDAVP	desmopressin acetate (synthetic analogue of vasopressin)
DHS	Drug Hypersensitivity Syndrome
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DIP	desquamative interstitial pneumonia
DIP	distal interphalangeal

LIST OF ABBREVIATIONS

DKA	diabetic ketoacidosis
DMARDs	disease modifying anti-rheumatic drugs
DMD	Duchenne's muscular dystrophy
DNAR	do not attempt resuscitation
DNCB	dinitrochlorobenzene
DPA	Data Protection Act (1998)
DPCP	diphenylcyclopropanone
DPP-IV	dipeptidyl peptidase IV
DRESS	Drug Reaction with Eosinophilia and Systemic Symptoms
DVLA	Driver and Vehicle Licensing Agency
DVT	deep vein thrombosis
EAA	extrinsic allergic alveolitis
EBV	Epstein-Barr virus
ECCE	extracapsular cataract extraction
ECG	electrocardiogram
EDIC	Epidemiology of Diabetes Interventions and Complications trial
EDTA	ethylenediaminetetraacetic acid
EEG	electroencephalogram
EIA	enzyme immunoassay
EMG	electromyography
ENA	extractable nuclear antigen
ENT	ear, nose, and throat
EPS	electrophysiological studies
ERCP	endoscopic retrograde cholangiopancreatography
ERG	electroretinogram
ERS	European Respiratory Society
ESR	erythrocyte sedimentation rate
FAP	familial adenomatous polyposis
FBC	full blood count
FDA	Food and Drug Administration
FDR	first-degree relative
FET	forced expiratory time
FEV	forced expiratory volume
FH	family history
FNA	fine-needle aspiration
FOB	faecal occult blood
FRC	functional residual capacity
FSH	follicle stimulating hormone
FTA	fluorescent treponemal antibody
FVC	forced vital capacity
GALS	gait, arms, legs, spine
GBS	Guillain-Barré syndrome
GFR	glomerular filtration rate
GGT	gamma glutamyl transpeptidase
GH	growth hormone
GHRH	growth hormone-releasing hormone
GI	gastrointestinal
GIST	gastrointestinal stromal tumour
GLP-1	Glucagon-like peptide 1
GMC	General Medical Council

GN	glomerulonephritis
GORD	gastrooesophageal reflux disease
GTN	glyceryl trinitrate
HAART	highly active anti-retroviral therapy
Hb	haemoglobin
HCC	hepatocellular carcinoma
hCG	human chorionic gonadotrophin
HCV	hepatitis C virus
HDL	high density lipoprotein
HELLP	Haemolysis, Elevated Liver enzymes, Low Platelets
HHT	hereditary haemorrhagic telangiectasia
HHV-6	human herpesvirus 6
HIV	human immunodeficiency virus
HL	Hodgkin's lymphoma
HMSN	hereditary motor and sensory neuropathy
HNPCC	hereditary nonpolyposis colorectal cancer
HNPP	hereditary neuropathy with pressure palsy
HONK	hyperosmolar hyperglycaemic non-ketotic
HR	heart rate
HRA	Human Rights Act (1998)
HRCT	high resolution CT (scan)
HSV	herpes simplex virus
HTLV-1	human T-lymphotropic virus-1
IBD	inflammatory bowel disease
IBS	irritable bowel syndrome
ICAS	Independent Complaints Advocacy Service
ICD	implantable cardioverter defibrillator
ICCE	intracapsular cataract extraction
ICU	Intensive Care Unit
IDA	iron-deficiency anaemia
IGF-1	insulin-like growth factor-I
IH	immunohistochemistry
IM	intramuscular
IMCA	independent mental capacity advocates
INO	internuclear ophthalmoplegia
INR	international normalized ratio
IPV	inactivated polio virus
IV	intravenous
IVC	inferior vena cava
IVIG	intravenous immunoglobulin
IVTA	Intravitreal triamcinolone acetonide
JVP	jugular venous pressure
LA	left atrium
LABA	long acting β_2 agonist
LACI	lacunar circulation infarct
LADA	latent autoimmune disease of the adult
LDH	lactate dehydrogenase
LDL	low density lipoprotein
LEMS	Lambert-Eaton myasthenic syndrome
LFT	liver function tests

LIST OF ABBREVIATIONS

LH	luteinising hormone
LIP	lymphoid interstitial pneumonia
LMN	lower motor neurone
LP	lumbar puncture
LPHs	lipotropins
LV	left ventricle
MALT	mucosal-associated lymphoid tissue
MCA	middle cerebral artery
MCP	metacarpophalangeal
MDR TB	multi-drug resistant tuberculosis
MEN	multiple endocrine neoplasia
MGUS	Monoclonal gammopathy of undetermined significance
MI	myocardial infarction
MMSE	Mini Mental State Examination
MND	motor neurone disease
MPTP	1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine
MPZ	myelin protein zero gene
MRA	magnetic resonance angiogram
MRCP	magnetic resonance cholangiopancreatography
MRI	magnetic resonance imaging
MRSA	Methicillin resistant staphylococcus aureus
MRV	magnetic resonance venography
MSA	multiple systems atrophy
MSH	melanocyte-stimulating hormone
MSI	microsatellite instability
MSM	men who sleep with men
MuSK	muscle-specific kinase
NAAT	nucleic acid amplification test
nAChR	nicotinic acetylcholine receptors
NAFLD	non-alcoholic fatty liver disease
NCS	nerve conduction study
NHL	non-Hodgkin's lymphoma
NICE	National Institute for Health and Clinical Excellence
NMDA	N-methyl-D-aspartic acid
NO	nitric oxide
NSAID	nonsteroidal anti-inflammatory drug
NSCLC	non-small cell carcinoma
NSIP	non-specific interstitial pneumonia
NSTEMI	non-ST segment elevation myocardial infarction
NYHA	New York Heart Association
OA	osteoarthritis
OIs	opportunistic infections
OGD	oesophago-gastro-duodenoscopy
OPV	oral live attenuated polio virus
PA	pulmonary artery
PACI	partial anterior circulation infarct
P-AD	Palmar-ADduct
p-ANCA	perinuclear anti-neutrophil cytoplasmic antibody
PALS	Patient Advocacy and Liaison
PAN	polyarteritis nodosa

PBC	primary biliary cirrhosis
PCI	percutaneous coronary intervention
PCP	pneumocystis carinii pneumonia
PCR	polymerase chain reaction
PCWP	pulmonary capillary wedge pressure
PDA	patent ductus arteriosus
PE	pulmonary embolism
PEFR	peak expiratory flow rate
PEG	percutaneous gastrostomy
PFO	patent foramen ovale
PFT	pulmonary function test
PI	protease inhibitor
PICA	posterior inferior cerebellar artery
PIP	proximal interphalangeal
PJS	Peutz-Jeghers syndrome
PMF	pulmonary massive fibrosis
PMP22	peripheral myelin protein-22 gene
POCI	posterior circulation infarct
POEMS	<u>P</u> olyneuropathy, <u>O</u> rganomegaly, <u>E</u> ndocrinopathy, <u>M</u> onoclonal gammopathy, and <u>S</u> kin changes
POMC	proopiomelanocortin
PPD	purified protein derivative
PR	per rectum
PSA	Prostate Specific Antigen
PSC	primary sclerosing cholangitis
PSP	progressive supranuclear palsy
PTH	parathyroid hormone
PTU	propylthiouracil
PVS	persistent vegetative state
QALYS	Quality Adjusted Life Years
RA	rheumatoid arthritis
RA	right atrium
RAPD	relative afferent pupillary defects
RBC	red blood cell
RB-ILD	respiratory bronchiolitis-interstitial lung disease
REM	rapid eye movement
RhF	Rheumatoid factor
RNA	ribonucleic acid
RNP	ribonuclear protein
RPGN	Rapidly Progressive Glomerulonephritis
RPR	Rapid Plasmin Reagin
RV	right ventricle
SADBE	squaric acid dinitrile ester
SAH	subarachnoid haemorrhage
SCC	squamous cell carcinoma
SCLC	small cell lung carcinoma
SHBG	sex hormone-binding globulin
SHO	Senior House Officer
SIADH	syndrome of inappropriate anti-diuretic hormone
SLE	systemic lupus erythematosus

LIST OF ABBREVIATIONS

SPECT	Single Photon Emission Computed Tomography
SSRI	selective serotonin reuptake inhibitor
STEMI	ST segment elevation myocardial infarction
STI	sexually transmitted infection
SUDEP	Sudden Unexpected Death in Epilepsy
SVC	superior vena cava
SVTs	supraventricular tachycardias
TACI	total anterior circulation infarct
TB	tuberculosis
TED	thrombo-embolus deterrent
TGF	transforming growth factor
TIA	transient ischaemic attack
TIPS	transjugular intrahepatic portosystemic shunt
TLC	total lung capacity
TNF	tumour necrosis factor
TNM	classification system: spread of primary tumour (T); extent of lymph node involvement (N); and presence or absence of metastases (M)
TPHA	<i>T pallidum</i> haemagglutination
TTP	thrombotic thrombocytopenic purpura
TSH	thyroid stimulating hormone
TSI	thyroid stimulating immunoglobulin
TVO	transient visual obscuration
U&E	urea and electrolytes
UC	ulcerative colitis
UIP	usual interstitial pneumonia
UKPDS	UK Prospective Diabetes Study
UMN	upper motor neurone
USA	unstable angina
VDRL	Venereal Disease Reference Laboratory
VEGF	vascular endothelial growth factor
VF	ventricular fibrillation
VHFs	viral haemorrhagic fevers
V-Q	ventilation-perfusion
VSD	ventricular septal defect
VT	ventricular tachycardia
VZV	varicella zoster virus
WBC	white blood cell
WCC	white cell count
WHO	World Health Organization
WPW	Wolff-Parkinson-White syndrome

Station 2 ♦ History Taking Skills

Core Concepts and Overview

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Core concepts and overview

Recent years have seen a shift in the perception of communication skills from being considered subjective personal traits, to an objective, evidence-based curriculum of skills and techniques. This development was founded on the recognition that core communication skills are identifiable, can be taught, and form the basis of the majority of professional encounters.

The core skills required for the patient interview in the MRCP (PACES) examination can be broadly divided into three areas: *content* skills, *process* skills, and *perceptual* skills. The *content* of the medical interview refers to the information the candidate is attempting to gather, or to give, during the course of the interview. The detail of this content is covered in the subsequent case histories of this book. However, the ability of the candidate to communicate or acquire this content depends largely on the *process* of the interview, and their *perceptual* skills.

The *process* of the interview refers to the manner in which the candidate communicates with the patient. Although the candidate's agenda may be to establish the content of the interview, this will not be successful unless the patient's agenda is considered. In Stations 2 and 4 of the PACES examination, the candidate is provided with clear written instructions (such as a GP letter), followed by 14 minutes for patient interaction. The candidate then has 1 minute for reflection, followed by 5 minutes of discussion with the examiners. Despite the limited time available in the PACES Stations 2 and 4, the examiners will expect the candidate to develop rapport, show empathy, appropriately use silence during the interview, and interpret non-verbal cues. Indeed, these processes are the key to obtaining the content of the interview, not just for the examination but also for clinical practice.

Perceptual skills refer to the candidate's decision making, problem solving, and clinical reasoning skills. These are complex, higher-order skills, which reflect the individual's attitudes, beliefs, and self-perception. Indeed, if the content of the interview reflects the candidate's 'knowledge', and the process of the interview is a reflection of 'technique', then these perceptual skills are a demonstration of appropriate 'behaviour' or 'performance'.

Nevertheless, the distinction between these skills is artificial since they are inextricably linked, and the MRCP candidate must employ all three areas to be considered a professional—the summation of knowledge, technique, and behaviour. The purpose of this section of the book is to provide the candidate with a framework to develop the necessary techniques for successful medical interviewing.

The most commonly used framework for acquiring a medical history is the traditional model, which reflects the 'content' of the interview (Box 2.1). Whilst this model is the established order of case presentation, and the 'currency' of communicating salient facts to colleagues, this model has also become embedded in the *process* of the interview. This linear model is based around the clinician's agenda of acquiring information, and promotes a closed questioning style to fulfil this agenda. Moreover, in the examination setting, candidates use the history-taking station to demonstrate their knowledge by saying it out loud in the form of the questions they ask. Inevitably, this involves closed questions, which neglect the patient's agenda and the doctor–patient relationship.

Box 2.1 Traditional history-taking model
• Presenting complaint • History of presenting complaint • Past medical history • Drug history • Allergy history • Family history • Social history • Systems review

Instead, the art of patient-centred communication lies in using the process of the interview to establish the patient’s perspective of the consultation, and *translating* that information into the traditional history structure when presenting to colleagues or documenting written notes. Time constraints, whether in the PACES examination or in clinical practice, often compel candidates to adopt an interrogative interview technique in an attempt to be more time efficient. However, this approach will isolate the patient and dismay examiners, even if it satisfies the candidate’s agenda. The Calgary-Cambridge guide by Kurtz and colleagues¹ is a useful framework to the process of effective medical interviewing, and is one of the most widely used tools in patient communication. Although there is only a window of 14 minutes to acquire the history in the PACES examination, application of the abridged framework presented below will allow the candidate to target the consultation to the patient’s agenda, and thus establish the content of the consultaion in the most efficient manner.

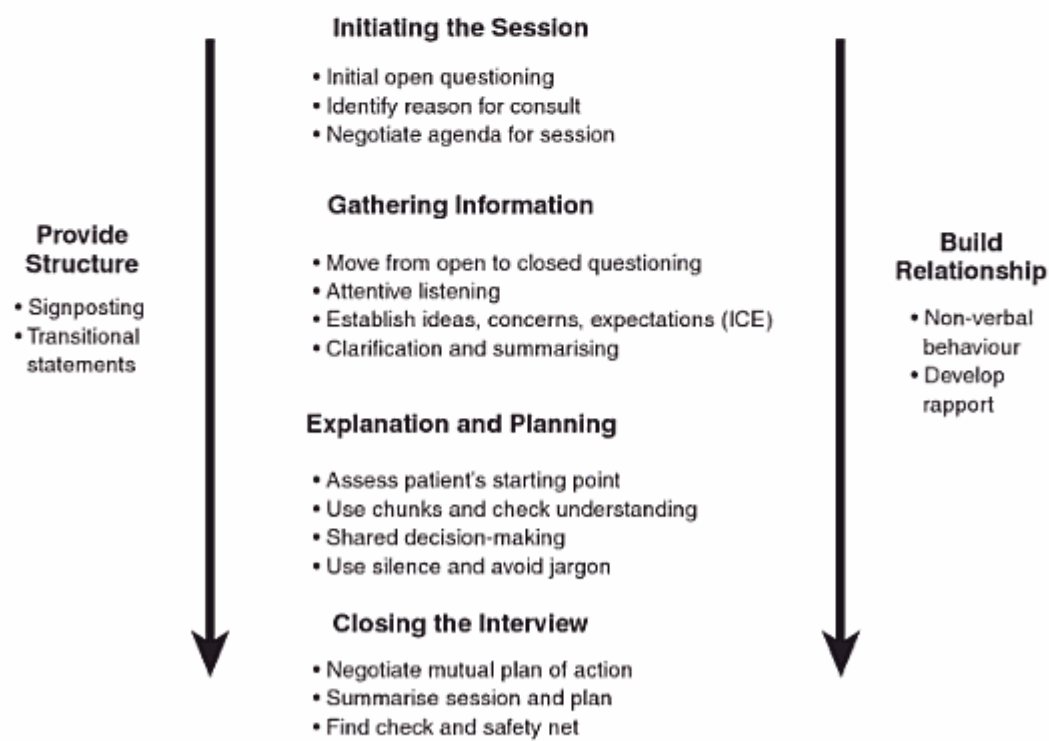


Figure 1 Calgary-Cambridge framework.
Modified from Kurtz and Silverman¹

Initiating the session

Begin by greeting the patient and obtaining their name. Introduce yourself, your role, and the nature of the interview. If appropriate, obtain verbal consent for the interview: *'Is it alright if we discuss the reason why you are here today?'* Identify the reason for the consult by asking appropriate open questions, e.g. *'What would you like to discuss today?'*, or *'What questions were you hoping to ask me today?'*

Use *attentive listening* to hear the patient's initial response without interruption or direction. This may feel like a long time in the examination scenario, but allowing the patient to make an uninterrupted opening statement minimizes the risk that 'hidden agendas' will arise later in the interview. Once the patient has finished speaking, use *summarizing* and *checking* to maintain a structure to the interview, and screen for other problems: *'So that's abdominal pain and vomiting, anything else?'*

If the list of problems is unrealistic for a single consultation, it may be necessary to negotiate the agenda with the patient: *'Since the pain and the vomiting are new, shall we concentrate on those today and talk about your eye drops at the next appointment?'* However, even though the scenario in the PACES examination will give a background to the patient's problem, they may still have a hidden agenda and it is important not to dismiss the patient's issues just because they are not listed in the introduction to the scenario.

Gathering information

Encourage the patient to tell the story in their own words, from beginning to end. Use open questioning initially; *'Tell me how your symptoms started?'* Move on to use closed questions for details and to establish dates and times: *'What prompted you to finally go to the doctor?'*, *'How long was it before you came to hospital?'*

Use *clarification* if the patient uses statements that are unclear: *'Could you explain what you mean by sick? Do you mean vomiting or unwell?'* Avoid the use of jargon, e.g. gastrostomy, echocardiogram.

Actively try to determine:

- I the patient's *ideas* about their illness; *'What do you think may be causing your symptoms?'*
- C their *concerns*, *'Is there anything you are worried this may be?'*
- E their *expectations*: *'What do you see happening to yourself after treatment?'*, *'I see you are keen to start treatment "quickly"— is there anything specific that you are trying to get better for?'*

During the process of acquiring the content of the interview, use *signposting* to direct the interview and maintain structure, *'I'm now going to ask you some questions about your bowel habits'*. Sharing thinking with the patient, if done sensitively and cautiously, also encourages patient involvement and develops rapport, *'What I'm thinking now is this may be a problem with your digestion. Have you had these symptoms before?'*

Explanation and planning

Begin by assessing the patient's starting point, by asking how much they understand about their problems or the process so far. Use small chunks of information when explaining a plan, and check for understanding at each stage. Use the patient's responses and non-verbal cues as a guide to how to proceed. Use visual aids or diagrams if possible.

The goal is to achieve a shared understanding of the medical problem. This involves relating the issues to the patient's perspective, and to their ideas, concerns, and expectations that were previously discussed. This phase of the interview is built upon the relationship that has been established by the candidate's techniques, behaviour, and professionalism. Sharing the interviewer's thoughts and dilemmas during the course of the interview is an important way of

empowering the patient to engage in shared decision making. *'I can see that you are concerned about the thought of having a biopsy. Let me explain the issues from my perspective so we can make this decision together.'* *'Whilst there is a lot of research in this area, we still don't know the best treatment. Let me explain why I think we should avoid antibiotics.'*

Finally, summarize the session and check with the patient that their concerns have been addressed. A safety net must also be mentioned—what to do if the plan is not working, e.g. call the secretary, attend the emergency department. Finish by asking for final questions and check agreement with the plan of action.

Whilst this framework provides a structure for the candidate to fall back on in the PACES examination, it must be remembered that allowing the patient to determine the content of the interview facilitates patient-centred interviewing. The art of the skilled interviewer is to use the techniques mentioned above to provide structure to the interview, without encroaching on the patient's agenda. If the candidate can apply these techniques on the background of the evidence-base presented in the forthcoming case histories, they will have the foundation for effective communication in the PACES examination and their professional practice.

References

1. Kurtz SM, Silverman JD. The Calgary-Cambridge Referenced Observation Guides: an aid to defining the curriculum and organizing the teaching in communication training programmes. *Medical Education* 1986; 30:83–9.

History-Taking Skills

Case 1 ♦ Hypertension

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 52-year old gentleman with an elevated blood pressure. He has been found to have three successive blood pressure readings of 158/98 mmHg, 160/95 mmHg, and 156/96 mmHg. He has been taking bendroflumethiazide 2.5mg daily for the last 4 months, with little effect. There is no history of stroke or ischaemic heart disease. He is a smoker of 20 cigarettes per day, and drinks up to 12 pints of lager at weekends. He is currently a self-employed electrician.

Many thanks for your opinion.

Acquiring the history

A. History of presenting complaint:

- Are there symptoms of uncontrolled hypertension—headache, visual disturbance?
- Are there symptoms of end-organ damage—chest pain, palpitations, dyspnoea, ankle oedema, neurological symptoms?
 - ♦ **MACROVASCULAR DISEASE**
 - Coronary artery disease:** chest pain, breathlessness, orthopnoea, paroxysmal nocturnal dyspnoea, ankle oedema
 - Peripheral vascular disease:** cold extremities, claudication
 - Cerebrovascular disease:** visual disturbances, neurological deficit
 - ♦ **MICROVASCULAR DISEASE**
 - Retinal disease:** visual disturbance
 - Renal disease:** nausea, lethargy, ankle swelling
- Are there symptoms of an underlying medical disorder causing secondary hypertension (see question 1).
 - ♦ Enquire about symptoms of endocrine disease:
 - Hyperthyroidism:** anxiety, sweating, palpitations, tremor, weight loss, diarrhoea
 - Acromegaly:** headaches, change in appearance, glove and shoe size, visual field disturbance
 - Cushing's syndrome:** steroid use, striae, purpura, centripetal fat deposition, proximal myopathy
 - Conn's syndrome:** weakness, lethargy, muscle cramps (hypokalaemia)
 - Phaeochromocytoma:** palpitations, sweating and weight loss.
 - ♦ Enquire about symptoms of renal disease. Are there any urinary symptoms, obstructive symptoms, or symptoms of uraemia, i.e. lethargy, nausea, pruritis?
- Are there symptoms of an underlying anxiety disorder, or of 'white-coat hypertension'? Has the patient had their blood pressure measured outside a hospital or clinic?

B. Relevant medical and family history:

- Ask about conditions which predispose to secondary hypertension (see question 1)
- The major complications of hypertension are coronary heart disease (CHD), stroke, renal disease, heart failure and peripheral vascular disease. Ask specifically about these conditions.
- A family history of hypertension makes essential hypertension more likely. However, a family history of endocrine disease (*specifically MEN 2*), CHD and risk factors such as dyslipidaemia should be sought.

C. Medications and interactions:

- Remember, most hypertensive patients will use over the counter or prescribed medications which alter blood pressure, or interact with common antihypertensive medications:
 - Nonsteroidal anti-inflammatory drugs (NSAIDs) can cause sodium retention, and resistance to hypertension treatment.
 - Over the counter nasal sprays and decongestants may contain vasoactive agents such as ephedrine and pseudoephedrine, which can induce hypertension.
- Oral contraceptive agents frequently cause a mild elevation in blood pressure, although oestrogen containing compounds may induce overt hypertension.
- Corticosteroids (topical and systemic) cause sodium retention and hypertension. Ask specifically about symptoms of Cushing's syndrome.
- Ciclosporin may also cause progressive hypertension.
- Illicit drug use, such as cocaine, MDMA (ecstasy) and amphetamines, must not be overlooked in patients with resistant hypertension.
- Ask about anabolic steroid use in young patients, especially athletic male patients.
- Ask about compliance with prescribed medications. If compliance is poor, ask about common adverse reactions to therapy (e.g. bendroflumethiazide—impotence, gout; calcium-channel blockers—ankle swelling, constipation; angiotensin converting enzyme (ACE) inhibitors—cough).

D. Social issues:

- Alcohol excess and obesity are the most common causes of reversible hypertension. Ask about lifestyle, diet (caffeine and salt intake) and alcohol use.
- Cigarette smoking is also a major contributor to CHD risk.
- Enquire about illicit drug use (see above).
- Ask about occupation, and how the patient is supporting himself financially.
- If any risk factors for impotence are present, such as diabetes mellitus, peripheral vascular disease, or bendroflumethiazide or beta blocker use, ask sensitively about marital problems.

Formulating a plan of action

- Explain that the diagnosis of hypertension is not in itself serious, but control of blood pressure is necessary to prevent serious complications like heart disease and stroke, and that causes are primary (essential hypertension) and secondary. If 'white coat hypertension' is likely, then an ambulatory 24-hour BP monitor may be necessary.
- Tell the patient that you would like to request routine blood tests (to exclude possible secondary causes of hypertension) and an ECG (electrocardiogram) (to look for evidence of left ventricular hypertrophy).
- Advise about lifestyle factors and modifications.
- Tell the patient that a follow-up appointment will be given to discuss the results of above tests.

Questions commonly asked by examiners

What are the causes of secondary hypertension, and how common is it?

Secondary hypertension accounts for 5% of the prevalence of hypertension in primary care.

The causes are:

- **Renal**
 - ♦ Renal parenchymal disease
 - ♦ Renovascular disease
 - ♦ Chronic renal disease of any aetiology
- **Endocrine**
 - ♦ Cushing's syndrome
 - ♦ Hyperaldosteronism
 - ♦ Adrenal hyperplasia
 - ♦ Pheochromocytoma
 - ♦ Acromegaly
 - ♦ Thyroid disease
- **Cardiorespiratory**
 - ♦ Coarctation of the aorta
 - ♦ Obstructive sleep apnoea
- **Drug-induced**

Which patients with hypertension should receive treatment?

The National Institute for Health and Clinical Excellence (NICE) guidelines¹ (2006) recommend that all hypertensive patients with an estimated 10-year CHD risk of greater than 20%, or a persistent blood pressure over 160/100mmHg, should receive pharmacological treatment. All patients with diabetes mellitus or pre-existing cardiovascular disease should also be treated, as guided by the National Service Frameworks for these diseases.

How is cardiovascular risk assessed in a hypertensive patient?

The 10-year CHD risk is estimated using Coronary Risk Prediction Charts,² derived from data acquired from the Framingham Heart Study. These charts provide an estimate of CHD risk based on gender, age, systolic blood pressure, smoking status, total cholesterol, and HDL cholesterol. However, these charts are only suitable for primary prevention. These charts cannot be used for patients with established cardiovascular disease, familial hypercholesterolaemia or other inherited dyslipidaemias, chronic renal disease or diabetes mellitus.

How would you manage this patient?

- **Confirm diagnosis** – hypertension should be diagnosed with an appropriate sized blood pressure cuff, with an elevated blood pressure found on three separate occasions.
- **Lifestyle interventions** – advice should be provided regarding reducing salt intake, decreasing alcohol consumption, smoking cessation, and regular exercise.
- **Manage cardiovascular risk** – modifiable risk factors such as obesity, hyperlipidaemia, diabetes mellitus and smoking must be identified and treated. All patients with a 10-year CHD risk greater than 30% should receive lipid lowering therapy.
- **Drug treatment** – patients aged over 55 should be treated according to the NICE guidelines (Figure 1.1).

What blood pressure would you be aiming for with treatment?

For patients without diabetes mellitus, the aim of treatment should be a systolic pressure <140mmHg, and a diastolic pressure <85mmHg.

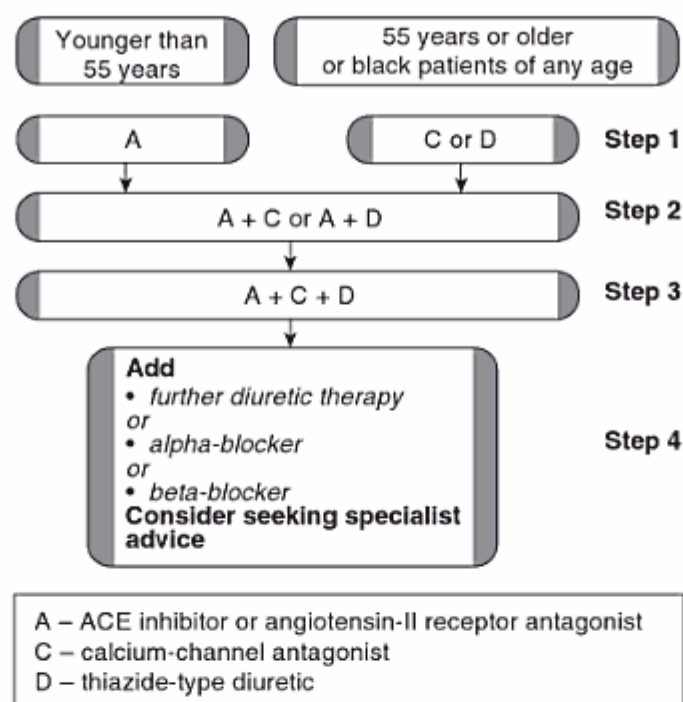


Figure 1.1 BHS/NICE flow chart.

In patients with diabetes mellitus, chronic renal disease, or established cardiovascular disease, the optimal treatment goals are systolic pressure <130mmHg, and diastolic pressure <80mmHg.

What is the role of beta blockers in the management of hypertension?

Beta blockers are no longer considered first-line antihypertensives for patients over the age of 55 in the most recent NICE guidance.¹ This is following a series of studies suggesting decreased efficacy in reducing stroke risk, including the ASCOT³ and LIFE⁴ trials. A meta-analysis of these trials demonstrated a 16% higher incidence of stroke among patients treated with beta blockers, primarily atenolol, than those treated with other antihypertensive medications.⁵

Beta blockers may still be useful in younger patients, or those with associated CHD.

What about elderly patients?

Until recently, there was no data to support the treatment of hypertension in patients over the age of 80. However, the recent HYVET study was stopped early due to a lower rate of fatal or non-fatal stroke in elderly patients receiving indapamide, with or without perindopril, over placebo.⁶

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Case 2 ♦ Dyspepsia

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 42-year old lady with a three month history of bloating and epigastric pain. She feels she may also have lost some weight, however her bowel habit is regular. She has been taking lansoprazole 15mg daily with some benefit, but is still symptomatic. She smokes 30 cigarettes per day. There is no other significant past medical history.

Many thanks for your opinion.

Acquiring the history

A. History of presenting complaint:

- Characterize the abdominal pain—site, type, radiation, intensity, duration, onset, frequency, previous episodes?
- Is there associated nausea or vomiting?
- Are there associated reflux symptoms—retrosternal burning, acid brash, regurgitation, worse on lying flat?
- Is the pain relieved by, or precipitated by, meals?
- Is the pain pancreatic in origin—radiates to back, precipitated by eating, relieved by leaning forward?
- Is the pain biliary in origin—right sided abdominal pain, precipitated by eating, with associated nausea?
- Is the pain colonic in origin—lower abdominal pain, colicky in nature, partially relieved by defaecation?
- Could the pain be cardiac in origin—exertional chest pain, dyspnoea?
- Are there any alarm features, suggesting gastrointestinal malignancy?
 - ♦ Dysphagia?
 - ♦ Weight loss?
 - ♦ Early satiety?
 - ♦ Jaundice?
 - ♦ Anaemia?
 - ♦ Progressive vomiting?
 - ♦ Previous gastric ulcer?
 - ♦ Previous gastric surgery?
- Nocturnal symptoms? Posture during sleep (number of pillows, etc.)?
- Is the patient eating late in the day, precipitating nocturnal symptoms?
- Are the symptoms affecting the patient's quality of life?

B. Relevant medical and family history:

- Previous gastric ulcer or gastric surgery?
- Previous pancreatitis?
- Previous gall stone disease or cholecystectomy?
- Haemoglobinopathy (pigment gall stones)?
- Iron deficiency anaemia (except premenopausal women)?
- Any previous endoscopic or radiological investigations?
- Family history of gastrointestinal malignancy?
- Hyperlipidaemia (markedly elevated triglycerides are a risk factor for pancreatitis)?

C. Medications and interactions:

- Ask about proton pump inhibitor use, or other antacid compounds?
- Ask about compliance with current drug regimen?
- Any drugs which may cause gastric ulceration—NSAIDs, corticosteroids, bisphosphonates?
- Any drugs which may precipitate gastro-oesophageal reflux—nitrates, calcium antagonists, theophyllines, bisphosphonates?
- Any drugs which may cause pancreatitis—azathioprine, antiretroviral drugs, loop and thiazide diuretics?

D. Social issues:

- Alcohol excess and obesity are common precipitants of abdominal pain. Ask about lifestyle, diet, and alcohol use.
- Cigarette smoking is also a major contributor to dyspepsia, and gastrointestinal cancer risk.
- Ask about occupation, and how the patient is supporting himself financially.

Formulating a plan of action

- Explain that there are several possible diagnoses of abdominal pain and dyspepsia, but a trial of therapy may be the best initial strategy prior to investigation.
- Explain that endoscopy would be an initial test if alarm symptoms are present, or if the patient is aged over 55.¹
- If endoscopy is not indicated initially, tell the patient that the initial management would be a trial of proton pump inhibitor for four weeks, followed by *Helicobacter pylori* testing if the patient is still symptomatic.
- Suggest an abdominal ultrasound if biliary or pancreatic pain is a feature.
- If reflux symptoms are present, advise the patient to elevate the head of their bed, and avoid eating late in the day, to prevent nocturnal symptoms.
- Provide general advice about weight loss, smoking cessation, limiting alcohol intake, and avoiding dietary precipitants of dyspepsia—although avoid the concept of ‘food allergy’.
- Tell the patient that a follow-up appointment will be given to discuss the results of above tests.

Questions commonly asked by examiners

What are the major risk factors for gastric cancer?

- *Helicobacter pylori* infection—causes atrophic gastritis, progressing to metaplasia, dysplasia and cancer
- Previous gastric surgery—due to hypochlorhydria, or bile reflux
- Cigarette smoking—associated with a 1.5 fold increase in risk
- Blood group A—may be in linkage disequilibrium with other genes close to blood group antigens
- Gastric cancer has been described in association with certain cancer syndromes, including hereditary non-polyposis colorectal cancer, familial adenomatous polyposis, and Peutz-Jehger’s syndrome.

Tell me about methods of diagnosing *Helicobacter pylori* infection?

Non-invasive methods of diagnosis include ^{13}C -urea breath testing, and *Helicobacter* stool antigen detection. The sensitivity and specificity of both these tests exceeds 95%, although the use of proton pump inhibitors may lead to false negative results.

Rapid urease testing from endoscopic biopsies has a sensitivity and specificity of 90–95%. *Helicobacter* can also be diagnosed on routine histology from the gastric antrum and body. It can also provide information about intestinal metaplasia and mucosal-associated lymphoid tissue (MALT), both of which are associated with *Helicobacter* infection.

Bacterial culture and sensitivity testing is not routinely recommended, but may provide information in refractory disease.

What is role of *Helicobacter pylori* infection in peptic ulcer disease?

Epidemiologically, *Helicobacter* is associated with up to 95% of patients with duodenal ulcers. Furthermore, treatment of *Helicobacter* reduces the incidence of ulcer recurrence.

Helicobacter is also found in 65–95% of patients with gastric ulcers, and 70–90% of patients with gastric cancer. However, *Helicobacter* eradication has not been proven to reduce gastric adenocarcinoma risk, although MALT-type lymphomas may achieve remission following *Helicobacter* eradication.

The mechanism of disease is thought to be due to bacterial effects on gastric acid secretion, gastric metaplasia, immune responses to infection, and mucosal defence mechanisms.

What is the role of *Helicobacter pylori* infection in non-ulcerative dyspepsia?

Helicobacter infection has also been reported in 20–60% of patients with non-ulcerative dyspepsia, although the prevalence in asymptomatic individuals is 20–45%. The effect of treating *Helicobacter* in these patients has been studied several times, although the studies have provided mixed results, with only a small benefit on symptoms demonstrated on meta-analysis.

References

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Case 3 ♦ Weight Loss

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 82-year old gentleman who has lost 3 stone in the last 2 months. His appetite is poor, although there are no other symptoms of note. He is an ex-smoker and has also has chronic obstructive pulmonary disease (COPD) and hypertension which are managed with inhalers and antihypertensives.

Many thanks for your opinion.

Acquiring the history

Weight loss is a common but worrying symptom for patients, particularly the elderly. The aim of the history is to detect features of serious physical and psychological illness, and to establish the need for further invasive investigation.

The focus of much of the history is to screen for underlying causes of weight loss, hence many of the questions are 'closed'. Therefore, initially establish that weight loss is the patients' only complaint, and ask openly if the patient has any other concerns or is worried about anything in particular; 'Before we discuss your weight loss, may I ask if there is anything else bothering you? Is there anything in particular you are worried may be causing the weight loss?'.

A. History of presenting complaint:

- **Determine nature of weight loss:**
 - Quantify degree of weight loss: Clinically significant weight loss may be defined as greater than 5% of body weight over a period of up to 12 months, and is associated with increased mortality. However, patients may under-estimate or over-estimate the degree of weight loss. Up to one-third of patients complaining of weight loss have not actually lost weight. Attempt to quantify the amount and duration of weight loss. 'How much weight have you lost?', 'Over how long?'. If possible, calculate the Body Mass Index—weight(kg)/height(m)². Additionally, corroborate the weight loss by asking family members for an opinion and asking about changes in clothing size, 'Have you noticed your clothes becoming looser or hanging off you?'.
 - Is the weight loss voluntary or involuntary?
 - Does the weight loss coincide with a change in diet, physical activity or lifestyle?
 - Establish if the patient has lost their appetite, 'Do you still feel hungry?', 'Are you enjoying your food?', 'How is your appetite?'.

Involuntary weight loss with preserved appetite

Uncontrolled diabetes mellitus
Hyperthyroidism
Malabsorption
Pheochromocytoma

Involuntary weight loss with loss of appetite

Malignancy
Severe cardiac or respiratory failure
Advanced chronic renal disease
Oesophageal disease (causing dysphagia or odynophagia)
Gastroduodenal disease (causing vomiting or abdominal pain)
Depression
HIV
Chronic inflammatory disease of any cause

- **Screen for alarm symptoms:**
 - Dysphagia: 'Do you have problems swallowing, or does the food ever get stuck?' See Case 6, Dysphagia for subsidiary questions.
 - Early satiety: 'Are you able to manage a meal, or have you been getting full quickly?'
 - Abdominal pain: 'Do you ever experience any pain in your stomach or abdomen?'. Ask about post-prandial pain (suggesting gastroduodenal disease or mesenteric ischaemia) and pancreatic pain in particular. 'Does the pain come on after meals? Does it go through to your back?' See Case 8, Abdominal pain.
 - Change in bowel habit: 'Have you noticed a change in your bowels? Any diarrhoea or constipation? What colour are the stools?' This is often a difficult question for patients to answer accurately, so a closed question may help 'Are they pale, like this?' whilst pointing to a pale yellow object (often the colour of casenotes folders!).

- Melaena/haematochezia: 'Have you passed any blood in your motions? Is the blood red or dark? Is it bright red—like a letterbox?'
- Respiratory symptoms: 'Have you noticed a change in your breathing or cough? Have you coughed up any blood?'
- Bone pain: 'Have you had any pain in your back or your joints?'. Bone pain or night pain may suggest metastatic malignancy.
- Endocrine symptoms: hyperthyroidism—'Have you had sweats recently, or been unusually warm or nervous?', diabetes—'Have you been unusually thirsty or passing a lot of urine?', adrenal insufficiency—'Have you had any nausea or dizziness recently?'
- Other constitutional symptoms: night sweats, fatigue.

• **Establish the patient's nutritional intake:**

An assessment of nutritional intake should be made, along with an assessment of the patient's nutritional requirements which are influenced by co-morbid conditions and functional status.

Ask about:

- the number of meals and snacks consumed during a typical 24-hour period
- the consumption of meat and dairy produce
- who prepares the food, and whether the patient finishes the portions
- the use of nutritional and vitamin supplements
- previous dietetic consults

Also ask about any other problems with eating meals, such as problems with dentition or painful swallowing (odynophagia).

Remember that the physiological energy requirements will be higher for patients who have chronic illnesses and those that are particularly mobile.

• **Screen for psychological causes of weight loss:**

Psychiatric disease is a common cause of weight loss, particularly in young females or in the elderly.

- 'Have you ever tried to lose weight deliberately?'
- 'Does your weight fluctuate a lot?'
- 'How many diets have you been on in the past year?'
- 'How has your mood been over recent weeks?'

B. Relevant medical and family history:

- Several chronic medical conditions can cause weight loss: COPD, cardiac failure, diabetes, chronic renal disease.
- Ask about gastrointestinal (GI) conditions such as peptic ulcer disease, pancreatic disease, or Crohn's disease.
- Ask about previous GI surgery which may predispose to malabsorption, such as small bowel resection or pancreatic surgery.
- Patients with peripheral vascular disease, and/or vascular risk factors are prone to mesenteric ischaemia.
- Ask about a family history of malignancy, particularly colorectal, breast, and ovarian cancer.

C. Medications and interactions:

- Some medications may directly contribute to weight loss by affecting appetite: selective serotonin reuptake inhibitors (SSRIs), levodopa, metformin, theophylline, and digoxin.
- GI side effects may also contribute to weight loss:
 - Dry mouth: anticholinergics, diuretics
 - Dysphagia: bisphosphonates, NSAIDs, theophylline
 - Nausea: antibiotics, digoxin, metformin, iron

HISTORY-TAKING SKILLS

- Pancreatitis may be caused by azathioprine, HIV anti-retrovirals, loop diuretics, thiazide diuretics and metronidazole. Ensure that patients with chronic pancreatitis on pancreatic enzyme replacements are also on acid-suppression, since gastric acid may denature the enzyme supplements.
- Illicit drug use, such as cocaine and amphetamines, must not be overlooked in young patients with weight loss.

D. Social issues:

- Alcohol excess is a common cause of chronic pancreatitis.
- Cigarette smoking is a risk factor for several malignancies.
- Ask about HIV risk factors, since HIV is a cause of unexplained weight loss.
- Document travel history as a risk factor for chronic infection (eg. Giardia, Campylobacter, Cryptosporidium)

Formulating a plan of action

- Reassure the patient that whilst weight loss is a serious symptom, a cause is usually found (in 75% of cases).
- Tell the patient that you would like to perform a full examination and request routine blood tests. Explain that a dietetic consultation may also be helpful.
- Initial tests to consider include:
 - Full blood count (FBC), electrolytes, liver function tests (LFTs), albumin (not a sensitive marker of nutritional status, since it varies with severe co-morbidity), C-reactive protein (CRP), iron studies.
 - Thyroid function tests, glucose, 9am cortisol or synacthen test.
 - Serum B12, calcium, vitamin D, prothrombin time (markers of vitamin malabsorption).
 - Tumor markers: CEA, Ca19-9, Ca-125, α FP, PSA.
 - Faecal sudan stain (qualitative marker of fat malabsorption), faecal elastase (low in chronic pancreatitis).
 - Coeliac antibodies
- Tell the patient that a follow-up appointment will be given to discuss the results of above tests, and establish the need for further investigations (such as imaging or endoscopy).

Questions commonly asked by examiners

How would you assess a patient's nutritional status?

The Subjective Global Assessment¹ is a validated dietetic tool to assess nutritional status. It is based on six features, which are assessed by history and examination:

- (i) Change in weight
- (ii) Dietary intake
- (iii) Functional capacity
- (iv) Gastrointestinal symptoms with nutritional impact (e.g. diarrhoea)
- (v) Metabolic stress of current disease
- (vi) Examination findings of poor nutrition

The patient's nutritional requirements are affected by functional capacity and co-morbidity, but can be estimated:

$$1000 + (10 \times \text{body weight}) = \text{approximate energy requirement (kcal)}$$

An assessment must also include an approximation of the patient's fluid requirements, which will be influenced by fluid losses due to fever, malabsorption, stoma output, etc.

Clinical examination will help to determine fluid balance status, however the degree of fat and muscle stores can also be estimated:

- (i) fat stores: triceps skinfold thickness
- (ii) muscle stores: arm muscle circumference, temporalis muscle wasting
- (iii) muscle function: hand grip dynamometry

Biochemistry is also used to assist nutritional assessment, however no single test directly reflects nutritional state. In particular, the serum albumin is inversely correlated with the CRP, and may therefore reflect inflammation rather than the nutritional state. Moreover, the albumin only falls in severe malnutrition, hence patients may have significant undernutrition with a normal albumin.

Measurement of electrolytes, in particular the sodium, potassium, calcium, magnesium, and phosphate, is important when commencing feeding for early detection of re-feeding syndrome (see below).

What are the principles of management of malnutrition?

1. Eliminate sepsis—since nutritional support is ineffective in the presence of active sepsis, infection must be detected and treated. Malnourished patients may not exhibit the classical signs of sepsis, so a high degree of suspicion is required.
2. Diagnose and treat GI disease—malabsorption may be a consequence of coeliac disease, Crohn's disease, bacterial overgrowth, pancreatic insufficiency, lactose deficiency or short bowel syndrome. This must be treated alongside nutritional supplementation.
3. Treat micronutrient deficiencies—all patients should have thiamine supplementation before feeding is commenced, since re-feeding may precipitate Wernicke's encephalopathy in malnourished patients with thiamine deficiency.
4. Attempt nutritional support enterally—if the GI tract is intact and functioning then this is the preferred route of supplementation, since enteral feeding prevents gram-negative bacterial translocation across the gut lumen, and is not associated with the complications of parenteral line-feeding.
5. Monitor and replace electrolytes to prevent re-feeding syndrome—in malnourished patients who are undergoing gluconeogenesis, the sudden introduction of nutrition causes a surge in insulin to facilitate glucose uptake. However, this insulin also causes an intracellular shift in potassium, phosphate, and magnesium, resulting in very low serum levels. This may cause clinical consequences including arrhythmias, rhabdomyolysis, and death. Therefore, these electrolytes must be measured and replaced daily until feeding is established and there are no further electrolyte shifts.
6. Parenteral nutrition—this is administered through a tunnelled intravenous catheter (Broviac catheter). The short-term complications include those from line placement (bleeding, pneumothorax, line sepsis, and thrombosis), hyperglycaemia, re-feeding syndrome, and abnormal LFTs. If excess nutrition is provided, greater than 500kcal over energy requirements, then acute fatty liver, jaundice, and liver failure may occur. The long-term complications include line sepsis, line thrombosis, gallstones, renal stones, and bone disease.

How would you approach weight loss in elderly patients?

Weight loss in elderly patients may not be due to medical conditions, but may be consequence of dementia or social factors. Elderly patients also have impaired smell and taste, and altered GI function, which affect appetite and intake. Furthermore, polypharmacy is common in this group and medications may contribute to weight loss directly (see above) or indirectly by causing GI side effects (e.g. altered taste, nausea).

All elderly patients with weight loss should undergo screening for depression and dementia. Furthermore, elderly patients with weight loss and iron-deficiency anaemia should undergo evaluation of the upper and lower GI tract. The causes of weight loss in the elderly are listed in Box 1.

Box 1 Unintentional weight loss in the elderly: 'Meals on Wheels'

M	Medication effects
E	Emotional problems (eg. depression)
A	Anorexia, alcoholism
L	Late-life paranoia
S	Swallowing disorders
O	Oral health (e.g. poorly fitting dentures)
N	No money
W	Wandering and dementia behaviour
H	Hyper/hypothyroidism, hypercalcaemia, hypoadrenalism
E	Enteric problems (eg. malabsorption)
E	Eating problems (eg. inability to feed oneself)
L	Low salt, low cholesterol diets
S	Social problems (e.g. isolation, difficulty shopping)

Source: Adapted from Morley and Silver¹

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2. Morley JE, Silver AJ. Nutritional issues in nursing home care. Ann Intern Med 1995;123(11):850–9.

Case 4 ♦ Change in Bowel Habit

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 48-year old lady with a 3-month history of altered bowel habit. She has lost a stone in weight over this time, but has not had any rectal bleeding. She has a history of longstanding type 1 diabetes, for which she takes insulin. She is concerned because she has a cousin with ulcerative colitis and her uncle recently died from colon cancer. I wonder if she needs a colonoscopy?

Many thanks for your opinion.

Acquiring the history

Altered bowel habit is a common symptom, and the challenge for the candidate is not only to consider the broad list of differential diagnoses, but also to determine which patients require further invasive investigation. Moreover, distinguishing between the many causes of diarrhoea and constipation requires questioning about other GI and systemic symptoms. Therefore, sensitive

and open questioning is essential to detect symptoms that the patient may regard as trivial or embarrassing, but which may be highly relevant to their investigation and management.

A. History of presenting complaint:

- **Determine what the patient means by a change in bowel habit.**

'What has been the trouble with your bowels recently?'; 'Can you tell me exactly what you mean by constipation/diarrhoea/loose stool?'; 'How is this different from your normal bowel habit?'

- **Constipation:** Determine what the patient means by 'constipation'. The classic definition is of fewer than 3 bowel movements per week, however patients use the term to describe a broad range of symptoms. Determine the patient's 'normal' bowel habit, and how things have changed.

Ask about stool frequency, consistency, associated straining, and the sensation of incomplete evacuation. *'How often do you open your bowels in an average week?'; 'What do you pass?'; 'Is the stool hard or soft? Does it hurt or do you strain when you pass stool?'; 'Have you noticed any blood in the toilet or in your stool?'; 'When you finish, do you ever have the feeling that you still need to open your bowels?'*

Ask about onset, duration, and progression of symptoms. Acute or recent-onset constipation is more likely to represent colon cancer or intestinal obstruction. *'Are you still passing wind?'*—failure to pass flatus suggests complete intestinal obstruction.

Defecatory disorders are the cause in a significant proportion of patients with constipation (20–25%). These are caused by pelvic floor dysfunction, where the puborectalis muscle and external anal sphincter fail to relax during defecation, possibly due to incorrect learned behaviour earlier in life. In fact, this syndrome is associated with psychological symptoms such as depression and anxiety, and a large proportion of patients (40%) have a history of sexual abuse. Sensitive and open questioning is therefore essential, particularly once initial questioning is complete and rapport has been established (see below). More focused questions include, *'Do you ever have difficulty passing soft stools as well as hard stools?'; 'Do you ever find it difficult to relax or let go in order to pass stool?'; 'Do you ever need to push around, or put your finger into, your vagina/rectum to help you pass stool?'*. Also ask about incontinence; *'Do you ever lose control of your bowels?'*. In the presence of urgency this suggests proctitis (see below), although 'overflow diarrhoea' due to faecal impaction typically presents with incontinence in the absence of urgency.

- **Diarrhoea:** Again, confirm what the patient means by 'diarrhoea'. Patients may use the term 'diarrhoea' to describe faecal incontinence, an increased frequency of normal stool or an acute diarrhoeal illness. Several definitions of diarrhoea exist—increased stool weight (>200g/day) or a general change increase in liquidity of stool.

There are several patterns of presentation of 'diarrhoea' which may give a clue to diagnosis:

- (i) Large volumes of watery stool, unaffected by fasting, suggesting a secretory process
- (ii) Moderate volume of loose stool, which improves on fasting, suggesting an osmotic process
- (iii) Bloody diarrhoea and abdominal pain, suggesting an inflammatory process
- (iv) Steatorrhoea, the passage of pale, greasy stools suggesting malabsorption (see Case 3 Weight Loss).
- (v) Urgency suggests proctitis, or inflammation of the rectum.
- (vi) Nocturnal diarrhoea, or waking from sleep to pass stool, suggests organic disease such as inflammatory bowel disease (IBD) or autonomic neuropathy due to diabetes mellitus

Therefore, ask specifically whether the diarrhoea improves at night or if the patient fasts for a while. Ask about pale stools and the presence of blood (melaena and haematochezia). Urgency or incontinence can be extremely disabling for patients: for example, they may be unable to use public transport. Appreciation of this through the use of empathy may help with rapport; *'I can see that must make your life very difficult, particularly if you have to travel to work'*. Ask sensitively how the patient is coping with their symptoms—how do they travel, do they use incontinence pads?

- **Associated GI symptoms:** Screen with focused questions for:
 - ♦ Upper GI symptoms: nausea, vomiting, dysphagia, reflux, dyspepsia, early satiety (see Case 2 Dyspepsia)
 - ♦ Abdominal pain: in particular, pain that is relieved by defaecation suggesting irritable bowel syndrome (IBS), or pancreatic pain suggesting chronic pancreatitis. (see Case 8 Abdominal Pain)
 - ♦ Weight loss (see Case 3 Weight Loss)
- **Associated systemic symptoms:**
 - ♦ Fever, tachycardia: systemic upset suggests IBD or infectious colitis (see below).
 - ♦ Extraintestinal symptoms: joint pains, oral ulceration, rash, red or painful eyes. These may be features of IBD or may occur following GI infection (reactive arthritis or Reiter's syndrome; see Case 23 Joint Pains).

- **Associated risk factors:**

If the diarrhoea is of recent onset:

- ♦ 'Have you recently started any new medications?'; (see below).
- ♦ 'Have you noticed any foods that make your symptoms better or worse? How about dairy products?'. Patients often attribute their symptoms to diet, although genuine food allergy/intolerance is rare as is lactose intolerance and coeliac disease. True lactase deficiency causing lactose intolerance is common in African and Asian populations, and is diagnosed using a lactose-hydrogen breath test. In the remaining patients this symptom should not be ignored, since a systematic food elimination approach under the supervision of a dietitian may be indicated.
- ♦ 'Have you recently had any antibiotics? Have you been hospitalized or attended hospital in the last 2 months?' *Clostridium difficile* infection is an increasing cause of acute diarrhoea in both hospitals and the community. These patients may have systemic symptoms such as fever. Viral infections may also be acquired from healthcare settings.
- ♦ 'Have you ever had surgery on your abdomen? In particular, have you had surgery to your gall bladder, surgery for ulcers, or removal of a segment of intestine?' GI surgery may cause abdominal pain and intestinal obstruction due to adhesions. Bile acid malabsorption is also a cause of chronic watery diarrhoea—this occurs following terminal ileal resection or following cholecystectomy or vagotomy.
- ♦ 'Have you been in contact with anyone with similar symptoms?' 'Have you eaten anything unusual over recent days?' 'How long after eating did the symptoms start?'
 - <6 hours following ingestion—*Staphylococcus aureus* infection (mayonnaise, poultry); *Bacillus cereus*
 - 8–14 hours—*Clostridium perfringens* infection (re-heated meat or poultry).
 - >14 hours—viral infection

B. Relevant medical and family history:

- Ask about a past personal and family history of IBD and colorectal carcinoma (CRC).
- Other conditions associated with constipation include:
 - ♦ Hypothyroidism
 - ♦ Hypercalcaemia—due to malignancy, hyperparathyroidism, sarcoidosis, etc.
 - ♦ Previous spinal cord injury or spinal surgery
 - ♦ Diverticular disease
- Other conditions associated with diarrhoea include:
 - ♦ Diabetes mellitus (see below)
 - ♦ Chronic pancreatitis

- Hyperthyroidism
- Antibody mediated autoimmune disease (eg. vitiligo, Grave's disease)—associated with coeliac disease
- Systemic sclerosis—associated with small bowel bacterial overgrowth
- Carcinoid syndrome—patients may have diarrhoea associated with flushing, wheeze, and rash (pellagra)

C. Medications and interactions:

- Several medications (including alcohol) can cause diarrhoea:
 - Antibiotics
 - ACE inhibitors
 - Digoxin
 - SSRIs
 - Statins
 - Proton pump inhibitors – particularly lansoprazole.
 - Laxatives
 - Magnesium in antacid preparations

D. Social issues:

- Alcohol excess is a cause of diarrhoea and of chronic pancreatitis.
- Cigarette smoking is a common cause of exacerbation of Crohn's disease, and a risk factor for colorectal cancer.
- Dietary habits, such as the use of laxatives, should also be enquired about. This may be related to underlying psychological disease such as an eating disorder, and should therefore be approached sensitively. Excessive use of sugar-free gum may also cause diarrhoea, since sorbitol may be an inadvertent source of laxative.
- Ask sensitively about HIV risk factors (see Case 17 Sexually Transmitted Infection). Diarrhoea in HIV is common, and may be due to infection in an immunocompromised host, or due to other complications such as small intestinal lymphoma. Infectious agents include *Cryptosporidium*, *Microsporidium*, *Isospora*, *Cytomegalovirus* (CMV), and *Mycobacterium avium* complex.
- Anal intercourse also predisposes to infectious proctitis, in the absence of HIV, by agents such as *Neisseria gonorrhoea* and *Chlamydia trachomatis* (causing lymphogranuloma venereum). These infections are often mistaken for proctitis due to ulcerative colitis.

Formulating a plan of action

- Use the technique of summarizing to demonstrate to the patient that you have heard their concerns, and to clarify what may be a complicated history. At this point, it may be helpful to ask openly, 'What do you think may be causing your symptoms?'. A significant proportion of patients have functional, non-organic symptoms, although this is difficult to accept for many. Further sensitive questioning regarding other aspects of the patient's life may be helpful, 'Has there been a particular change in your life during the time you have had these symptoms?', 'Did the symptoms come on during a particular time of stress?'.
 - If there is considerable anxiety, the patient may insist on further investigation even if not indicated. In such cases, conflict resolution is a key communication skill that the candidate must demonstrate (see Case 5 Family History of Cancer).
 - Explain to the patient that initial investigations will involve blood tests, and possibly stool samples if the diarrhoea is of recent onset (see below). Initial tests to consider include:
 - FBC, electrolytes, LFTs, albumin, CRP.
 - Thyroid function tests.

- Iron studies, serum B12, calcium, vitamin D, prothrombin time (markers of iron-deficiency anaemia (IDA) or malabsorption).
- Coeliac antibodies.
- Stool microscopy, culture and sensitivity including *Clostridium difficile* toxin
- Faecal elastase (low in chronic pancreatitis), faecal sudan stain (qualitative marker of fat malabsorption).
- Some patients may merit further investigations (see below). The reasons for this, along with the risks and benefits, should be clearly explained to the patient. Tell the patient that a follow-up appointment will be given to discuss the results of the above tests, and establish the need for further investigations (such as imaging or endoscopy).

Questions commonly asked by examiners

How do you treat a patient with a flare of IBD colitis?

The principles of treatment of IBD-related colitis are similar, regardless of whether the aetiology is Crohn's disease or ulcerative colitis (UC). The differences relate to the decreased efficacy of 5-ASA drugs in Crohn's disease, and whether anti-TNF (tumour necrosis factor) therapy is of benefit in acute UC.

All patients with a flare of IBD should have infection excluded by stool cultures for community-acquired bacterial pathogens and *C. Difficile*.

- *Proctitis*: First-line treatment is topical 5-ASA suppositories used once-daily, with steroid foams used once-daily as a second-line addition.
- *Mild/moderate left-sided colitis*: In the absence of systemic symptoms, patients can be treated with 5-ASA enemas, with steroid foam enemas as a second-line addition. High-dose oral 5-ASA tablets can be added to topical 5-ASA therapy, in a 'top-down, bottom-up regimen'. For example, 2g twice-daily mesalazine may be added to a 1g daily mesalazine enema.¹ If rectal bleeding persists beyond 10–14 days, oral steroids should be added.²
- *Extensive colitis*: Patients with severe colitis, as determined by the Truelove and Witt criteria (see below) require hospitalization and should receive intravenous steroids and subcutaneous low molecular weight heparin for thromboprophylaxis. Patients should have a daily abdominal X-ray to rule out progression to toxic megacolon (colonic diameter >6cm), and GI surgeons should be involved early in the patient's management. For patients with ulcerative colitis, if the patient's CRP is >45mg/L after 72 hours of intravenous steroids along with greater than 3 stools in 24 hours, the risk of colectomy on that admission is 85%.³ Therefore, these patients should be considered for colectomy after 3 days of medical therapy. Alternatives include intravenous ciclosporin, or for UC a single dose of infliximab. These decisions should be made by a specialist IBD team, including physicians, surgeons, and nurses.

Box 2 Truelove and Witt Criteria for Severe Colitis

Bowel frequency >6 times per 24 hours *and* one or more of the systemic manifestations:

- Haemoglobin <10.5g/dL
- erythrocyte sedimentation rate (ESR) >30mm/hr
- Pulse rate >90 beats per min
- Temperature >37.5°C

What are the causes of diarrhoea in diabetes mellitus?

Potential causes of diarrhoea in the diabetic patient include:

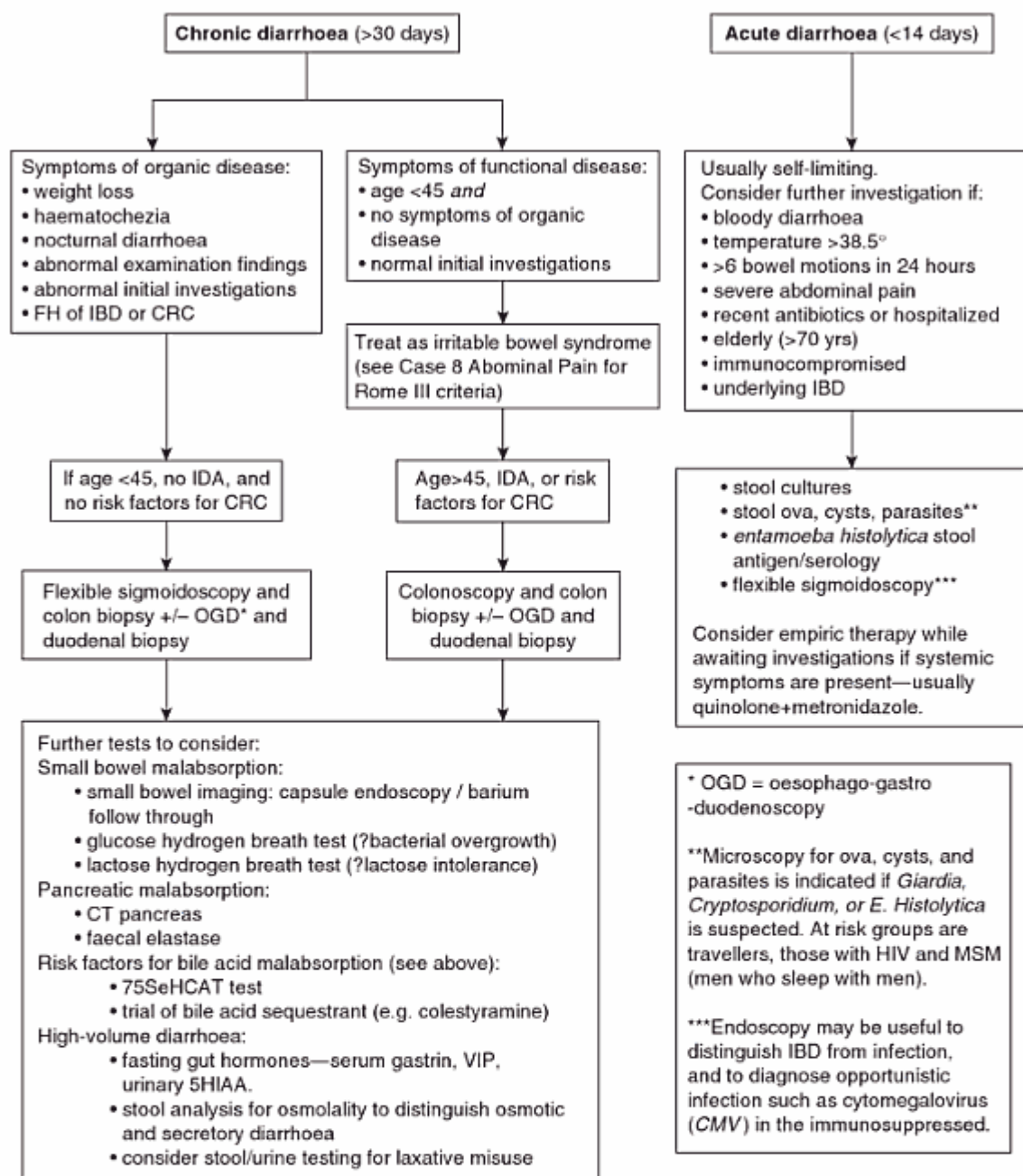


Figure 4.1

Source: Modified from Thomas, et al. Guidelines for the investigation of chronic diarrhoea, 2nd edn. Gut. 2003; 52 Suppl 5:v1–15.

- Diabetic autonomic neuropathy of the enteric nervous system. This may cause delayed or increased small bowel motility. The diarrhoea is typically painless and watery, and occurs at night. Anorectal dysfunction may also occur causing faecal incontinence.
- Small bowel bacterial overgrowth, causing chronic diarrhoea, may occur in patients with decreased small bowel motility.
- Coeliac disease is associated with type 1 diabetes mellitus, since both are antibody-mediated autoimmune diseases. The prevalence of coeliac disease in type 1 diabetic adults is around 5%.

- Metformin causes GI side effects, including diarrhoea, in 30% of patients.
- Sorbitol in artificial sweeteners, frequently used by diabetic patients, may cause osmotic diarrhoea.

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Case 5 ♦ Family History of Cancer

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 53-year old lady, whose son was recently diagnosed with metastatic colon cancer. She has a past history of endometrial cancer, for which she underwent hysterectomy and oophorectomy. I am concerned that she may need a colonoscopy. Please could you see and advise.

Many thanks for your opinion.

Acquiring the history

Acquiring the family history is a routine part of clinical practice. However, an accurate and detailed family history has gained renewed importance with the advent of sophisticated molecular techniques to diagnose hereditary cancer syndromes. Indeed, a detailed family history is essential to differentiate asymptomatic patients who require further screening from patients who require reassurance without invasive investigation.

A. History of presenting complaint:

Since the patient may be concerned although asymptomatic, initial open questioning is vital to identify symptoms which the patient may perceive as trivial yet may be clinically relevant: 'How have you been feeling recently? Have there been any recent changes to your health or your lifestyle?'

Remember that patients with a recent bereavement or illness in their family are likely to be anxious or still grieving. In these situations, *empathic* responses are usually the most effective ways of developing a rapport with the patient, rather than using a *sympathetic* response or ignoring the patient's anxieties altogether (see introduction for further detail on empathic and sympathetic responses). An example of an empathic response may be 'I can see things have been difficult for you during your son's illness. Tell me how you have been feeling?'. By contrast, a sympathetic response would invoke 'pity' as a means of dealing with the patient's anxieties and proceeding with the history according to the 'physician's agenda'—'I'm so sorry to hear about your son's illness. Have you had any health problems recently?'

Even in the time-pressured situation of the MRCP (PACES) examination, examiners will look favourably on candidates who persevere with *active listening* and *empathy* rather than pursue their own agenda or turn the history into an 'interrogation'.

More focused questioning should cover symptoms relevant to the common inherited cancer syndromes:

- Lynch syndrome (Hereditary Non-Polyposis Colorectal Cancer)
- 1. Colonic symptoms:
 - change in bowel habit?
 - rectal bleeding?
 - weight loss?
 - previous colonoscopy / colonic polyps?
- 2. Extra-colonic symptoms:
 - post-menopausal vaginal bleeding (endometrial cancer)?
 - abdominal or pelvic pain (ovarian cancer)?
 - jaundice or epigastric pain (hepatobiliary / pancreatic cancer)?
 - early satiety or dyspepsia (gastric cancer)?
 - any unusual skin lesions or moles (sebaceous adenomas, carcinomas, and keratocanthomas)?
- Multiple endocrine neoplasia:
 - history of thyroid / parathyroid disease?
 - symptoms of hypercalcaemia (musculoskeletal pain, constipation)?
 - history of pituitary disease?
 - visual disturbance—particularly loss of peripheral vision?
- BRCA1 and BRCA2 mutations:
 - Past history of breast lumps / lumpectomy?

B. Family history:

Determine the family history of the patient, going back three generations. For each cancer, attempt to document:

- Age of diagnosis (rather than treatment or death)?
- Site/type of cancer?
- At which medical unit was the patient treated?
- Type of treatment (surgical/adjvant/neo-adjvant)

Differentiate first-degree relatives (parent, sibling, child) from second-degree relatives (grandparent, cousin, niece, nephew). Also ask about a family history of colonic polyps, for which the patient's relative may have had colonoscopy and polypectomy.

For the patient referral above, the Amsterdam II criteria* may be used to determine the risk of Lynch syndrome:

- Three or more relatives with Lynch syndrome-associated cancers (colorectal cancer, endometrial cancer, small-intestine tumours, or renal cell cancer), one of whom is a first-degree relative of the other two and in whom FAP has been excluded.
- Lynch syndrome-associated cancers involving at least two generations.
- One or more cancers diagnosed before the age of 50.

* These criteria can be remembered by the '3-2-1 rule': 3 affected members over 2 generations, of whom 1 is under age 50.

However, these criteria only have a sensitivity of 50% for Lynch syndrome as defined by genetic testing. Therefore, if suspicion of Lynch syndrome remains high but the family does not meet Amsterdam II criteria, then they should not be falsely reassured but should be referred for genetic counselling and genetic testing for Lynch syndrome (see below). In some cases, tumour specimens from the affected relative may be obtained and tested for genetic markers of Lynch syndrome, so asking where the patient's relative underwent treatment is an important question. Even if genetic testing for Lynch syndrome is negative, these patients remain at increased risk due to their significant family history and require a higher level of colon surveillance (see below).

C. Relevant past medical history:

- Other risk factors for colorectal cancer: adenomatous polyps, inflammatory bowel disease, diabetes mellitus, cigarette smoking.
- Other risk factors for breast cancer: benign breast disease, early menarche, later menopause, use of HRT. High parity and oophorectomy before the age of 40 are protective.

E. Relevant social history:

As with other cases, the social history should include questioning about lifestyle, occupation and disability. Additionally, as mentioned above, the patient may still be grieving about a recent bereavement. Although the focus of this history is not the patient's stage of bereavement, a brief open question about the patient's loss will be appreciated by the examiners: *'How have you been coping since the death of your son?'* The phases of grief have been described as: denial and isolation, anger, bargaining, depression, and finally acceptance.¹ Whilst there is not time to counsel the patient fully in the MRCP (PACES) examination, the good candidate will be able to make an assessment of the patient's progress through these stages of grief.

Formulating a plan of action

- Reassure the patient that in the absence of symptoms, significant pathology is unlikely. However, in view of the family history, it is sensible to investigate further to determine the risk of cancer and treat any 'small growths (polyps) that may turn into cancer over years'.
- If the patient's family history fulfils the Amsterdam II criteria, or if the patient has colonic symptoms, then explain that colon investigation will be necessary. This may be either colonoscopy (optical colonoscopy) or CT (computed tomography) colonography (virtual colonoscopy). Both require bowel preparation, although optical colonoscopy is the preferred modality (see below).

However, a definitive diagnosis of Lynch syndrome requires genetic testing, initially on tumour tissue from the affected relative: microsatellite instability (MSI), and immunohistochemistry (IHC). This is because tumours from Lynch kindreds typically have high MSI, and they also do not possess the Lynch syndrome gene product, which can be demonstrated by IHC. The next step is gene testing of the patient for mismatch-repair gene mutations (see below). Since this diagnosis carries implications for the whole family, testing is only done following genetic counselling, and if there is a strong suspicion of Lynch syndrome.

- Finally, even if the patient does not fulfil the Amsterdam II criteria, it may be that the patient requires colon screening at an earlier age than usual (before the age of 50). There is a marginal benefit in colon screening for people with one affected first-degree relative (FDR) aged under 45 years or two affected FDRs. Patients with a lesser family history do not require surveillance over and above that of the general population, and should be reassured as such.
- If the patient's anxiety persists over a risk of cancer, despite no clinical features to mandate colon surveillance, then the initial response should be empathic; *'Since your son's illness was*

such a shock, I can see that you must be worried about your own health'. It may be necessary to explore the reasons for the patients' anxiety, most likely due to not completing the grieving process (above), although this should be done sensitively without engaging in conflict about further tests.

If the patient is adamant about the need for a colonoscopy, then the role of the physician is to share their agenda with the patient at the same time as facilitating patient autonomy (see Calgary–Cambridge model in introduction). Conflict resolution is a key communication skill and frequently arises in the MRCP (PACES) examination, especially in the setting of a patient demanding a test/treatment. In general, conflict can be minimized by developing rapport with the patient and using empathic responses. Reassurance should only be provided once the patient's agenda has been established—premature reassurance may sound false and be counter-productive. Facilitating autonomy involves explanation about the risks/benefits of colonoscopy in this setting, and fully explaining other options including routine blood tests and giving the patient time to consider their options. Ideally, the consultation should finish with a shared plan made jointly between physician and patient.

Questions commonly asked by examiners

What do you know about the hereditary colorectal cancer syndromes?

(i) Lynch syndrome

This is the most common of the hereditary colon cancer syndromes, accounting for 2–3% of all CRC and 2% of uterine cancer. It is an autosomal dominant disorder caused by a germline mutation in one of several DNA mismatch repair genes:

- hMSH2 (human MutS homolog 2)—chromosome 2p16
- hMLH1 (human MutL homolog 1)—chromosome 3p21
- hPMS1 (human postmeiotic segregation 1)—chromosome 2q31
- hPMS2 (human postmeiotic segregation 2)—chromosome 7p22
- hMSH6 (human MutS homolog 6)—chromosome 2p16

Patients with Lynch syndrome have a markedly increased risk of CRC, as well as other cancers including gynaecological, urological, gastric, small intestine, biliary, pancreatic, skin, and brain. The overall cancer risk is 80%, and for CRC is 50–70%. CRC presents typically 10–20 years earlier than sporadic CRC. Cancers are thought to develop from adenomas in similar fashion to sporadic CRC, although the polyps have a 'flat' morphology, the cancers occur more proximally in the colon and are usually less differentiated.

Two variants of Lynch syndrome are Muir-Torre (Lynch syndrome with associated sebaceous tumors, cutaneous keratoacanthomas, and visceral carcinomas) and Turcot syndrome (Lynch syndrome with associated brain tumors, typically gliomas). These are likely to be types of Lynch syndrome rather than distinct disease entities.

The genetic hallmark of Lynch syndrome is MSI. This refers to the expansion or contraction of short repetitive DNA sequences due to defective DNA repair. MSI can be tested for in tumours using the polymerase chain reaction (PCR) to amplify a DNA sequence. The presence of MSI is highly sensitive for Lynch syndrome, although not specific, hence further genetic tests are required to confirm the diagnosis.

Surveillance for patients from Lynch families involves annual/biannual colonoscopy for patients beginning at age 20–25. Patients also undergo annual ultrasound screening for ovarian and endometrial cancer from age 30–35, annual urinalysis and urine cytology from age 25–35, annual skin surveillance and periodic upper GI endoscopy.

Although regular drug treatment with aspirin has been shown to decrease the incidence of sporadic colonic adenomas and CRC, no benefit was found in a recent large study of patients with Lynch syndrome.²

(ii) Familial adenomatous polyposis

Familial adenomatous polyposis (FAP) is also an autosomal dominant disorder, characterized by the presence of more than 100 (often hundreds) of colorectal adenomas. The disease is caused by mutations in the adenomatous polyposis coli (APC) gene. FAP accounts for less than 1% of the burden of CRC. However, FAP is also associated with an increased risk of duodenal ampullary carcinoma, follicular or papillary thyroid cancer, and gastric carcinoma.

Gardner's syndrome is a variant of FAP with additional extra-intestinal features, such as desmoid tumours, sebaceous or epidermoid cysts, lipomas, osteomas, supernumerary teeth, and gastric polyps. Turcot's syndrome is a further variant of FAP associated with central nervous system (CNS) medulloblastomas.

The risk of CRC in classical FAP is almost 100% by age 45. For this reason, most patients undergo colectomy after adolescence—this may be a complete proctocolectomy with ileo-anal anastomosis or a subtotal colectomy with ongoing surveillance of the rectum. Patients should also have upper GI endoscopy for surveillance of gastroduodenal polyps. Cox-2 inhibitors have also been shown to reduce GI polyps in FAP,³ unlike in Lynch syndrome, although they should be used in addition to colectomy rather than as a replacement for surgery.

(iii) Peutz-Jeghers Syndrome

Peutz-Jeghers syndrome (PJS) is also an autosomal dominant syndrome. It is characterized by the presence of pigmented lesions on the lips and buccal mucosa, and multiple GI hamartomatous polyps (see Volume 1, Case 136 Peutz-Jeghers Syndrome). Hamartomas occur most frequently in the small intestine (65–95%), although they may also occur in the colon (60%) and stomach (50%).

The cancer risk is markedly elevated in PJS, for sites including the colon, small intestine, stomach, pancreas, and breast. The lifetime risk of CRC is 39%. Genetic testing is not widely available, hence FDRs are screened with upper and lower GI endoscopy and small intestine imaging. Affected individuals also require surveillance for GI, pancreatic, and breast tumours.

What do you know about colorectal cancer screening in the United Kingdom?

CRC screening using faecal occult blood (FOB) testing, annually or biannually, has been shown to reduce cumulative CRC mortality over an 18-year follow-up.⁴ On the basis of this evidence and pilot studies, a CRC screening programme was introduced in the UK in 2009. This involves offering biannual FOB testing for all individuals aged 60–69. Abnormal tests are found in 2% of the population, and these are followed up with a screening colonoscopy. The major disadvantage of the FOB test is the high false positive rate.

Aside from population screening, which other patients require surveillance for colorectal cancer?

What is the role of CT in screening for colorectal cancer?

CT colonography (virtual colonoscopy) is performed following bowel preparation with laxatives in a similar fashion to optical colonoscopy. Subsequently, the colon is inflated with air, passed via rectal tube, and CT images are acquired. Sophisticated software is then used to reconstruct an image of the colon which is viewed in a similar way to conventional optical colonoscopy. The sensitivity of CT for polyps greater than 1cm in size and for CRC is similar to colonoscopy.

The advantages of CT are a lower risk of perforation, lack of sedation, and the possible benefit of detecting other extra-colonic lesions on the images. However, patients with polyps will need a

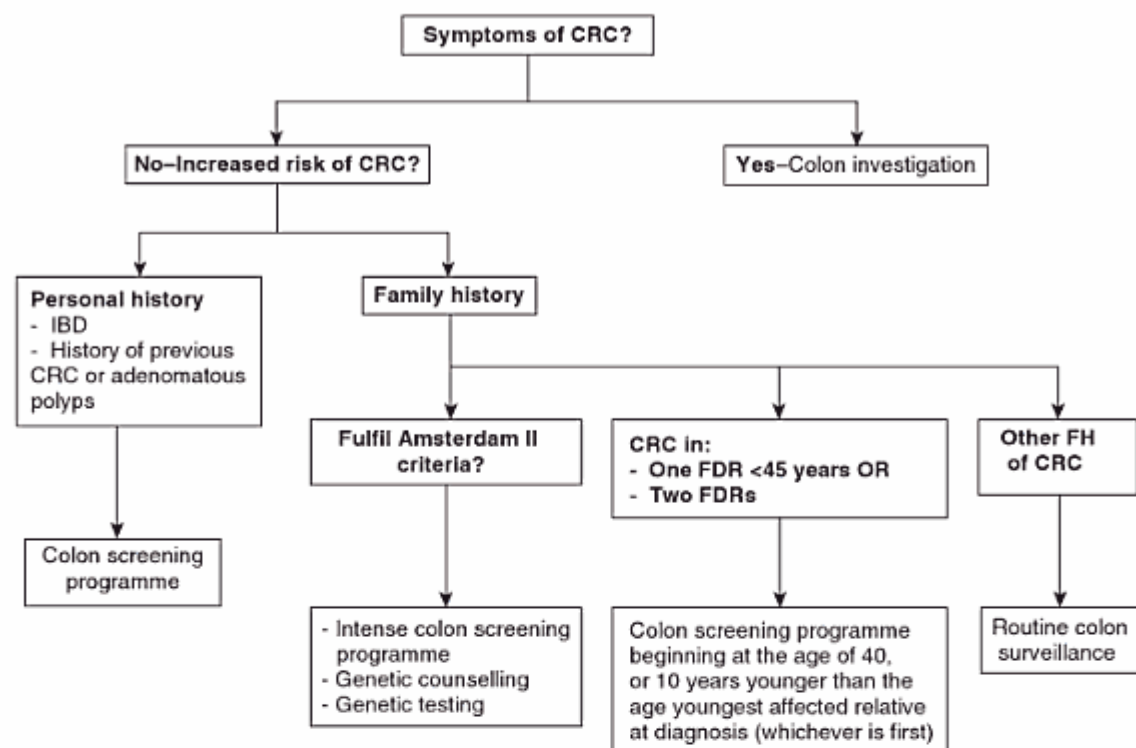


Figure 5.1

subsequent colonoscopy to remove them, thus leading to an extra procedure with cumulative cost and risk. A further disadvantage is the radiation dose—less than a barium enema, but the long-term effects of repeated screening are unknown. Moreover, the trials of CT screening of CRC have been mainly performed in younger adults, so the efficacy of CT screening the elderly has not been proven.⁵

CT is currently used for patients unable (for example cannot lie on their side or cannot receive sedation) or unwilling to undergo colonoscopy. It may play a wider role in screening if future studies confirm safety and effectiveness in older populations.

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Case 6 ♦ Dysphagia

INFORMATION FOR THE CANDIDATE

Dear Doctor,

This 78-year-old lady has had difficulty swallowing for the last two months. She is struggling with solids and liquids, and has lost a stone in weight. Her past medical history is of arthritis and hypertension, for which she takes amlodipine, bendroflumethiazide, and diclofenac. I wonder if she needs an endoscopy?

Many thanks for your opinion.

Acquiring the history

The history is a vital part of the assessment of patients with dysphagia, since the history can distinguish oropharyngeal from oesophageal dysphagia in over 80% of cases. For this reason, dysphagia is a relatively common case in the MRCP (PACES) history-taking station.

A. History of presenting complaint:

Begin with an open question to allow the patient to describe their symptoms. *'I gather from your doctor that you have had difficulty swallowing. Could you tell me about it?'; 'Describe to me what happens when you swallow.'*

The aim of this part of the history is to elicit the features that differentiate oropharyngeal and oesophageal dysphagia, using focused questions for clarification only after the patient has finished describing their symptoms.

Oropharyngeal dysphagia	Oesophageal dysphagia
Difficulty <i>initiating</i> swallowing	No difficulty in initiation, but food 'sticks' soon after swallowing
Associated nasal <i>regurgitation</i>	Worse with solids than liquids
Associated <i>coughing, choking</i>	May be associated <i>reflux</i> and <i>dyspepsia</i>
Worse with liquids than solids	Symptoms may be <i>progressive</i>
History of aspiration pneumonia	May be associated with <i>alarm symptoms</i>
History of neurological disease, including stroke	
Associated with other neurological symptoms (e.g. dysarthria, weakness)	

Consider the following areas:

- Age of onset—older age, male sex, and the presence of heartburn or weight loss predict mechanical (obstructive) causes of dysphagia.
- Characteristics
 - ♦ Initiation – *'Do you have trouble starting your swallow?'; 'Do you need to turn your head or change position to swallow?'*. Patients with oropharyngeal dysphagia, who have difficulty transferring food from the mouth to the pharynx, may reposition their body or use their fingers to move food into the pharynx.

- Associated features of regurgitation and aspiration. *'Do you cough or choke after you swallow?'; 'Does food ever come back up your nose when you swallow?'*
- Type of food: *'Do you have difficulty with liquids, solids, or both?'* If mainly solids, *'Is it worse with tough foods like bread and meat? What food have you been eating?'* If mainly liquids, *'Have you tried anything to make liquids easier to swallow? Is it easier with thicker liquids?'*

Dysphagia that is worse with solids is likely to be a mechanical cause of dysphagia. Symptoms that are worse with liquids suggests an oropharyngeal cause. Dysphagia that affects solids and liquids equally may be an oesophageal motility disorder.

- Progression—*'Are your symptoms getting worse, or do they come and go? Have you lost weight?'* Rapidly progressive symptoms raise the possibility of malignancy. Intermittent, non-progressive symptoms suggest an oesophageal web or ring.
- Location—*'Where exactly does the food get stuck?'* Patients with oropharyngeal dysphagia may point to their pharynx, whereas those with oesophageal dysphagia are more likely to point to their sternum.

Associated symptoms:

- **Gastrointestinal**
 - Odynophagia—*'Is it painful to swallow?'* This suggests an inflammatory cause, such as oesophagitis or oesophageal infection.
 - Chest pain—*'Does it hurt anywhere in your chest when you swallow?'* Associated with motor disorders of the oesophagus (e.g. diffuse oesophageal spasm, achalasia, systemic sclerosis).
 - Heartburn / dysphagia—*'Have you had any heartburn during the time you have been unwell? What about during the night?'* Acid reflux predisposes to oesophagitis, oesophageal strictures, and oesophageal adenocarcinoma.
 - Acid brash / regurgitation—*'Do you ever wake up with an acidic taste in your mouth in the mornings?'; 'Do you ever bring up food you have just eaten?'* Acid brash is associated with acid reflux. Passive regurgitation may occur with benign oesophageal lesions, such as an oesophageal diverticulum or achalasia.
- **Neurological**
 - Tremor—Parkinson's disease is associated with neuromuscular dysphagia.
 - Ptosis or intermittent weakness—Myasthenia gravis is also a cause of neuromuscular dysphagia.
 - Weakness, dysarthria—Stroke is the most common cause of oropharyngeal dysphagia—45% of all stroke patients experience dysphagia at 3 months.

B. Relevant medical history:

Past medical history:

- Neurological disease: Stroke, Parkinson's disease, multiple sclerosis, myasthenia gravis, amyotrophic lateral sclerosis, cerebral palsy.
- Sjögren's disease—ask about dry eyes and a dry mouth.
- Systemic sclerosis.
- Osteoarthritis—cervical spondylosis may cause oesophageal compression.
- Previous radiotherapy to the thorax—radiation oesophagitis.
- Valvular heart disease—an enlarged left atrium may also compress the oesophagus.
- HIV / immunosuppressive treatment—predispose to oesophageal infection.
- Atopy / hayfever—associated with eosinophilic oesophagitis.

C. Medications:

Drugs causing oesophageal injury

Several drugs are corrosive to the oesophageal mucosa, and can cause 'pill-oesophagitis':

- Doxycycline
- Bisphosphonates
- NSAIDs
- Iron sulphate

Drugs affecting lower oesophageal sphincter pressure

- Nitrates
- Calcium antagonists

D. Social issues:

- Take a careful smoking and alcohol history. Both cigarette smoking and alcohol use are associated with oropharyngeal, laryngeal, and oesophageal squamous cell carcinomas. Cigarette smoking is also a risk factor for oesophageal adenocarcinoma.
- Ask how the patient has been feeding themselves, and maintaining their nutritional intake.
- Sensitively enquire about psychological symptoms—*globus pharyngeus* is the sensation of a lump or foreign body in the throat, in the absence of dysphagia, odynophagia, and evidence of gastro-oesophageal reflux. Symptoms are thought to be a form of somatization. 'How have things been in your life generally over recent months? What was going on in your life at the time the symptoms came on? Do you have any particular stresses or worries in your life at the moment?'

Formulating a plan of action

Explain to the patient that further investigation will be needed to determine the cause of dysphagia. The most likely initial test is upper GI endoscopy for almost all cases of dysphagia (see Case 2 Dyspepsia, for alarm symptoms for endoscopy) even if a neuromuscular cause is suspected—to rule out a concomitant oesophageal lesion. An alternative approach is to arrange a barium study first if an oropharyngeal lesion or oesophageal diverticulum is suspected. This is because intubation of the upper oesophagus is not well visualized at endoscopy, so there is a small risk of perforation if there is upper oesophageal pathology.

Explain that the endoscopy may be done with or without a sedative, and is a very quick day-case procedure. The initial results will be available on the day of the test, but a follow-up appointment will be arranged to discuss the results of biopsies and blood tests.

Questions commonly asked by examiners

What is the differential diagnosis of oesophageal dysphagia?

- *Peptic stricture*—this is complication of acid reflux, affecting 10% of patients who seek medical attention for reflux. Symptoms are of gradually progressive dysphagia to solids. Endoscopic biopsy is used to confirm that the stricture is benign, following which the stricture can be endoscopically dilated.
- *Oesophageal carcinoma*—squamous cell carcinoma is associated with smoking and alcohol use, and is common in South East Asia. Adenocarcinoma is associated with acid reflux and Barrett's oesophagus, and the incidence is one of the most rapidly increasing amongst cancers in the Western world. Symptoms are of rapidly progressive dysphagia and weight loss.

- *Achalasia*—is characterized by loss of peristalsis in the distal oesophagus, and failure of the lower oesophageal sphincter to relax. Almost complete dysphagia may occur to both solids and liquids. Oesophageal manometry studies will diagnose elevated lower oesophageal pressure. However, endoscopy is required to rule out 'pseudo-achalasia', due to gastric cancer causing malignant infiltration of the myenteric plexus. Treatment may be endoscopic botulinum injection, endoscopic dilatation, or surgery.
- *Diffuse oesophageal spasm*—is a motility disorder characterized by simultaneous, uncoordinated oesophageal contractions. Symptoms are of intermittent chest pain and dysphagia, which may be triggered by acid reflux or hot and cold food. Calcium antagonists are used for therapy.
- *Systemic sclerosis*—is associated with low amplitude oesophageal contractions and acid reflux. Peptic strictures are also common. Patients may also experience delayed gastric emptying, leading to recurrent vomiting, and are also at risk of small-bowel bacterial overgrowth causing chronic diarrhoea and malabsorption.
- *Eosinophilic oesophagitis*—is a condition associated with atopy and food allergies. It presents in younger patients with food bolus impaction.
- *Radiation injury*—radiation oesophagitis and subsequent fibrosis and stricturing may occur following radiotherapy to the trunk.
- *Extrinsic compression*—an enlarged left atrium, thoracic aortic aneurysm, or mediastinal lymph nodes may cause oesophageal compression.
- *Oesophageal infection*—i.e. fungal or CMV infection, may cause dysphagia and odynophagia in immunocompromised patients

Tell me about surveillance for Barrett's oesophagitis?

Barrett's oesophagitis develops as a consequence of chronic gastrooesophageal reflux disease (GORD), and predisposes to the development of adenocarcinoma of the oesophagus. The stratified squamous epithelium that normally lines the distal oesophagus is replaced by columnar epithelium.

The prevalence of Barrett's oesophagitis is between 1 and 4%. The rate of transformation to adenocarcinoma is 0.2–2% per year. Endoscopy has been used as a surveillance tool, on the basis that curable dysplasia may be detected earlier, although this approach has not yet been proven to reduce mortality. The British Society of Gastroenterology, and American Society for Gastrointestinal Endoscopy, recommend endoscopic surveillance every 2–3 years for patients who would be candidates for oesophagectomy.

What is the approach to the diagnosis of oropharyngeal dysphagia?

After upper GI endoscopy, and possibly oesophageal manometry, to rule out an oesophageal lesion, most patients are investigated under the supervision of speech therapists and ear, nose, and throat (ENT) surgeons. Video fluoroscopy is an X-ray study using barium, which is filmed during real-time from both anterior–posterior and lateral directions, to closely study the mechanisms of swallowing. The elevation of the hyoid bone and larynx, relaxation of the upper oesophageal sphincter and contraction of the pharynx can be examined.

If neuromuscular abnormalities are found, by features such as aspiration or pooling of barium, or muscle paralysis, treatment can be introduced. This may involve thickening of fluids, or manoeuvres during swallowing which reduce dysphagia and aspiration (e.g. head tilting, forced valsalva manoeuvre).

Case 7 ♦ Jaundice

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 49-year old man with a 5-day history of jaundice. He tells me his urine has also been dark in colour, but he is otherwise well with no pale stools or fevers.

Many thanks for your help.

Acquiring the history

Patients rarely complain of 'jaundice' themselves—they, or someone else, may have noticed a change in the colour of their eyes or skin, but more commonly they will complain of associated symptoms. Since jaundice usually is a sign of a serious illness, it is vital to allow the patient time to disclose their other symptoms before proceeding to closed questioning.

A. Presenting complaint:

Begin with an open question, without focusing on the jaundice referred to in the referral letter. 'I understand from your doctor that you haven't been feeling so well over recent weeks—when did you last feel your health was normal?'; 'How have you been feeling since?'; 'Can you tell me a little more about that...?'

B. History of presenting complaint:

Now focus on the onset and duration of the jaundice. 'When did you notice that your eyes/skin had changed colour?'; 'How have things progressed since then?'

- Acute onset (days):
 - ♦ Gall stone disease (choledocholithiasis, cholangitis)
 - ♦ acute hepatitis
 - ♦ Acute Budd-Chiari syndrome
 - ♦ Haemolysis
- Subacute onset (weeks–months):
 - ♦ Pancreatic and hepatobiliary malignancy
 - ♦ Intrahepatic cholestasis (eg. drug-induced, autoimmune, infiltrative liver disease)
 - ♦ Right-sided heart failure
- Recurrent episodes:
 - ♦ Gallstone disease (choledocholithiasis, cholangitis)
 - ♦ Disorder of bile transport (e.g. Gilbert's syndrome)

Ask about these associated symptoms:

- *Fever*: may occur in cholangitis, viral hepatitis, or rare causes of cholecystitis causing jaundice (e.g. Mirizzi's syndrome—compression of the hepatic duct by chronic inflammation in Hartmann's pouch of the gallbladder). Fever is also a feature of alcoholic hepatitis.
- *Right upper quadrant pain*: this suggests cholangitis or acute hepatitis in the context of jaundice. Other causes are Budd-Chiari syndrome causing acute liver failure, or Mirizzi's syndrome (as above). Right upper quadrant pain in the absence of jaundice has a wider

differential diagnosis (see Case 8 Abdominal Pain). Gradual-onset 'painless' cholestatic jaundice classically suggests pancreatic or bile duct malignancy, but drug-related or autoimmune cholestasis are also usually painless.

- *Confusion*: the presence of altered mental status strongly suggests a serious underlying cause, such as sepsis due to cholangitis, or hepatic encephalopathy due to acute or chronic liver failure. Other causes include intracranial haemorrhage as a consequence of coagulopathy caused by liver failure, hypoglycaemia due to liver failure, or a post-ictal state following a seizure due to alcohol or substance withdrawal.
- *Mucosal bleeding / bruising*: ask specifically about gum bleeding, nosebleeds, and easy bruising. Aside from coagulopathy caused by liver failure, other causes of mucosal bleeding and jaundice include disseminated intravascular coagulation (DIC) due to cholangitis and sepsis, thrombocytopenia due to portal hypertension (hypersplenism), thrombotic thrombocytopenic purpura (TTP), or severe malaria.
- *Back pain*: is a feature of viral hepatitis (along with right upper quadrant pain) and severe haemolysis.
- *Dark urine / pale stools*: these are classically symptoms of 'obstructive' jaundice, which causes excess conjugated bile to appear in the urine. Additionally, the lack of conjugated bile secreted into the intestines leads to a lack of stool pigment—'pale' stools. However, severe haemolysis may cause dark urine due to haemoglobinuria. Therefore, these questions may be better at following the progress of jaundice once the diagnosis is known, rather than distinguishing obstructive jaundice from haemolysis.
- *Pruritus*: is a feature of all cholestatic processes, including bile duct obstruction, drug-induced and autoimmune. Other systemic diseases causing pruritus include chronic renal disease, haematological malignancy, and thyrotoxicosis.
- *Weight loss*: involuntary weight loss is associated with pancreatic or hepatobiliary malignancy. Patients with advanced chronic liver disease are also usually malnourished, although their weight loss may be balanced by the development of ascites.

C. Associated risk factors:

Risk factors for viral hepatitis:

- Needle and blood exposure—shared needles, tattoos, piercings, dental, or medical care abroad?
- Sexual history—ask sensitively about sexual contacts, type of encounter (heterosexual, homosexual), number of partners and the use of barrier protection (see Case 17 Sexually Transmitted Infection).
- Exposure to hepatitis A—exposure to individuals with viral illness or history of eating shellfish? Document travel history in last 6 weeks.
- Recent immunosuppression—patients who may be asymptomatic carriers of hepatitis B may develop liver failure due to viral reactivation after starting immunosuppressant therapy (e.g. steroids, chemotherapy).

Risk factors for alcoholic hepatitis and acute liver failure

- Alcohol intake—ask openly and non-judgementally about alcohol intake. Many patients may under-report the amount of alcohol they consume. After an open question, such as 'How much alcohol do you drink in an average weekend?', it may help to provide the patient with a choice of answers, to allow them to acknowledge their alcohol intake without fear of judgement. For example, 'Would you say you drink more like one to two beers/whiskies a night, or eight to ten beers/whiskies a night?'. It may also help to provide the patient with a social 'excuse' for drinking, 'A lot of people find that a drink helps them sleep at night—do you ever do the same?'.

- Medications—‘Do you take painkillers or cold remedies containing paracetamol?’; ‘Have you recently started any new medications or over-the-counter remedies?’; ‘Do you drink herbal teas or herbal remedies?’. Several drugs are associated with acute liver failure, including paracetamol, antituberculous medications and antiepileptics. Herbal teas containing pyrrolizidine alkaloids may also cause hepatic veno-occlusive disease and liver failure.
- Vascular risk factors for Budd-Chiari syndrome—‘Have you, or anyone in your family, ever had a blood clot?’; ‘Have you ever been diagnosed with a blood disorder?’. Up to half of cases of Budd-Chiari syndrome may be due to an underlying myeloproliferative disorder, such as polycythaemia vera or essential thrombocythaemia. In women of reproductive age, also ask about use of the contraceptive pill.

Risk factors for cholestatic jaundice

- Gallstones—ask about a history of gallstones, and a past history of post-prandial right upper quadrant pain. Also ask about risk factors for gallstones, such as a haemolytic anaemia (pigment stones), previous parenteral nutrition, gastric bypass surgery, or use of somatostatin analogues such as Lanreotide for carcinoid syndrome or acromegaly.
- Previous hepatic or biliary surgery—risk of biliary strictures.
- Previous pancreatitis may result in pancreatic pseudocyst formation, which can compress the biliary tree.
- History of sickle cell anaemia—associated with haemolysis and biliary disease due to pigment gallstones, but also ischaemic cholangiopathy caused by sickling in the end arteries supplying the biliary tree.
- History of ulcerative colitis—associated with primary sclerosing cholangitis in 1–4% of cases (67% of patients with primary sclerosing cholangitis have ulcerative colitis).

D. Travel history:

The incubation period of hepatitis A virus infection is 4–6 weeks. Therefore, document all areas visited in the preceding 2 months. Hepatitis B virus infection is also common (prevalence up to 20%) in South East Asia, Eastern Europe, and sub-Saharan Africa—ask specifically about travel to these areas. Additionally, liver fluke infection (Clonorchiasis and Opisthorchiasis) may be acquired in South East Asia. These infections may cause biliary strictures, resulting in jaundice and recurrent cholangitis.

E. Relevant family history:

Ask about a family history of liver disease, hepatitis, or blood disorders. Haemochromatosis, Wilson’s disease, and Gilbert’s syndrome are familial, as are several haemolytic anaemias such as sickle cell anaemia and G6PD deficiency. Hepatitis B may also be vertically transmitted.

F. Medications and vaccines:

- Ask about all prescription, over-the-counter, and herbal medications. Document starting date and compliance for each drug.
- Ask specifically about vaccinations for hepatitis B (see above) and hepatitis A.

G. Gynaecological and obstetric history:

- In women of child-bearing age, ask about last menstrual period and chances of pregnancy (see below - liver disease in pregnancy)
- In pregnant women, ask about a history of pre-eclampsia and liver abnormalities in previous pregnancies (see below).

Formulating a plan of action

- Use the technique of summarizing to ensure that you have covered all of the patient’s concerns. This is especially likely if the patient was unaware of the jaundice prior to the consultation. ‘Is there anything that we haven’t discussed that you are worried about?’

- Explain that a full examination, blood tests, and an ultrasound scan of the liver will be necessary to determine the diagnosis.
- Aside from routine blood tests and a screen for causes of hepatitis and chronic liver disease (see below), the most important test is an ultrasound scan which will differentiate 'obstructive' from 'non-obstructive' jaundice. The normal diameter of the common bile duct on ultrasound in adults is 7mm. This may increase in the very elderly or following cholecystectomy, but in general if the common bile duct is greater than 7mm in diameter this suggests biliary obstruction.

Blood tests in patients presenting with jaundice:

- FBC
- LFTs (AST, ALT, γ GT, ALP)*
- Urea, creatinine, electrolytes
- Bilirubin
- Albumin, prothrombin time*
- Viral hepatitis serology (Hepatitis A IgM, Hepatitis B sAg, Hepatitis C Ab, EBV IgM).
- Liver autoantibodies (Anti-smooth muscle/mitochondrial/LKS antibodies)
- Serum iron, transferrin, ferritin, ceruloplasmin, and α 1 anti trypsin levels

Consider:

- Tumor markers (including Ca19-9, α FP, CEA, PSA, Ca125)—if malignancy is suspected.
- Proportion of unconjugated/conjugated bilirubin—to differentiate 'pre-hepatic' from 'hepatic' and 'post-hepatic' jaundice. Unconjugated jaundice due to pre-hepatic causes is rarely greater than 100 μ mol/L. Deep jaundice is usually conjugated. Urine dipstick testing for urobilinogen is unreliable.
- Haemolysis screen (blood film, LDH, haptoglobin, reticulocyte count)
- Hepatitis E IgM—if a history of travel to endemic area or in pregnant women
- Thrombophilia screen (including antiphospholipid antibodies, Protein C, Protein S, and Antithrombin levels, Factor V Leiden genotype, JAK2 genotype)—if evidence of Budd-Chiari syndrome.
- Bile acid concentration in pregnancy—elevated cholic acid and chenodeoxycholic acid may be the only abnormalities in obstetric cholestasis. In pregnancy, the alkaline phosphatase is raised due to placental production, and thus is not useful for cholestasis.
- HIV test in high risk patients.

Questions commonly asked by examiners

Could you briefly outline the pathophysiology of jaundice?

Unconjugated bilirubin is formed from haemoglobin degradation in the reticuloendothelial system (spleen, liver, bone marrow). The unconjugated bilirubin is not soluble in water; and is transported to the liver bound to albumin so that it is not excreted in the urine. In the liver, it is conjugated to glucuronic acid within hepatocytes to form conjugated bilirubin which is water soluble. Conjugated bilirubin is excreted into the intestine in bile. Here, it is metabolized by colonic bacteria to urobilinogen, then to stercobilinogen, and finally stercobilin. The stercobilin gives faeces its brown colour. Some urobilinogen is reabsorbed and excreted in the urine as urobilin.

* The AST, ALT, γ GT, ALP are not true tests of liver 'function'—the degree of elevation does not correspond with severity of liver disease. The albumin and prothrombin time are better tests of liver 'function' (see Vol.1 Case 1 Chronic Liver Disease).

- Pre-hepatic jaundice is due to increased serum unconjugated bilirubin, and increased urine urobilinogen. The major cause of pre-hepatic jaundice is excess haemolysis.
- Hepatic (or hepatocellular) jaundice is due to liver dysfunction, and thus failure to conjugate bilirubin. The serum unconjugated bilirubin is elevated, but the conjugated bilirubin levels are also usually elevated due to intrahepatic cholestasis from hepatocellular necrosis. Therefore, hepatocellular jaundice usually has a mixed picture.
- Post-hepatic jaundice, due to obstruction of the bile ducts, presents with an elevation in conjugated bilirubin and decreased urine urobilinogen.

How does one diagnose and manage alcoholic hepatitis?

Alcoholic hepatitis is a severe illness, caused by acute liver inflammation due to alcohol. The classical presentation is with fever, jaundice, and elevated leucocyte count. Not all patients have underlying cirrhosis.

Blood tests are very characteristic for alcoholic hepatitis. The AST and ALT are elevated, but rarely greater than 400 IU/L. The AST may be elevated greater than the ALT, at a ratio of 2:1. The prothrombin time and creatinine may also be elevated. Liver biopsy may be necessary to exclude other causes, but usually will need to be done via transjugular liver access because coagulopathy and ascites are contraindications to percutaneous biopsy.

Severity of disease is assessed by scoring systems—the Maddrey discriminant function or Glasgow Alcoholic Hepatitis Score are most widely used.

$$\text{Maddrey's discriminant function} = 4.6 \times (\text{patients PT} - \text{control PT}) + \frac{\text{total bilirubin } (\mu\text{mol/L})}{17}$$

If Maddrey's discriminant function is greater than 32, this implies severe disease with a 50% mortality at 30 days. These patients may benefit from a trial of steroids or of pentoxifylline (an oral drug with weak anti-TNF α activity), although both these therapies remain controversial because of mixed trial results.

The remainder of the treatment is supportive, including nutrition and treatment of sepsis.

What is the differential diagnosis of liver abnormalities in pregnancy?

Liver diseases unrelated to pregnancy

Pregnant women are predisposed to some diseases which are not specific to pregnancy:

- Thrombotic disease (e.g. Budd-Chiari syndrome)
- Hepatitis E infection—pregnant women are more susceptible to liver failure (15–20%)

Liver diseases specific to pregnancy

- Obstetric cholestasis—occurs in the second and third trimesters, and is characterized by intractable pruritus and elevated bile acids. Symptoms tend to recur in subsequent pregnancies. Ultrasound of the bile ducts is typically normal. Ursodeoxycholic acid is safe, and may be of benefit.
- Acute fatty liver of pregnancy (AFLP)—occurs in the latter half of pregnancy, usually the third trimester. The disease is associated with pre-eclampsia in half of patients. The typical presentation is with nausea, vomiting, abdominal pain, and jaundice. Liver failure and complications such as DIC and encephalopathy may occur. Liver enzymes and bilirubin are markedly raised. Liver biopsy is diagnostic, showing fat infiltration, although may not be necessary if the diagnosis is clear clinically. Treatment is delivery of the baby, following supportive intensive care therapy.
- HELLP syndrome (Haemolysis, Elevated Liver enzymes, Low Platelets)—has significant overlap with AFLP. Symptoms develop in the third trimester, with abdominal pain, nausea,

and vomiting, and there is a history of pre-eclampsia in 80% of cases. Haemolysis is microangiopathic on blood smear along with other laboratory features of haemolysis, although the liver enzymes and bilirubin need not be markedly raised and liver failure is less common than AFLP. Treatment is delivery of the baby. Hypertension and convulsions are managed similarly to patients with pre-eclampsia.

Case 8 ♦ Abdominal Pain

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 39-year old lady who has a one-week history of increasingly severe abdominal pain. She tells me the pain is 'like a knife', and comes every day without any association with meals. She is a primary school teacher, and has been unable to work for the past week.

Many thanks for your opinion.

Acquiring the history

Acute abdominal pain may be a medical emergency, and in these cases the history should be taken after initial resuscitation and triage. The scenario in the history-taking section of the MRCP (PACES) is more likely to be a case of chronic abdominal pain, although the technique of goal-oriented history-taking whilst stabilizing a patient may be required in Station 2 of the examination.

The differential diagnosis of chronic abdominal pain is broad, and may be associated with many different physical and psychological symptoms. Therefore, in a PACES station where time is limited, the approach must be to identify the patient's agenda first, before screening for other symptoms.

A. History of presenting complaint:

Begin with open-ended questioning, 'I gather from your doctor you haven't been so well over the past few weeks. Can you tell me what's been happening?'

Let the patient describe their story without interruption. During the course of the discussion, ensure that the following characteristics of the abdominal pain are covered, but try to only use focused questions for clarification, or once the patient has finished voicing their concerns.

- Acute or subacute onset: 'Do you remember exactly when the pain started, or did it come on gradually?'
- Site: 'Where exactly does the pain come on? Can you show me?'
- Progression: 'Is the pain constant, or does it come and go in waves?'
- Nature of pain: 'What sort of pain is it—can you describe it?'; 'Is it more like a sharp stabbing pain, or a dull ache?'

- Radiation: 'Does the pain move anywhere from your abdomen?'; 'Does it go through to your back?'. Abdominal pain radiating to the back is worth asking specifically about, since it is associated with severe conditions such as pancreatitis and aortic aneurysm.
- Severity: 'Can you give me some idea of how bad the pain is? Is it the worst pain you have ever had? Is it worse now, or was it worse when it started?'
- Relationship to meals, movement, defecation: 'Is it better or worse when you eat/move/open your bowels?'
- Gastrointestinal symptoms: nausea, vomiting, reflux, bowel habit, weight loss
- Urinary symptoms: frequency, dysuria, haematuria, prostatic symptoms
- Gynaecological symptoms: menstrual and obstetric history, dyspareunia
- Other aggravating and relieving factors: 'Is there anything else that makes it better, or brings it on? How about painkillers—do they help? Which ones have you been taking?'

B. Patterns of presentation:

Abdominal pain, perhaps more than any other presentation, may present with a broad range of associated symptoms. The technique of active listening, along with clarification using the above focused questions, will hopefully cover the patient's agenda and yield most of the information required to form a differential diagnosis. However, since the differential is so broad, *pattern recognition* is an essential skill to interpret the patient's symptoms.

Abdominal pain is typically either *visceral* or *somatic*:

- *Visceral pain* is due to stretching of hollow organs, causing a dull, diffuse pain or 'ache'. The localization of visceral pain is imprecise, but in general pain from organs of the foregut (oesophagus, gallbladder, stomach, pancreas, first half of duodenum) is localized to the epigastric area. Pain from midgut organs (second half of duodenum, small intestine, ascending and part of transverse colon) is localized to the periumbilical area. Pain from hindgut organs (transverse colon to anus) is localized to the lower abdomen.
- *Somatic pain* is well-localized and sharp. It is due to inflammation of peritoneum covering abdominal organs.

The character and the site of the pain should lead you to consider the following patterns of presentation:

Right upper quadrant pain

1. Biliary pain is an example of visceral pain—due to postprandial contraction of the gallbladder or bile ducts onto a gallstone. The pain is usually *not* colicky, but is constant and often associated vomiting. Biliary pain may be due to:
 - (a) Biliary colic—usually lasts a few hours until the stone falls away back into the gall bladder.
 - (b) Cholecystitis—a similar type of pain, although progressive and associated with fever.
 - (c) cholangitis—biliary pain, fever, and jaundice (Charcot's triad of cholangitis is fever, jaundice and abdominal pain) (see Case 7 Jaundice).
 - (d) sphincter of Oddi dysfunction—a rare condition, which presents with biliary pain without evidence of biliary obstruction. The cause is sphincter of Oddi spasm, and the diagnosis is typically made post-cholecystectomy.

Left upper quadrant pain

2. Splenic pain is also an example of visceral pain. This may be due to splenic infarction or splenic artery aneurysm. Risk factors for splenic infarction are thromboembolism (atrial fibrillation (AF), prosthetic heart valves), haemoglobinopathy or myeloproliferative disorders.
3. Atypical pain from myocardial infarction or pneumonia may present with left upper quadrant pain. Ask about associated dyspnoea and radiation of pain to the jaw or arm.

Epigastric pain

4. The most common causes of epigastric pain are acid-related disorders (see Case 2 Dyspepsia). Ask about alarm symptoms, such as dysphagia, weight loss, early satiety, and progressive vomiting.
5. Pancreatic pain is typically 'piercing through to the back'. Patients may use hot water bottles to relieve the pain, or find that sitting forward in the 'pancreatic position' helps. Ask specifically about posture when asking about aggravating and relieving factors. The patient may also have weight loss, steatorrhoea, or diabetes mellitus. Ask about alcohol use and a history of gallstones as risk factors for chronic pancreatitis. Other causes of chronic pancreatitis are hereditary, autoimmune, and drug-induced. Ask about a family history of pancreas problems, a history of autoimmune disease such as systemic lupus erythematosus (SLE) or Sjögren's disease, or drugs such as antiretrovirals, azathioprine or loop and thiazide diuretics.

Lower abdominal pain

6. Irritable bowel syndrome characteristically presents with cramping, colonic pain affecting the left side of the abdomen, and altered bowel habit (see Case 4 Change in Bowel Habit). Colonic pain is visceral in character. The pain is often relieved by defaecation (see below).
7. Colon diverticular disease is usually asymptomatic, unless a complication such as diverticulitis occurs. This causes somatic pain, usually in the left side of the abdomen, with associated fever.

Right iliac fossa pain

8. Appendicitis usually presents with periumbilical, visceral pain (since the appendix is midgut in origin), which localizes to the right iliac fossa when the peritoneum becomes inflamed causing somatic pain.
9. Crohn's disease may cause pain anywhere in the abdomen, but the right iliac fossa is common since Crohn's typically affects the terminal ileum. The pain is visceral with associated diarrhoea and weight loss. Stricture formation may cause pain due to obstructive symptoms—ask about intermittent pain and abdominal distension. Crohn's disease may also cause abscesses, resulting in somatic pain and fever.

Pelvic pain

10. Consider gynaecological causes in all female patients. Document menstrual history, contraception and sexual history (see Case 17 Sexually transmitted infection). Ask about the relationship and timing of the pain to menstruation and sexual activity.
11. Renal colic typically presents with flank pain, although ureteric pain radiates to the groin and may resemble iliac fossa pain. (see Case 36 Haematuria). Gout, hyperparathyroidism, sarcoidosis, and Crohn's disease are risk factors for renal calculi.

Central abdominal pain

12. Central abdominal pain is of particular importance, since it may represent life threatening conditions such as mesenteric ischaemia, ruptured aortic aneurysm or pancreatitis. These should be considered in any case of severe central abdominal pain. Acute appendicitis may also present with acute periumbilical pain in the early stages.
13. Mesenteric ischaemia may cause chronic postprandial abdominal pain, leading to weight loss in 30–40% of cases. Midgut ischaemia is due to superior mesenteric artery disease, causing central abdominal pain. Coeliac artery disease typically causes epigastric pain due to foregut ischaemia. Inferior mesenteric artery disease is more likely to cause acute ischaemic colitis, possibly associated with embolic risk factors such as AF.

C. Relevant medical history:

- Ask about a history of gallstones, jaundice, and pancreatic disease.
- Haematological disease (e.g. sickle cell anaemia) may predispose to pigment gallstones and splenic infarction.

HISTORY-TAKING SKILLS

- Autoimmune disease is associated with autoimmune pancreatitis.
- Ensure to ask about previous gastroduodenal disease and surgery, and for upper GI and colonic alarm symptoms.

D. Medications and allergies:

Patients may have been using analgesics or antispasmodics for their pain. Take note of:

- Drugs which may cause gastric ulceration—NSAIDs, corticosteroids.
- Drugs which may cause gastro-oesophageal reflux—nitrates, calcium antagonists, theophyllines, bisphosphonates.
- Drugs which may cause pancreatitis—azathioprine, antiretroviral drugs, loop and thiazide diuretics?

E. Social issues:

- Ensure to take a thorough alcohol history (see Case 7 Jaundice for alcohol history).
- Determine the impact of the patient's symptoms on their occupation and daily activities.
- Since irritable bowel syndrome is associated with several psychological symptoms, ask empathetically and openly about the impact of their pain. *'It must be quite a burden to be in severe pain every day. How have you been feeling in yourself?'; 'Did the symptoms come on during a stressful time in your life?'; 'How about now—is there anything else that is worrying you, or that you would like to talk about?'*

Formulating a plan of action

- Explain to the patient that a full clinical examination is required. Investigations will include routine blood and urine tests, as well as a pregnancy test in female patients of child-bearing age.
- Emphasize that further tests may involve an ultrasound, CT scan, or endoscopy, but a follow-up appointment will be arranged first to discuss the results of the blood tests.
- Empirical treatment is unlikely to be helpful without a firm diagnosis, but a trial of proton pump inhibitor (e.g. omeprazole) or antispasmodic (e.g. colpermin) may be tried for an acid-related disorder or irritable bowel syndrome respectively.

Questions commonly asked by examiners

How does one diagnose irritable bowel syndrome?

Irritable bowel syndrome may be diagnosed using the Rome III criteria (Box 1), although if any alarm features are present then further colon investigation is necessary. Furthermore, the pain of irritable bowel syndrome is rarely progressive. Pain that is progressive, or wakes the patient from sleep, requires further investigation, usually with imaging. Many gastroenterologists recommend a baseline abdominal ultrasound in female patients over the age of 40 to exclude ovarian pathology.

Alarm symptoms for colon investigation

- Aged above 65
- Unintentional weight loss
- Change in bowel habit
- Iron-deficiency anaemia
- Rectal bleeding

- Frequent nocturnal symptoms
- Family history of colonic cancer

Box 1 Rome III criteria for the diagnosis of irritable bowel syndrome

The Rome III criteria (2006) for the diagnosis of irritable bowel syndrome require that patients must have recurrent abdominal pain or discomfort at least 3 days per month during the previous 3 months that is associated with two or more of the following:

- relieved by defecation
- onset associated with a change in stool frequency
- onset associated with a change in stool form or appearance

Supporting symptoms include the following:

- altered stool frequency
- altered stool form
- altered stool passage (straining and/or urgency)
- mucous with stool
- abdominal bloating or subjective distension

How would you approach abdominal pain in elderly patients or those with HIV?

Serious causes of abdominal pain must be considered in all elderly patients, with a low threshold for investigation. This is not only because malignancy, hepatobiliary disease, and bowel obstruction are more common in this group, but also because signs such as fever and peritonism are less frequently present.

Abdominal pain in HIV may be drug-related, such as didanosine-induced pancreatitis, or related to the underlying condition. Whilst common causes of abdominal pain may occur, these patients are also at risk of complications of HIV such as enteritis and peritonitis due to bacterial or opportunistic infections, and HIV-associated malignancies such as lymphoma and Kaposi's sarcoma.

Can you think of any other causes of abdominal pain that need to be considered if initial investigations are negative?

1. Vasculitis, particularly polyarteritis nodosa, may cause mesenteric ischaemia and recurrent abdominal pain. A proportion of these patients will present with acute abdominal pain and intestinal perforation. There is no specific test for polyarteritis nodosa, although autoimmune assays such as anti-neutrophil cytoplasmic antibody (ANCA) may rarely be positive. GI lesions may be visible during endoscopy, although they rarely provide histological confirmation of vasculitis, therefore a full thickness intestinal biopsy (not a superficial endoscopic biopsy) may be required.
2. Familial Mediterranean fever presents with acute attacks of abdominal pain, serositis, and fever. Patients may have a family history and the diagnosis can be confirmed by genetic testing.
3. Acute intermittent porphyria may cause acute abdominal pain, and may have associated psychiatric symptoms.

- Adrenal insufficiency may present with non-specific nausea and abdominal pain, and these symptoms often correlate with the severity of adrenal insufficiency.

Case 9 ♦ Chest Pain

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 53-year old gentleman who has recently started to complain of chest pain. He has had chest pain since returning from holiday in Turkey, and he is concerned that he may have a pulmonary embolus since his mother has suffered from recurrent pulmonary emboli. He has a history of hypertension, although has no other medical history. He is a smoker. A sublingual glyceryl trinitrate (GTN) spray has been helpful on some occasions. A 12-lead ECG taken at our clinic has been unremarkable. I would be grateful for your assessment.

Acquiring the history

Chest pain is a common presenting symptom for acute medical admissions. The differential diagnosis is broad, and it is the history that plays a key role in determining subsequent investigations and management. When taking a history from a patient with chest pain, it is important to be aware of the broad differential diagnosis list and preliminary questioning will appropriately narrow this differential diagnosis. Subsequently, specific targeted questions will lead to the diagnosis.

A. History of presenting complaint:

This is the most important part of the history that will determine the aetiology of chest pain. Initially the questions should be broad, followed by focused questions to help narrow the differential diagnosis.

Characteristic of chest pain

- **Site**
 - ♦ **Cardiac ischaemic pain:** central, and may radiate to the jaw, neck, and left arm
 - ♦ **Respiratory chest pain:** localizes to the site of pathology, i.e. infection or pneumothorax. However, patient with asthma or obstructive airways disease may complain of central chest pain or tightness, but other features in the subsequent history will help differentiate this from cardiac ischaemia.
 - ♦ **Musculoskeletal chest pain:** localizes to site of pathology or injury or may relate to the posterior chest in the region of the spine.
 - ♦ **Peptic ulcer disease and gastro-oesophageal reflux:** lower chest and epigastrium.
- **Nature**
 - ♦ **Cardiac ischaemic chest pain:** dull pressure like sensation that is not pleuritic.
 - ♦ **Respiratory and musculoskeletal:** sharp and pleuritic

- ♦ **Pericarditis:** sharp and pleuritic.
- ♦ **Nerve root pain:** band like shooting pain around the chest from the back to the anterior chest
- ♦ **Gastro-oesophageal reflux:** sharp and burning-like sensation
- **Radiation**
 - ♦ **Cardiac ischaemic pain:** jaw, neck, and arms (often the left arm)
 - ♦ **Aortic dissection, peptic ulcer disease, and pancreatitis:** back
 - ♦ **Nerve root pain:** around the chest wall resulting in a band-like pain sensation
- **Onset, duration and frequency**
 - ♦ *'When did the chest pain start?'* It is important to establish duration of symptoms. In the given scenario, it is important to relate the onset of symptoms in relation to the recent holiday. It is equally important to establish if there were any symptoms prior to or during the holiday. It is often the case that symptoms may have been present for sometime before seeking medical advice and opinion.
 - ♦ *'Does the pain come on suddenly or gradually?'*
 - ♦ *'Is it constant or intermittent?'*
 - ♦ If intermittent, *'How frequently do you experience chest pain?'*

Precipitating factors

- **Exertion:** cardiac ischaemia
- **Deep inspiration:** respiratory, musculoskeletal, and pericarditis
- **Movement:** musculoskeletal
- **Eating:** peptic ulcer disease, gastro-oesophageal reflux
- **Position:** pericarditis, pancreatitis, and gastro-oesophageal reflux (worse lying down); bending forward can exacerbate gastro-oesophageal reflux

Relieving factors

- **Rest:** Cardiac ischaemic pain
- **Sublingual nitrates:** cardiac ischaemic pain and oesophageal spasm
- **Antacid preparation:** peptic ulcer disease and gastro-oesophageal reflux
- **Simple analgesics:** musculoskeletal pain, respiratory pain, and pericarditis
- **Bronchodilators:** asthma and obstructive airways disease

Associated features

- **Cardiac ischaemic chest pain:** nausea, vomiting, sweating, pallor, breathlessness (may be a manifestation of ischaemia or left ventricular dysfunction), palpitations and dizziness (arrhythmias) and symptoms of left ventricular dysfunction (orthopnoea, paroxysmal nocturnal dyspnoea, ankle oedema, and reduced exercise tolerance)-*'Do you ever wake up at night feeling breathless?'* (paroxysmal nocturnal dyspnoea), *'How many pillows do you sleep on at night?'* (orthopnoea)
- **Respiratory tract infection:** cough (productive or non-productive), sputum (colour and consistency), fever, and haemoptysis
- **Pulmonary embolism (PE):** non-productive cough, haemoptysis, low-grade fever, calf pain, and swelling (indicating underlying deep vein thrombosis)
- **Lung malignancy:** productive cough, haemoptysis, weight loss, and loss of appetite
- **Pericarditis:** cold and flu-like symptoms

HISTORY-TAKING SKILLS

- **Peptic ulcer disease:** nausea, vomiting, haematemesis, and melaena
- **Gastro-oesophageal reflux:** acid taste in mouth, dysphagia (oesophageal strictures)
- **Musculoskeletal:** back and joint pains
- **Nerve root pain:** symptoms of underlying occult malignancy (weight loss, loss of appetite, change in bowel habit, noticeable masses or lumps) would indicate underlying metastatic disease; neurological symptoms in lower limbs, urinary and faecal incontinence (spinal cord involvement)

Other factors

- Enquire about any recent history of chest trauma.

B. Relevant past medical and family history:

Past medical history

- **Cardiovascular risk factors** (smoking, hypertension, hypercholesterolaemia, diabetes, family history, previous history of myocardial infarction)
- If risk factors are present, enquire about risk factor control and compliance to therapy. *'Do you remember the last blood pressure measurement?'* *'Have you had your cholesterol level checked?'* If so, *'Do you remember what it was?'*
- If previous cardiac history, then enquire about previous myocardial infarction, coronary angiography, coronary angioplasty, or coronary artery bypass graft (CABG) surgery
- **Menopause**—enquire about menstrual cycle in females, as the risk of ischaemic heart disease is increased in the post-menopausal period
- **Thrombotic risk factors** (previous history of thromboembolic disease, thrombophilia, malignancy, immobility, recent surgery or long-haul flight, and oral contraceptive pill use [females])
- **Peptic ulcer disease**—enquire about previous endoscopy
- **Asthma/COPD**
- **Pneumothorax**—recurrence of pneumothorax is common (15–40%) and up to 15% of recurrences can be on the contralateral side

Family history

- **Ischaemic heart disease**—enquire about the age at first presentation (establish risk of premature coronary artery disease)
- **Thrombo-embolic disease**—enquire about thrombophilia

C. Medications:

- Enquire about full drug history and compliance to therapy
- Enquire about any use of medications (including over-the-counter drugs) to alleviate symptoms (sublingual nitrates, simple analgesics, antacids, and bronchodilators)
- Enquire about side-effects of therapy, i.e. cough (ACE inhibitors), headaches (nitrates) and myalgia (statins)
- Oral contraceptive pill use in females increases thromboembolic risk

D. Social issues:

- Smoking habits (cardiovascular risk factor)
- Alcohol consumption (risk factor for peptic ulcer disease and pancreatitis)
- Enquire about job. Heavy lifting and manual labour may contribute to musculoskeletal chest pain and aggravate exertional cardiac ischaemia

- Does the patient drive? A diagnosis of myocardial infarction will have an impact on driving restrictions
- Impact of symptoms on daily life
- Marital status and sexual history. Sexual intercourse may potentiate cardiac ischaemia, and discussion related to this forms an important part of cardiac rehabilitation
- Elicit the patient's concerns about the symptoms

Formulating a plan of action

Explain to the patient that there are possible causes for chest pain and this will warrant further investigation. Given the above scenario the possible causes for chest pain are cardiac ischaemia or PE. In this case it would be appropriate to initially exclude PE. Once this is excluded, only then will it be appropriate to investigate further for myocardial ischaemia.

- **Bloods**
 - **Anaemia** can be seen in peptic ulcer disease with GI blood loss or in any chronic disease, including malignancy (anaemia can potentiate cardiac ischaemia)
 - **Leucocytosis** and **raised inflammatory markers** would suggest underlying infection
 - **Elevated D-dimer** for suspected PE
 - **Cardiac enzymes** if suspecting myocardial ischaemia.
- **ECG**
 - **Myocardial ischaemia:** the ECG can be normal at rest in a patient with exertional angina
 - **PE:** sinus tachycardia is the most common finding, often combined with non-specific ST segment and T-wave changes
- **Chest radiograph**
 - To exclude other causes of chest pain, e.g. pneumothorax, collapse, consolidation, and malignancy.

The following specific tests may be indicated:

- **Doppler USS of the leg** for suspected deep vein thrombosis
- **CT pulmonary angiogram** for suspected PE
- **Exercise ECG** to look for inducible cardiac ischaemia
- **Dobutamine stress echocardiography** to look for inducible ischaemia (if patient unable to perform exercise ECG)
- **Coronary angiography** if there is evidence of inducible ischaemia on exercise ECG or dobutamine stress echocardiography
- **Endoscopy** if suspecting peptic ulcer disease

Questions commonly asked by examiners

What are the causes of pleuritic chest pain?

- PE
- Pneumonia
- Malignancy
- Pleurisy
- Pneumothorax
- Musculoskeletal chest pain
- Pericarditis

What do you understand by the term acute coronary syndrome?

Acute coronary syndromes include unstable angina (USA), non-ST segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI) (see Figure 9.1¹). Acute coronary syndromes with persistent ST-elevation (STEMI) generally require urgent reperfusion therapy with thrombolysis or primary percutaneous coronary intervention. Those without persistent ST-elevation represent a continuum from USA to NSTEMI and can be further classified on the basis of troponin release, a biochemical marker of myocardial cell death. This varies between different laboratories and troponin assays used. Troponin levels should be measured after 12 hours from the onset of chest pain.

What is the initial management of acute coronary syndrome?

- Oxygen
- Aspirin 300mg PO
- Clopidogrel 300mg PO
- Sublingual nitrates
- Analgesia (morphine with metoclopramide)

STEMI

- primary percutaneous coronary intervention or thrombolysis*
- beta blocker (contraindications: bradycardia, heart block, hypotension, pulmonary oedema)
- ACE inhibitors
- Statin

* Patients receiving thrombolysis with recombinant tissue plasminogen activators (rt-PA) require concurrent unfractionated heparin. Recent evidence suggests that low molecular weight heparin can be used in this setting.¹

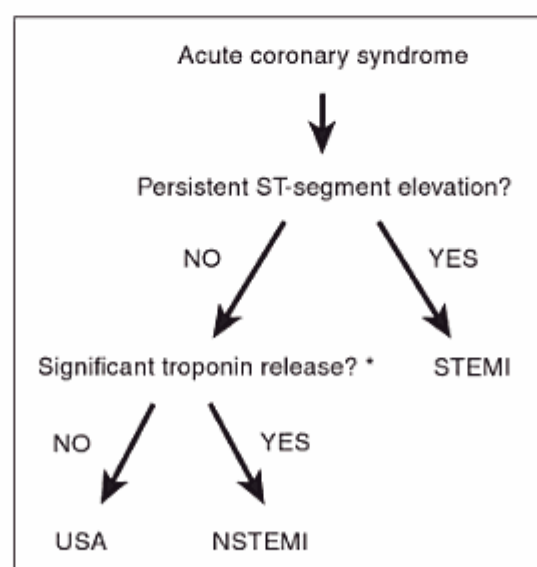


Figure 9.1

MB Iqbal, MA Westwood, and RH Swanton. Recent development in acute coronary syndromes. Clin Med. 2008; 8:42–8.

NSTEMI

- Low molecular weight heparin
- beta blocker (contraindications: bradycardia, heart block, hypotension, pulmonary oedema)
- Glycoprotein IIb/IIIa inhibitors in high risk patients*
- ACE inhibitors
- Statin

What do you know about the TIMI risk score?

This is a risk stratification tool in patients with acute coronary syndrome. A TIMI score ≥ 3 indicates high risk.² A score of 1 is assigned for the following 7 factors:

1. **age ≥ 75 years**
2. **≥ 3 risk factors for coronary artery disease**
3. **50% coronary stenosis**
4. **ST segment deviation**
5. **≥ 2 anginal episodes in 24 hours**
6. **positive troponin**
7. **aspirin use in the last 7 days**

What is the role of an early invasive strategy in patients with USA or NSTEMI?

Recent research has compared clinical outcomes associated with early invasive strategy versus an early conservative therapy in patients with USA or NSTEMI. An early conservative strategy involves aggressive medical therapy, with coronary angiography and revascularization reserved only for patients with recurrent or inducible ischaemia. With this early invasive strategy, all patients undergo early coronary angiography within 12–48 hours of presentation, and revascularization if indicated. Current evidence suggests that a routine early invasive strategy is associated with better long-term outcomes, particularly in high-risk patients. In the TACTICS-TIMI 18 trial,³ the benefits of an early invasive strategy were only observed in high-risk patients, defined as those with troponin elevation, ST segment deviation or TIMI risk score ≥ 3 . Pooled data from 7 trials has shown that although a routine invasive strategy is associated with higher early mortality during initial hospitalization, it is associated with better *long-term* outcomes with a significant reduction in death, myocardial infarction, recurrent angina, and re-hospitalization.⁴

What do you know about the DVLA guidelines for restrictions on driving following a myocardial infarction?

It is important that patients and doctors are aware of the restrictions on driving following myocardial infarction. Different criteria must be satisfied depending on the type of vehicle driven:

- GROUP 1: cars and motorcycles
- GROUP 2: lorries, vans, ambulances, and public transport (including taxi)

The DVLA guidelines for all cardiovascular conditions are summarized in the table.

* Patients at high-risk include those with ongoing chest pain, haemodynamic instability, dynamic ST/T wave changes on the ECG and an elevated troponin. Another tool to identify high-risk is a TIMI score ≥ 3 .

HISTORY-TAKING SKILLS

	GROUP 1	GROUP 2
Angina	Cannot drive if symptoms at rest. Can recommence when symptoms are controlled.	Can drive when free from anginal symptoms for more than 6 weeks and obtains satisfactory exercise ECG
STEMI	1 month (1 week if percutaneous coronary intervention (PCI))	(completing stage 3 of the BRUCE protocol) off anti-anginal therapy (≥ 48 hours).
NSTEMI	1 month (1 week if PCI)	
CABG	1 month	6 weeks
Following pacemaker insertion	1 week	Cannot drive
Following ICD insertion (primary prevention)	1 month	
Following ICD insertion (secondary prevention)	Can drive after a 6-month shock-free period. Following alteration of anti-arrhythmic medications, cannot drive for 1 month	
Arrhythmia	Cannot drive if arrhythmia causes incapacity. Can recommence when cause identified and controlled for 1 month.	Cannot drive if arrhythmia causes incapacity. Can recommence when cause identified and controlled for 3 months.
Unexplained syncope	Cannot drive if arrhythmia causes incapacity. Can recommence when cause identified and controlled for 1 month. If no cause identified, then can recommence only if free of symptoms for 6 months	Can drive 3 months after if the cause has been identified and treated. If no cause identified, then licence refused/revoked for 1 year.
Simple faint (definite provocational factors, associated prodromal symptoms and unlikely to occur at rest)	No restrictions	No restrictions

References

1. Antman EM, Morrow DA, McCabe CH, Murphy SA, Ruda M, Sadowski Z et al; ExTRACT-TIMI 25 Investigators. Enoxaparin versus unfractionated heparin with fibrinolysis for ST-elevation myocardial infarction. *N Engl J Med*. 2006; 354:1477–88.
2. Iqbal MB, Westwood MA, Swanton RH. Recent developments in acute coronary syndromes. *Clin Med*. 2008; 8:42–48.
3. Cannon CP, Weintraub WS, Demopoulos LA, Vicari R, Frey MJ, Lakkis N et al; TACTICS (Treat Angina with Aggrastat and Determine Cost of Therapy with an Invasive or Conservative Strategy)-Thrombolysis in Myocardial Infarction 18 Investigators. Comparison of early invasive and conservative strategies in patients with unstable coronary syndromes treated with the glycoprotein IIb/IIIa inhibitor tirofiban. *N Engl J Med*. 2001; 344:1879–87.
4. Mehta SR, Cannon CP, Fox KA, Wallentin L, Boden WE, Spacek R et al. Routine vs selective invasive strategies in patients with acute coronary syndromes: a collaborative meta-analysis of randomized trials. *JAMA*. 2005; 293:2908–17.

Case 10 ♦ Breathlessness

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 73-year old gentleman who gives a 3-month history of worsening breathlessness on exertion. He previously had unrestricted exercise tolerance, but now becomes extremely breathless on walking 30 yards. He has a history of hypertension, hypercholesterolaemia, diabetes, and previous coronary artery bypass surgery. He has since been free of anginal symptoms.

Many thanks for your opinion.

Acquiring a history

A. History of presenting complaint:

- **Onset, duration and frequency**
 - ♦ It is important to establish the duration of breathlessness
 - ♦ Ask about the breathing prior to this, to establish baseline functional status: 'What was your breathing like 6 months ago?'
 - ♦ 'Does the breathlessness come on suddenly or gradually?'
 - ♦ 'Is the breathlessness constant or intermittent?'
 - ♦ If intermittent, 'How frequently do you experience breathlessness?'
- **Precipitating factors**
 - ♦ Ask about any factors that precipitate or worsen breathlessness?
 - ♦ 'Is it exertional?' 'Is it present at rest?'
 - ♦ 'Does lying down make it worse?' (orthopnoea)
- **Relieving factors**
 - ♦ 'Is it relieved with rest?'
 - ♦ Enquire about any medications, e.g. sublingual nitrates or inhalers that may have relieved symptoms
- **Associated symptoms**
 - ♦ **Chest pain** suggests underlying cardiac ischaemia, but may rarely be a feature of asthma or COPD. If chest pain is present, it is important to differentiate cardiac ischaemic chest pain from other causes (see Case 9 Chest Pain).
 - ♦ **Palpitations** suggest the presence of underlying arrhythmia (which can be exertional), particular AF/flutter or ventricular arrhythmias. If palpitations are present, then further questioning will be necessary to characterize the arrhythmia (see Case 11 Palpitations) 'Have you measured your pulse during these episodes of palpitations?' 'Are they regular or irregular?' alternatively, patients may complain of a slow heart rate and bradycardia can manifest as exertional breathlessness
 - ♦ **Pre-syncope or syncope** can be associated with arrhythmia or underlying left ventricular outflow obstruction, i.e. aortic stenosis or hypertrophic obstructive cardiomyopathy
 - ♦ **Orthopnoea** indicates left ventricular dysfunction. It is important to note that a history of orthopnoea can often be elicited in patients with COPD with good left

ventricular function. This is primarily because of a hyper-inflated chest, and patients rely predominantly on vertical chest expansions and diaphragmatic excursion for breathing

- **Paroxysmal nocturnal dyspnoea** indicates left ventricular failure. *'Do you ever wake up at night feeling breathless?'*
- **Oedema** indicates congestive cardiac failure. Cor pulmonale can be seen in patients with good left ventricular function, but with pulmonary hypertension due to advanced lung disease, i.e. COPD or interstitial lung disease. Enquire about sites of fluid retention, i.e. abdominal distension (ascites) and scrotal oedema in males. If there is a history of leg swelling, enquire about asymmetry—asymmetrical leg swelling may suggest deep vein thrombosis thereby implicating PE (thromboembolic disease) as a cause of breathlessness
- **Respiratory symptoms**, i.e. cough, sputum, wheeze, and haemoptysis suggest underlying respiratory disease. A dry non-productive cough may indicate interstitial lung disease. A productive cough may indicate infection, suppurative lung disease or malignancy. Haemoptysis can occur in PE, infection, and malignancy. Haemoptysis can also be seen in patients with pulmonary congestion, especially with mitral stenosis. Wheeze signifies asthma or COPD
- **Thyroid disease**. Hyperthyroidism can cause breathlessness. Enquire about weight loss, heat intolerance, tremor, sweating
- **Neurological symptoms**. Neuromuscular weakness can result in breathlessness: Guillain-Barre Syndrome (recent respiratory or diarrhoeal illness, progressive ascending weakness and ptosis) and myasthenia gravis (fatiguability, diplopia, ptosis). Fatiguability is characterized by symptoms being worse at the end of the day
- **Vasculitic symptoms** (fever, joint aches, muscle aches, and rashes) would suggest pulmonary vasculitis
- **Renal disease**. Renal failure can lead to breathlessness as a result of fluid retention and/or metabolic acidosis. Enquire about uraemic symptoms, particularly restlessness and pruritus. Ask about urinary symptoms, particularly any noticeable reduction in urine output
- **Exercise tolerance**
 - It is important to quantify reduction in exercise tolerance.
 - Establish pre-morbid, and then current functional status and exercise tolerance.
 - This can be used to establish New York Heart Association (NYHA) functional class: I (unrestricted), II (breathless on heavy exertion), III (breathless on mild exertion), and IV (breathless at rest).
- Other factors
 - Enquire about any recent history of chest trauma (pneumothorax).

B. Relevant previous medical and family history:

Medical history

- **Cardiac disease**
 - Enquire about cardiovascular risk factors (smoking, hypertension, hypercholesterolaemia, diabetes, family history, previous history of myocardial infarction)
 - If risk factors are present, enquire about risk factor control and compliance to therapy. *'Do you remember the last blood pressure measurement?'* *'Have you had your cholesterol level checked?'* If so, *'Do you remember what it was?'*
 - If previous cardiac history, then enquire about previous myocardial infarction, coronary angiography, coronary angioplasty, or CABG surgery.

- Enquire about pre-existing left ventricular dysfunction or previous echocardiography. *'Have you ever had an ultrasound scan of the heart' If so, 'What did it show?'*
- Enquire about any history of valvular heart disease. *'Have you ever been told that you have heart murmur?' 'Is there a history of rheumatic fever?'*
- **Respiratory disease**
 - History of chronic lung diseases, i.e. asthma, COPD, bronchiectasis, interstitial lung disease, sarcoidosis (ask about fever, joint aches, and painful nodules on the legs)
 - Thrombotic risk factors (previous history of thromboembolic disease, thrombophilia, malignancy, immobility, recent surgery or long-haul flight, and oral contraceptive pill use).
- **Thyroid disease**
- **Renal disease**

Family history

- **Ischaemic heart disease**—enquire about the age at first presentation (establish risk of premature coronary artery disease)
- **Cardiomyopathy**—familial dilated or hypertrophic cardiomyopathy
- **Thrombo-embolic disease**—enquire about thrombophilia
- **Primary pulmonary hypertension**

C. Medications:

- Enquire full drug history and compliance to therapy
- Oral contraceptive pill use in females increases thromboembolic risk
- Enquire about any use of medications (including over-the-counter drugs) to alleviate symptoms (sublingual nitrates and bronchodilators)
- Appetite suppressors (e.g. fenfluramine) are associated with pulmonary hypertension

D. Social issues:

- Smoking habits (cardiovascular risk factor)
- Alcohol consumption (risk factor for dilated cardiomyopathy)
- Enquire about job. Heavy lifting and manual labour may aggravate breathlessness.
- Impact of symptoms on daily life
- Elicit the patient's concerns about the symptoms

Formulating a plan of action

Explain to the patient that there are possible causes for exertional breathlessness which are primarily cardiac or respiratory and this will warrant further investigation.

- Pulse oximetry
- Bloods
 - **Anaemia** can be potentiate cardiac ischaemia and cardiac failure
 - **Leucocytosis** and **raised inflammatory markers** would suggest underlying infection
 - **D-dimer** for suspected PE
 - **Cardiac enzymes** if suspecting myocardial ischaemia
 - **BNP** for heart failure
- ECG
 - **Myocardial ischaemia**: the ECG can be normal at rest in a patient with exertional angina
 - **Arrhythmia**: AF/flutter or ventricular arrhythmias can be easily detected

- **PE:** sinus tachycardia is the most common finding, often together with non-specific ST and T-wave changes
- Chest radiograph
 - Cardiomegaly and pulmonary congestion (left ventricular dysfunction)
 - To exclude other causes of chest pain, e.g. pneumothorax, collapse, consolidation, and malignancy.

The following specific tests may be indicated:

- **Echocardiogram:** to assess ventricular function, valvular heart disease, pulmonary artery systolic pressure and to detect shunts or a pericardial effusion
- **Ambulatory ECG monitoring:** to detect paroxysmal arrhythmia
- **Exercise ECG** to look for inducible cardiac ischaemia
- **Dobutamine stress echocardiography** to look for inducible ischaemia (if patient unable to perform exercise ECG)
- **Coronary angiography** if there is evidence of inducible ischaemia on exercise ECG or dobutamine stress echocardiography.
- **CT pulmonary angiogram** for suspected PE
- **Lung function tests:** obstructive or restrictive lung defect if suspecting respiratory disease
- **CT chest** if suspecting interstitial lung disease or lung malignancy
- **Arterial blood gas** to confirm hypoxia and/or metabolic acidosis

Questions commonly asked by examiners

What is the role of a d-dimer measurement in patients with suspected thromboembolic disease?

The d-dimer may be elevated in infection or with underlying malignancy. However, a negative d-dimer reliably excludes PE if there is a low pretest probability. However, a low d-dimer doesn't exclude PE if the pretest probability is moderate or high.

When should you consider a ventilation-perfusion (V-Q) scan as a first-line investigation for PE?

V-Q scan should only be considered as a first-line investigation for PE, if the chest radiograph is normal and there is no significant cardiopulmonary disease. In this setting, a normal V-Q scan reliably excludes PE, but an intermediate scan should be followed up by CT pulmonary angiography (which remains the gold standard).

What do you know about BNP?

This is one of the four known natriuretic peptides (ANP, BNP, CNP, and DNP) that is released from ventricles (left ventricle > right ventricle) during volume or pressure overload. Its physiological effects include dilatation of the arteries and veins, diuresis, and natriuresis. Normal levels are less than 100pg/mL. It has a negative predictive value of at least 96%, so heart failure can be confidently ruled out for patients in the normal range. The positive predictive value of BNP for diagnosing heart failure is 90%, as an elevated BNP value can be seen with renal failure, pulmonary hypertension, and PE.

What is the management of left ventricular systolic dysfunction?

Diuretics are useful in relieving congestive symptoms, especially in acute decompensated stages of heart failure. All patients with left ventricular systolic dysfunction should be started on ACE inhibitors provided there are no contraindications, irrespective of functional status. If they are not tolerated, then angiotensin II receptor blockers can be used. A rise in creatinine up to 20% can be expected after starting an ACE inhibitor or angiotensin receptor blocker. If the creatinine level

rises by > 20%, the ACE inhibitor should be discontinued and underlying renovascular disease should be considered. The presence of hypotension is not an absolute contraindication to starting ACE inhibitors. Patients often have low blood pressure readings in the context of a low cardiac output. Therapy may be initiated in patients with systolic blood pressures as low as 90mmHg, provided they do not become symptomatic as a result of hypotension. This also applies to other pharmacotherapeutic agents in heart failure.

Beta blockers should not be commenced in the acute stages of heart failure. Once considered a contraindication, there is now overwhelming data to support the use of β -blockers in stable heart failure. They should be initiated with NYHA class I–II status. However, they may be used cautiously in patients with NYHA class III–IV status. Beta blockers that are licensed for use in heart failure include bisoprolol, carvedilol, metoprolol, and nebivolol. Current guidelines suggest that all patients with stable heart failure should initially be started on an ACE inhibitor, followed by initiation of β -blockers therapy.

Spironolactone should be used for patients in NYHA class III–IV status. It is important to monitor the renal function and for hyperkalaemia (given that such patients may also be on ACE inhibitors or angiotensin II receptor blockers).

Digoxin should be used as a first-line treatment with ACE inhibitors in all patients with heart failure and AF. For patients in NYHA class III–IV status in sinus rhythm, digoxin has been shown to improve symptoms and reduce hospitalization, but without a reduction in mortality.

For patients with NYHA III–IV status, who are on maximal medical therapy (ACE inhibitor, β -blockers, spironolactone, and digoxin) with evidence of interventricular conduction delay (LBBB; QRS > 120mm), these patients may be considered for cardiac resynchronization therapy (biventricular pacemaker).

Further Reading

1. National Institute for Health and Clinical Excellence. Management of chronic heart failure. London: NICE; 2003.
2. National Institute for Health and Clinical Excellence. Cardiac resynchronization therapy for the treatment of heart failure. London: NICE; 2007.

Case 11 ♦ Palpitations

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 43-year-old female who complains of intermittent palpitations associated with light-headedness. She has no significant medical history and takes no regular medications. She works as a financial analyst and has an extremely stressful job. She has a large caffeine intake, which can range from 8 to 10 mugs of coffee per day. There is no family history of ischaemic heart disease, but her mother has a long-standing diagnosis of atrial fibrillation, and suffered a stroke as a complication. She is extremely anxious and concerned that she may have developed AF. A resting 12-lead ECG at the surgery showed normal sinus rhythm.

Thank you for your help.

Acquiring the history

Although the cause of palpitations is usually benign, they may occasionally herald an underlying life-threatening cardiac disorder. Since the differential diagnosis is broad, the focus of the history is to distinguish the minority of patients who require extensive diagnostic testing and management.

A. History of presenting complaint:

Age of onset

Consider supraventricular tachycardias (SVTs) in young patients presenting with a history of palpitations in childhood or adolescence. AF is the commonest arrhythmia occurring in patients over 65 years. ventricular tachycardia (VT) also occurs more commonly with age because of the higher incidence of structural heart disease, although congenital long QT syndromes may present in childhood and adolescence.

Character of the palpitations

- **Nature of palpitations.** Various terminologies are used to describe palpitations, although these descriptors are generally non-specific. Ask the patient an open question about the sensation they experience: *'What do the palpitations feel like?'*
 - ♦ *'Racing or tapping heart'* suggesting a fast arrhythmia.
 - ♦ *'Missed or extra beats'* followed by a *'Pounding or more forceful beat'* are indicative of ventricular ectopic beats.
 - ♦ *'Pounding in the neck'* or *'Everything stopped for a moment'* may suggest atrioventricular dissociation or bradycardia. The pounding feeling in the neck is due to cannon A waves which suggest atrial contraction against closed atrioventricular valves. Rapid and regular pounding in the neck is most typical of re-entrant supraventricular arrhythmias, particularly atrioventricular nodal re-entry tachycardia (AVNRT).
- **Rate and rhythm**
 - ♦ *'Are the palpitations fast or slow? Do you take your pulse at the time, if so what was it?'*
 - ♦ *'Are the palpitations regular?'* Some patients may be able to tap out the rhythm they experience during episodes, although *do not insist on this* since it is not always a useful exercise. Rapid, regular palpitations favour sinus tachycardia, SVT, and VT. Irregular palpitations suggest AF and extrasystoles.
- **Time course, onset and frequency of palpitations**
 - ♦ *'How long have you had the palpitations?'* Palpitations occurring since childhood strongly favour SVT.
 - ♦ *'How often do you experience the palpitations?'*
 - ♦ *'Do the palpitations occur gradually or suddenly?'* Sinus tachycardia is more likely to have a gradual onset and termination
 - ♦ *'How long do the episodes last?'*
- **Precipitants**
 - ♦ *'Is there anything that triggers the episodes—such as exercise, stress, alcohol or coffee?'* These can aggravate any type of arrhythmia. Exercise can precipitate VT and polymorphic VT in long QT syndrome. The latter can also be triggered by emotion and sudden arousal.
 - ♦ *Ask specifically about cocaine and amphetamine use.*
 - ♦ Recent illness can precipitate AF; ask about these during systems review.
- **Termination.** *'How do the palpitations stop—have you tried stopping them by straining or holding your breath?'* SVTs can be terminated by Valsalva manoeuvres. Ectopic beats terminate with exercise.

Associated symptoms

- Dyspnoea occurs with most arrhythmias.
- Syncope and presyncope are signs of haemodynamic compromise necessitating intervention.
- Chest pain, nausea, and sweating are signs of myocardial ischaemia associated with a tachyarrhythmia, which also requires urgent treatment.
- Exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, ankle swelling, and reduced exercise tolerance indicate cardiac failure. Determine whether these symptoms have worsened following the onset of palpitations, since tachyarrhythmias and bradyarrhythmia may aggravate cardiac failure.

Relevant medical and family history:**Past medical history**

- **Thyrotoxicosis**—weight loss, heat intolerance, tremor, sweating.
- **Hypothyroidism**—weight gain, cold intolerance, fatigue, constipation.
- **Hypoglycaemia**—hunger, headache, irritability, confusion, and sweating.
- **Phaeochromocytoma**—headaches, flushing, tremor, and excessive sweating.
- **Cardiac disease**—myocardial infarction, angina, known arrhythmias, valvular disease, and other structural cardiac disease.
- **Cerebrovascular disease** is a complication of inadequately treated AF.
- **Vascular risk factors**—hypertension, hyperlipidaemia, and diabetes mellitus.
- **Psychiatric illness** such as anxiety disorders.
- **Asthma**—treatment with β agonists can precipitate sinus tachycardia.

Family history

A family history of cardiac disease and sudden death is particularly relevant in younger patients presenting with palpitations. Hypertrophic cardiomyopathy and long QT syndrome are inherited conditions that can present with palpitations, syncope, and sudden death due to VT.

C. Medications:

Take a thorough drug history enquiring about medications that can precipitate palpitations. These are:

- β agonists
- Theophylline
- Levothyroxine
- Sympathomimetics such as monoamine oxidase inhibitors
- Drugs that prolong the QT segment may precipitate polymorphic VT
 - Antiarrhythmic drugs—quinidine, disopyramide, amiodarone, sotalol
 - Macrolide antibiotics—erythromycin, clarithromycin, azithromycin
 - Antihistamines—terfenadine
 - Psychotropic drugs—phenothiazines, butyrophenones, SSRIs, and tricyclic antidepressants.
 - GI motility agents—cisapride, domperidone
- Recreational drugs such as amphetamines and cocaine increase sympathetic drive causing palpitations.

D. Social issues:

- Ask specifically about alcohol and caffeine consumption.
- Ask about smoking habits.

- Determine the impact of the symptoms on daily activities.
- Does the patient drive?
- Elicit the patient's concerns about their symptoms.

Formulating a plan of action

- Explain to the patient that a clinical examination and ECG will be required in order to help determine the cause of the palpitations.
- Reassure the patient that the cause is likely to be benign, and further tests may not be required if the examination and ECG are normal. However, further investigation may be warranted to rule out structural heart disease, identify a possibly treatable arrhythmia, or to reassure the patient.
- Consider the following investigations:
 - ♦ **Blood count** may indicate an anaemia causing a sinus tachycardia. An infection precipitating or aggravating atrial arrhythmias is suggested by a leucocytosis or elevated CRP.
 - ♦ **Urea, creatinine, electrolytes** (including potassium, calcium, magnesium, and phosphate)—any electrolyte disturbances can precipitate arrhythmias either directly or by prolongation of the QT interval.
 - ♦ **Thyroid function tests** will indicate hypo/hyperthyroidism.
 - ♦ **Cardiac enzymes and 12-hour troponin level** if the history or ECG suggests myocardial ischaemia.
 - ♦ **Plain chest radiograph** may reveal signs of heart failure, chest infection, or mitral valve disease causing left atrial enlargement.
 - ♦ **Ambulatory ECG monitoring**—A 12-lead ECG is diagnostic in identifying the underlying arrhythmia *during* symptoms, however ambulatory ECG monitoring is required for recurrent, unexplained palpitations. The patient must record their symptoms for appropriate analysis of the ECG recording, since not all asymptomatic arrhythmias require treatment.
 - ♦ **Echocardiography** will demonstrate underlying structural heart disease, valvular disease, and left ventricular dysfunction.
 - ♦ **Exercise ECG** is indicated in patients who experience palpitations on exercise.
 - ♦ **Electrophysiological studies (EPS)** are indicated in patients in whom a tachycardia is suspected but has not been confirmed by non-invasive investigations. Ablation of causative pathways can also be performed during the procedure.
 - ♦ **Cardiac MRI** (magnetic resonance imaging) may be used in selected patients to investigate for cardiomyopathies.
- Other investigations to consider in the evaluation of palpitations as guided by the history
 - ♦ **D-Dimer and/or CT pulmonary angiogram** maybe considered if the history is suggestive of a pulmonary embolus triggering sinus tachycardia or AF.
 - ♦ **Urinary catecholamine metabolites** will be raised in the presence of a pheochromocytoma.
 - ♦ **Implantable loop recorders** are small devices that can be implanted subcutaneously that can record continuously for up to 3 years. These are useful for detecting infrequent arrhythmias.

Questions commonly asked by examiners

What are the causes of AF?

The common causes of AF are:

- Cardiac
 - ♦ Ischaemic heart disease
 - ♦ Hypertension
 - ♦ Mitral valve disease
 - ♦ Congestive cardiac failure
- Non-cardiac
 - ♦ Thyrotoxicosis
 - ♦ Acute illness, e.g. pneumonia, PE
 - ♦ Alcohol
 - ♦ Idiopathic AF

How is AF distinguished from ventricular ectopic beats clinically?

Ventricular ectopic beats disappear with exercise, whereas in AF the rhythm remains unchanged.

How is AF classified?

AF is classified into three categories according to the mode of termination of the arrhythmia:

1. Paroxysmal AF terminates spontaneously without intervention.
2. Persistent episodes of AF terminate with either electrical or pharmacological cardioversion.
3. Permanent AF does not revert to sinus rhythm despite electrical or pharmacological cardioversion.

What are the management strategies for haemodynamically stable persistent and permanent AF?

Management of AF consists of rhythm and/or rate control and anticoagulation to prevent thromboembolic events. Anticoagulation is discussed below.

Rhythm control

There is no significant difference in overall mortality in patients receiving rhythm or rate control therapy. However, the rhythm control strategy is often associated with adverse effects of anti-arrhythmic agents which can be proarrhythmic. Recent NICE guidelines¹ recommend rhythm control as the first-line treatment for persistent AF in:

- young patients (<65 years)
- symptomatic patients
- lone AF
- AF secondary to a treated precipitant, i.e. infection
- patients with congestive cardiac failure (restoration of atrial contraction can contribute 20% to cardiac output)

Sinus rhythm can be restored with electrical or pharmacological cardioversion after at least 4 weeks of therapeutic anticoagulation with warfarin (Target INR: 2–3). The recurrence rate of AF is approximately 50% and therefore post-cardioversion medical treatment is often required for maintenance of sinus rhythm. First-line treatment is with standard β -blockers. Sotalol and flecainide are second-line options; the latter is contraindicated in structural heart disease.

Amiodarone is considered when there is treatment failure with other agents. In some situations, a further attempt at cardioversion may be an option, often with concurrent anti-arrhythmic therapy. Rhythm control is unlikely if left atrial diameter is greater than 5cm, as these patients have a low chance of successful cardioversion and a high chance of recurrence of AF. In such cases, a rate control strategy is more appropriate.

Rate control

The rate control strategy is appropriate in patients with failed attempts at cardioversion, permanent AF and in persistent AF in whom warfarin anticoagulation is contraindicated (thus unable to receive cardioversion). First-line drugs for ventricular control are standard β -blockers and non-dihydropyridine calcium channel blockers (verapamil and diltiazem). Digoxin is added if further rate control is required, and amiodarone may be considered if there is failure of treatment.

Other options

Device therapy can be considered as an option for rhythm control under specialist care. Atrial pacing aims to suppress atrial ectopics that initiate the arrhythmia. Atrial defibrillators attempt to cardiovert episodes of AF. Radiofrequency ablations to the left atrium or pulmonary veins are used in selected cases. In patients with permanent AF and difficult rate control despite optimum rate control therapy, ablation of the AV node with implantation of permanent pacemaker is an option.

What is the role of anticoagulation in the management of AF?

The incidence of stroke in the presence of AF is 5% per year and the risk is increased with the presence of other vascular risk factors, warranting the need for thromboprophylaxis.² The risk of stroke is stratified into high, moderate, and low risk groups, taking into consideration contraindications to antithrombotic treatment and the presence of other risk factors for stroke (age >75 years, history of thromboembolic disease, hypertension, diabetes mellitus, valvular heart disease, left ventricular dysfunction, and thyrotoxicosis). Pooled data from trials comparing anti-thrombotic therapy with placebo has shown that warfarin reduces the risk of stroke by 62% and aspirin, alone, reduces the risk by 22%. Overall, in high risk patients, warfarin is better than aspirin in preventing strokes, with a relative risk reduction of 36%. Warfarin has been shown to be more effective at stroke prevention than combination antiplatelet therapy (clopidogrel and aspirin).³ However, aspirin may be appropriate in younger patients with no risk factors, or if there is a contraindication to warfarin. Please refer to the algorithm below for anticoagulation in AF.⁴

What is the role of implantable cardioverter defibrillators (ICDs) in the management of ventricular arrhythmias?

ICDs are implantable devices which monitor heart rhythm, and can either pace, cardiovert or defibrillate depending on the indication. Their role in the prevention of sudden cardiac death due to ventricular arrhythmias has been reviewed by NICE:⁵

- *Secondary prevention of sudden cardiac death—patients with one of:*
 - ♦ a previous cardiac arrest due to VT or ventricular fibrillation (VF)
 - ♦ sustained VT causing syncope or significant haemodynamic compromise
 - ♦ sustained VT without haemodynamic compromise, with a LVEF <35%
- *Primary prevention of sudden cardiac death—patients with a history of previous (>4 weeks) MI (myocardial infarction) and either*
 - ♦ LVEF <35% with non-sustained VT on ECG monitoring and electrophysiological testing, **or**
 - ♦ LVEF <30% and prolonged QRS interval.

Differential diagnosis of palpitations

Cardiac causes

Supraventricular arrhythmias

- Atrial tachycardia
- Atrial fibrillation and atrial flutter
- Supraventricular tachycardias (SVTs)
 - * Atrioventricular re-entry tachycardia (AVRT) and Wolff-Parkinson-White (WPW) syndrome
 - * Atrioventricular nodal re-entry tachycardia (AVNRT)

Ventricular tachyarrhythmias

- Sustained ventricular tachycardia
- Non-sustained ventricular tachycardia
- Normal heart ventricular tachycardia
- Polymorphic VT (Torsades de Pointes)

Atrial and ventricular ectopic beats

Bradycardia

- Intermittent atrioventricular block
- Sinoatrial node disease

Non-cardiac causes

Anaemia

Thyrotoxicosis (causes AF and sinus tachycardia)

Hypothyroidism (causes bradycardia)

Metabolic

- Hypoglycaemia
- Pheochromocytoma

Drugs

- Caffeine
- Alcohol
- β -agonists
- Thyroxine

Anxiety/panic attacks

Physiological

- Exercise
- Pregnancy

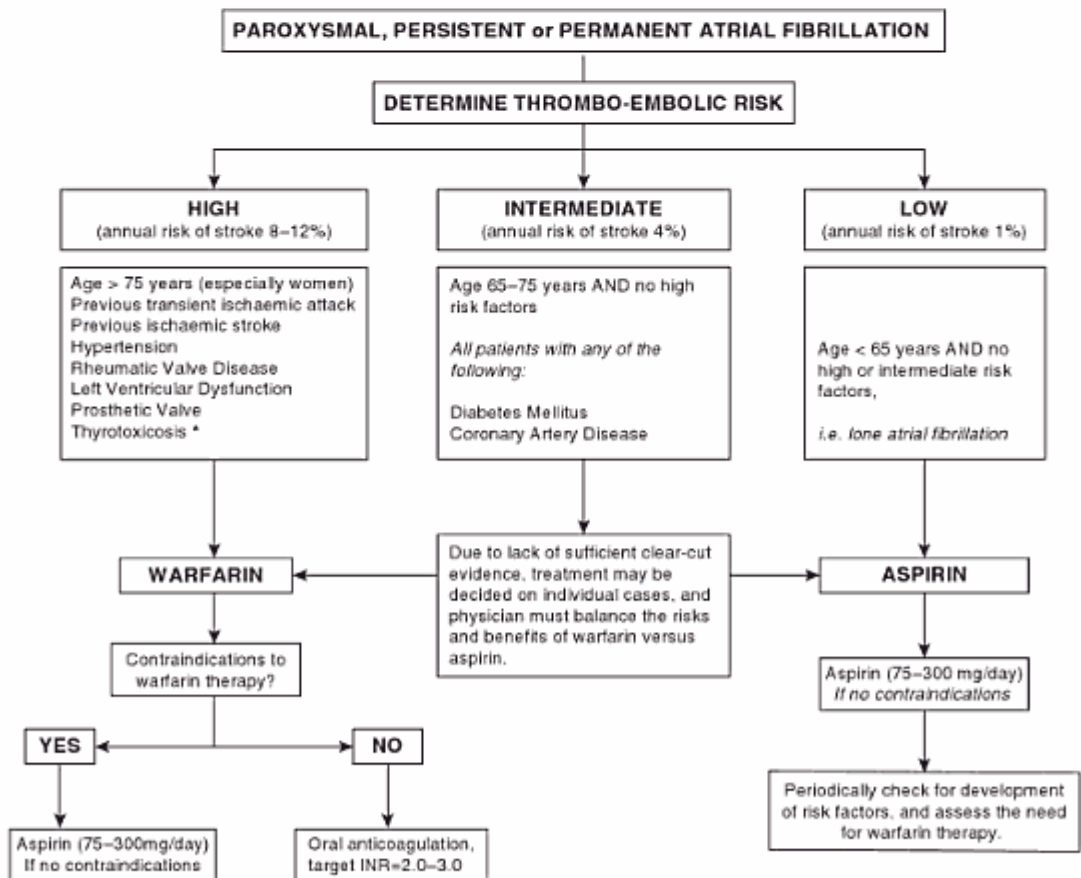


Figure 11.1 Algorithm for anticoagulation in atrial fibrillation.

Adapted from Iqbal et al. Recent developments in atrial fibrillation. BMJ 2005; 330:238–243.

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Case 12 ♦ Ankle Swelling

INFORMATION FOR THE CANDIDATE

Dear Doctor,

Thank you for seeing this 69-year old woman who gives a 6-month history of worsening ankle swelling. She has noticed a gradual reduction in exercise tolerance, which she attributes to her ankle swelling. There is no history of chest pain, orthopnoea, or paroxysmal nocturnal dyspnoea. She has a history of asthma, rheumatoid arthritis, diabetes, and hypertension. Her medications include aspirin 75mg OD, salbutamol inhaler 2 puffs PRN, ibuprofen 400mg TDS, metformin 500mg TDS, gliclazide 80mg BD, amlodipine 5mg OD, and furosemide 40mg OD.

I am very grateful for your opinion.

Acquiring a history

A. History of presenting complaint:

Onset and duration

- ‘When did the ankle swelling first start?’ If acute (<72 hours) onset, then deep vein thrombosis should be strongly considered.
- ‘Was it gradual and sudden?’
- ‘Has it been progressive or intermittent?’

Pattern, symmetry, and extent of involvement

- It is important to enquire about the pattern and symmetry early in the history.
- Remember the key causes of asymmetrical versus symmetrical involvement:
 - ♦ *Asymmetrical*: venous thrombosis, lower limb venous obstruction, trauma, infection, lymphoedema, and arthritis
 - ♦ *Symmetrical*: congestive cardiac failure, cor pulmonale, hypoalbuminaemia (nephrotic syndrome, chronic liver disease, poor nutrition), immobility, dependent oedema, lymphoedema, hypothyroidism, arthritis, and drugs
- Enquire about the extent of involvement. Start by asking open questions, and if necessary use closed questions: ‘Is it confined to the joints?’ (arthritis); ‘How far up the legs/thighs does the

swelling extend?'; 'Is there swelling of your groin?' (scrotal and/or penile swelling in males); 'Is there abdominal swelling?'

- Ask if the oedema is pitting in nature. 'Does pressing on the swelling with your fingers leave finger marks?' Lymphoedema and oedema secondary to hypothyroidism is often non-pitting in nature. Early lymphoedema can be associated with pitting oedema.

Aggravating factors

- Dependant oedema or oedema due to venous insufficiency is often worse at the end of the day.
- Although a full drug history will be taken later, it is important to clarify if the onset of the ankle oedema coincides with any drugs that may have been prescribed for other medical conditions, i.e. calcium antagonists for hypertension.
- Enquire about trauma, animal bites, and stings.

Relieving factors

- Dependant oedema or oedema due to venous insufficiency is relieved by leg elevation and/or compression stockings.
- Ask if diuretics have been prescribed, and if so, 'Have the diuretics reduced the ankle swelling?'

Other features

- 'Is there any associated leg weeping or oozing?'
- 'Is there any skin discoloration?' A brown haemosiderin discoloration over the medial aspect of the ankle suggests venous insufficiency. Erythema and tenderness suggests active inflammation, i.e. arthritis or cellulitis.
- Enquire about any trophic changes to the skin, suggesting previous ulceration.

Associated features

- **Deep venous thrombosis:** calf pain and tenderness. It is important to enquire if the oedema is painful. Pain is associated with deep vein thrombosis. Oedema secondary to chronic venous insufficiency, congestive cardiac failure, and renal disease can cause low-grade aching. Lymphoedema is usually painless.
- **Cellulitis:** erythema, tenderness, fever
- **Arthritis:** joint aches, tenderness, and swelling
- **Cardiac failure:** exertional breathlessness, orthopnoea, and paroxysmal nocturnal dyspnoea. Enquire about symptoms of cardiac ischaemia, i.e. exertional chest pain
- **Cor pulmonale:** enquire about symptoms of respiratory disease, i.e. chronic cough, sputum, haemoptysis; obstructive sleep apnoea (snoring, daytime somnolence, large collar size)
- **Chronic liver disease:** jaundice, pruritus, abdominal swelling, haematemesis, and melaena
- **Malabsorption:** enquires about symptoms of inflammatory bowel disease (weight loss, bloody diarrhoea, joint aches, and rash on legs [erythema nodosum]), and Coeliac disease (intolerance to gluten).
- **Nephrotic syndrome:** periorbital and generalized oedema, breathlessness (pleural effusions), nausea, vomiting.
- **Hypothyroidism:** goitre, weight gain, cold intolerance, fatigue, and constipation
- **Extrinsic venous compression:** is there any history to suggest underlying occult malignancy? Ask about weight loss, loss of appetite, change in bowel habit, and unusual bumps/lumps (lymphadenopathy).
- **Renal disease:** Renal failure can result in fluid retention and breathlessness. Enquire about uraemic symptoms, particularly restlessness and pruritus. Ask about urinary symptoms, particularly any noticeable reduction in urine output.

Exercise tolerance

- It is important to quantify reduction in exercise tolerance.
- Establish pre-morbid, and then current functional status and exercise tolerance.
- This can be used to establish New York Heart Association (NYHA) functional class: I (unrestricted), II (breathless on heavy exertion), III (breathless on mild exertion), and IV (breathless at rest).

B. Relevant previous medical and family history:

Medical history

- **Cardiac disease**
 - Enquire about cardiovascular risk factors (smoking, hypertension, hypercholesterolaemia, diabetes, family history, previous history of myocardial infarction)
 - If risk factors are present, enquire about risk factor control and compliance to therapy. *'Do you remember the last blood pressure measurement?'* *'Have you had your cholesterol level checked?'* If so, *'Do you remember what it was?'*
 - If previous cardiac history, then enquire about previous myocardial infarction, coronary angiography, coronary angioplasty, or CABG surgery.
 - Enquire about pre-existing left ventricular dysfunction or previous echocardiography. *'Have you ever had an ultrasound scan of the heart'* If so, *'What did it show?'*
 - Enquire about any history of valvular heart disease. *'Have you ever been told that you have heart murmur?'*; *'Is there a history of rheumatic fever?'*
- **Respiratory disease**
 - History of chronic lung diseases, i.e. asthma, COPD, bronchiectasis, interstitial lung disease, sarcoidosis (ask about fever, joint aches, and painful nodules on the legs). Vasculitis symptoms may implicate pulmonary vasculitis as a cause of pulmonary hypertension and cor pulmonale.
 - Thrombotic risk factors (previous history of thromboembolic disease, thrombophilia, immobility, recent surgery or long-haul flight, and oral contraceptive pill use). Chronic thromboembolic disease is an important cause of pulmonary hypertension, leading to cor pulmonale.
 - Obstructive sleep apnoea can cause cor pulmonale.
- **Rheumatological disease**
 - History of arthritis—enquire about distribution of arthritis. Does it correlate with sites of swelling?
 - Enquire about systemic lupus erythematosus (arthralgia, myalgia, and facial rash) and systemic sclerosis (arthralgia, myalgia, rash, skin changes). Limited cutaneous systemic sclerosis is associated with pulmonary hypertension.
 - Enquire about previous hip and knee replacements. This can result in pitting localized oedema.
- **Thyroid disease**
 - Hypothyroidism is associated with generalized subcutaneous oedema.
 - Graves' disease can cause hypothyroidism and hyperthyroidism, as well as pretibial myxoedema. Pretibial myxoedema in its extreme form can resemble lymphoedema.
- **Renal disease**
- **Nephrotic syndrome**
- **Chronic liver disease**
- **Malabsorption**
 - Enquire about Crohn's disease, ulcerative colitis, and Coeliac disease

- **Lymphoedema**
 - ♦ Previous surgery and irradiation (damage to lymphatic drainage)
- **Venous insufficiency**
 - ♦ Previous history of deep vein thrombosis or varicose veins.

Family history

- **Ischaemic heart disease**—enquire about the age at first presentation (establish risk of premature coronary artery disease)
- **Cardiomyopathy**—familial dilated or hypertrophic cardiomyopathy
- **Thrombo-embolic disease**—enquire about thrombophilia

C. Medications;

- Complete a full drug history and compliance to therapy.
- Enquire about complications of medications.
- Calcium antagonists, long-term steroids, hydralazine, minoxidil, methyldopa, thiazolidinediones (pioglitazone and rosiglitazone), monoamine oxidase inhibitors, and NSAIDs can cause fluid retention.
- Oral contraceptive pill and HRT use in females increases thromboembolic risk.
- Appetite suppressors, i.e. fenfluramine are associated with pulmonary hypertension.
- Nephrotoxic medications can potentiate and aggravate renal failure. (eg. Nsaids, penicillins, gentamicin, sulphonamides, ACE inhibitors, ciclosporin).

D. Social issues:

- Smoking habits (cardiovascular risk factor)
- Alcohol consumption (risk factor for dilated cardiomyopathy and chronic liver disease)
- Impact of symptoms on daily life
- Elicit the patient's concerns about the symptoms

Formulating a plan of action

Explain to the patient that there are possible causes for ankle oedema. It is important to exclude drug causes of ankle oedema and dependent oedema. This should be clear from the history. Subsequent investigations will reflect possible underlying causes. Preliminary screening investigations include:

- **Bloods**
 - ♦ **Anaemia** can potentiate cardiac ischaemia and cardiac failure
 - ♦ **Leucocytosis** and **raised inflammatory markers** would suggest underlying infection, i.e. cellulitis
 - ♦ **Urea and electrolytes (U&Es)**: renal failure
 - ♦ **D-dimer** for suspected thrombo-embolic disease
 - ♦ **Cardiac enzymes** if suspecting myocardial ischaemia
 - ♦ **Brain natriuretic peptide (BNP)** if suspecting cardiac failure
 - ♦ **LFT and clotting if suspecting chronic liver disease**
 - ♦ **Autoimmune profile** and **rheumatoid factor** if suspecting rheumatological disease
 - ♦ **Albumin**: hypoalbuminaemia is seen in nephrotic syndrome, chronic liver disease, advanced chronic illnesses, and poor nutritional status.
 - ♦ **Lipid profiles**: hypercholesterolaemia may be seen in nephrotic syndrome
 - ♦ **Thyroid function tests**

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