Cases for PACES

Second Edition

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Specialist Registrars
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Addenbrooke's Hospital
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Foreword

Amidst the turmoil of recent 'modernization' of medical careers, the fundamental essentials of the practice of clinical medicine have not changed at all. The doctor needs to be able to take a history from a patient, examine them and decide whether investigations and/or treatment are required. They then need to be able to discuss the various options with the patient in a manner that they can understand, hopefully reaching a sensible mutual understanding about how best to proceed. In some instances the doctor may need to give difficult and distressing information, and must learn how to do this in a manner that eases the pain rather than increases it. And all of these things must be done in a reasonable time frame: the next patient is waiting.

The MRCP PACES examination remains the measure that is most generally respected as indicating that a doctor has developed a fair degree of the knowledge, skills and behaviours that are necessary to do the things detailed above. They are not yet the finished article (beware of anyone who thinks they are), but they can proceed from a junior to a senior stage of training. The examination is not easy, with a pass rate of around 40%. Those preparing for it need to immerse themselves in clinical work. There is no substitute for seeing a lot of cases and taking histories and performing examinations, but – and here is where books such as *Cases for PACES* come in – it is not helpful to endlessly repeat sloppy practice. The physician examining you in the PACES examination is thinking: 'Is this doctor ready to be my SpR now? Can they sort things out in a reasonably efficient and sensible way? Would I get a lot of people wanting to see me because problems had been poorly explained or dealt with?'

What comes over in *Cases for PACES* is an approach that does sort the wood from the trees, which cuts pretty rapidly to the chase, and I recommend it to you. If you do what it says on the tin, you will stand a very good chance of passing the examination.

Dr John Firth
Consultant Physician, Addenbrooke's Hospital, Cambridge

Preface

PACES (Practical Assessment of Clinical Examination Skills) was initiated in June 2001 by the Royal College of Physicians as the final stage of the MRCP examination. The initial examination consisted of five stations in a carousel: Station 1: Respiratory/Abdominal (10 minutes each), Station 2: History Taking (20 minutes), Station 3: Cardiology/Neurology (10 minutes each), Station 4: Communication Skills and Ethics (20 minutes) and Station 5: Short Cases (Skin, Locomotor, Eyes and Endocrine: 5 minutes each). The main changes from the original MRCP long and short case format were that candidates had to take a history and communicate medical diagnoses to lay patients in front of their examiners. The viva was replaced by 'discussions' that occurred at the end of each case, which concentrated on management issues relating to the case.

PACES was refined in October 2009 by restructuring Station 5. There are now two 10-minute 'Brief Clinical Consultations' that reflect day-to-day practice at work rather than the four 5-minute cases. Candidates will be expected to take a targeted history and a focused rather than thorough examination each lasting 8 minutes with the remaining time for discussion. The cases previously encountered in Station 5 will be accommodated within the other stations and candidates must still prepare to examine these systems. New topics to Station 5 will include acute and geriatric medicine, previously underrepresented in the PACES examination.

Cases for PACES 2nd Edition prepares candidates for the updated PACES examination. It mimics the examination format, and is designed for use in an interactive way. The *2nd Edition* is a completely revised text, incorporating the changes to Station 5, as well as new cases. It has useful information on ethical and legal issues, history-taking advice and worked examples. It also provides mock questions for candidates to practise themselves. The short cases that appeared in the original Station 5 remain as an appendix, but Station 5 now contains new Brief Clinical Consultations including worked examples in acute and geriatric medicine. However, seeing medical patients on a busy receiving unit or outpatient department must be the best way to prepare for this new station!

Common cases rather than rarities have been deliberately chosen and are set out in an examination format. It is taken as read that candidates will be familiar in examination techniques and the appropriate order in which

to elicit the various signs. In the book, only the key diagnostic clinical signs are documented, followed by extra points that will ensure you score high marks in the case. What follows in the discussion are some of the potential topics that a candidate could be expected to comment on at the end of the case. Examiners are specifically monitoring for knowledge of the differential diagnosis and organized clinical judgement, whilst managing the patients' concerns and maintaining patient welfare. The detail is not exhaustive but rather what is reasonably needed to pass. There is additional room for candidates to make further notes as they see fit. This book is designed to enable groups of candidates to practise 'under examination conditions' at the bedside.

The aim of this book is to put the information that is frequently tested in the clinical PACES examination in a succinct format that will enable capable candidates to pass with ease.

Good luck.

Stephen Hoole
Andrew Fry
Daniel Hodson
Rachel Davies

Acknowledgements

We thank the doctors who taught us for our own PACES examination, and above all the patients who allow us to refine our examination techniques and teach the next generation of MRCP PACES candidates.

Abbreviations

ABG	Arterial blood gas
ACE	Angiotensin-converting enzyme
ACTH	Adrenocorticotrophic hormone
AF	Atrial fibrillation
AFP	Alpha-fetoprotein
AICD	Automated implantable cardiac defibrillator
ANA	Anti-nuclear antibody
AR	Aortic regurgitation
ARVD	arrhythmogenic right ventricular dysplasia
5-ASA	5-Aminosalicylic acid
ASD	Atrial septal defect
AVR	Aortic valve replacement
BIPAP	Bi-level positive airway pressure
BM	Bohereinger Manheim (glucose)
BMI	Body mass index
CABG	Coronary artery bypass graft
CAPD	Continuous ambulatory peritoneal dialysis
CCF	Congestive cardiac failure
CFA	Cryptogenic fibrosing alveolitis
CFTR	Cystic fibrosis transmembrane conductance regulator
CK	Creatine kinase
CML	Chronic myeloid leukaemia
CMV	Cytomegalovirus
COPD	Chronic obstructive pulmonary disease
COMT	Catechol-o-methyl transferase
CRP	C-reactive protein
CSF	Cerebrospinal fluid
CVA	Cerebrovascular accident
CXR	Chest X-ray (radiograph)
DIPJ	Distal interphalangeal joint
DM	Diabetes mellitus
DVLA	Driver and Vehicle Licensing Agency
DVT	Deep vein thrombosis
eGFR	Estimated glomerular filtration rate
EBV	Epstein–Barr virus
ECG	Electrocardiogram
EMG	Electromyogram
ESR	Erythrocyte sedimentation rate
FBC	Full blood count
FEV_1	Forced expiratory volume in 1 second
FTA	Fluorescent treponema antibodies
FVC	Forced vital capacity
GH	Growth hormone
Hb	Haemoglobin
HBV	Hepatitis B virus
HCG	Human chorionic gonadotrophin
HCV	Hepatitis C virus
HGV	Heavy goods vehicle

HLA	Human lymphocyte antigen
HOCM	Hypertrophic obstructive cardiomyopathy
HRT	Hormone replacement therapy
HSMN	Hereditary sensory motor neuropathy
HSV	Herpes simplex virus
IBD	Inflammatory bowel disease
IDDM	Insulin-dependent diabetes mellitus
IGF	Insulin-like growth factor
INR	International normalized ratio
ITP	Immune thrombocytopaenic purpura
IV	Intravenous
JVP	Jugular venous pressure
K_{CO}	Transfer coefficient
LAD	Left axis deviation
LDH	Lactate dehydrogenase
LFT	Liver function test
LMWH	Low molecular weight heparin
LQTS	Long QT syndrome
LV	Left ventricle
LVH	Left ventricular hypertrophy
mAb	Monoclonal antibody
MAO	Monoamine oxidase
MI	Myocardial infarction
MND	Motor neurone disease
MPTP	Methyl-phenyl-tetrahydropyridine
MR	Mitral regurgitation
MRI	Magnetic resonance imaging
MCPJ	Metacarpophalangeal joint
MTPJ	Metatarsophalangeal joint
MVR	Mitral valve replacement
NIPPV	Non-invasive positive pressure ventilation
NSAIDs	Non-steroidal anti-inflammatory drugs
NSCLC	Non-small cell lung cancer
OA	Osteoarthritis
P_a	Partial pressure (arterial)
PBC	Primary biliary cirrhosis
PCT	Primary Care Trust
PEG	Percutaneous endoscopic gastrostomy
PEFR	Peak expiratory flow rate
PET	Positron emission tomography
PIPJ	Proximal interphalangeal joint
PRL	Prolactin
PSC	Primary sclerosing cholangitis
PSV	Public service vehicle
PTHrP	Parathyroid hormone-related peptide
PUVA	Psoralen ultraviolet A
RA	Rheumatoid arthritis
RAD	Right axis deviation
RBBB	Right bundle branch block
RR	Respiratory rate
RV	Right ventricle
RVH	Right ventricular hypertrophy
Rx	Treatment

SCLC	Small cell lung cancer
SIADH	Syndrome of inappropriate anti-diuretic hormone
SLE	Systemic lupus erythematosus
SOA	Swelling of ankles
SSRI	Selective serotonin reuptake inhibitor
SVCO	Superior vena cava obstruction
T_4	Thyroxine
***T*°C**	Temperature
TIA	Transient ischaemic attack
TIMI	Thrombolysis in myocardial infarction
T_LCO	Carbon monoxide transfer factor
TNM	Tumour nodes metastasis (staging)
TOE	Transoesophageal echo
TPHA	*Treponema pallidum* haemaggutination assay
TR	Tricuspid regurgitation
TSH	Thyroid stimulating hormone
TTE	Transthoracic echo
U&E	Urea and electrolytes
UC	Ulcerative colitis
UFH	Unfractionated heparin
UIP	Usual interstitial pneumonia
UTI	Urinary tract infection
VEGF	Vascular endothelial growth factor
VSD	Ventricular septal defect
WCC	White cell count

Station 1
Abdominal and Respiratory

Chronic liver disease and hepatomegaly

This man complains of weight loss and abdominal discomfort. His GP has referred him to you for a further opinion. Please examine his abdomen.

Clinical signs

Signs of chronic liver disease

- **General:** cachexia, icterus (also in acute), excoriation and bruising
- **Hands:** leuconychia, clubbing, Dupuytren's contractures and palmar erythema
- **Face:** xanthelasma, parotid swelling and fetor hepaticus
- **Chest and abdomen:** spider naevi and caput medusa, reduced body hair, gynaecomastia and testicular atrophy (in males)

Signs of hepatomegaly

- Palpation and percussion:
  - Mass in the right upper quadrant that moves with respiration, that you are not able to get above and is dull to percussion
  - Estimate size (finger breadths below the diaphragm)
  - Smooth or craggy/nodular (malignancy/cirrhosis)
  - Pulsatile (TR in CCF)
- Auscultation
  - Bruit over liver (hepatocellular carcinoma)

Extra points

Evidence of an underlying cause of hepatomegaly

• Tattoos and needle marks	Infectious hepatitis/alcohol
• Pigmentation	Haemochromatosis
• Cachexia	Malignancy
• Mid-line sternotomy scar	CCF

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Evidence of treatment
- Ascitic drain/tap sites and peritonovenous shunts
- Surgical scars

Evidence of decompensation
- **A**scites: shifting dullness
- **A**sterixis: 'liver flap'
- **A**ltered consciousness: encephalopathy

Discussion
Causes of hepatomegaly
The **big three:**
Cirrhosis (alcoholic)
Carcinoma (secondaries)
Congestive cardiac failure
Plus: **I**nfectious (HBV and HCV)
Immune (PBC, PSC and autoimmune hepatitis)
Infiltrative (amyloid and myeloproliferative disorders)

Investigations
- Bloods: FBC, clotting, U&E, LFT and glucose
- Ultrasound scan abdomen
- Tap ascites (if present)

If cirrhotic
- Liver screen bloods:
  - Autoantibodies and immunoglobulins (PBC and autoimmune hepatitis)
  - Hepatitis B and C serology
  - Ferritin (haemochromatosis)
  - Caeruloplasmin (Wilson's disease)
  - α-1 antitrypsin
  - Autoantibodies and immunoglobulins (PBC and autoimmune hepatitis)
  - AFP (hepatocellular carcinoma)
- Hepatic synthetic function: INR (acute) and albumin (chronic)
- Liver biopsy (diagnosis and staging)
- ERCP (diagnose/exclude PSC)

If malignancy
- Imaging: CXR and CT abdomen/chest
- Colonoscopy/gastroscopy
- Biopsy

Complications of cirrhosis
- Variceal haemorrhage due to portal hypertension
- Hepatic encephalopathy
- Spontaneous bacterial peritonitis

Child-Pugh classification of cirrhosis
Prognostic score based on bilirubin/albumin/INR/ascites/encephalopathy

	Score	1 year prognosis
A:	5–6	100%
B:	7–9	81%
C:	10–15	45%

Causes of ascites
- **C**irrhosis (80%)
- **C**arcinomatosis
- **C**CF

Treatment of ascites in cirrhotics
- Abstinence from alcohol
- Salt restriction
- Diuretics (aim: 1 kg weight loss/day)
- Liver transplantation

Causes of palmar erythema
- Cirrhosis
- Hyperthyroidism
- Rheumatoid arthritis
- Pregnancy
- Polycythaemia

Causes of gynaecomastia
- Physiological: puberty and senility
- Kleinfelter's syndrome
- Cirrhosis
- Drugs, e.g. spironolactone and digoxin
- Testicular tumour/orchidectomy
- Endocrinopathy, e.g. hyper/hypothyroidism and Addison's

Haemochromatosis

This 52-year-old man was referred after a diagnosis of diabetes mellitus was made by his GP. Please examine him and discuss further investigations.

Clinical signs

- Increased skin pigmentation
- Stigmata of chronic liver disease
- Hepatomegaly

Extra points

Scars

- Venesection
- Liver biopsy
- Joint replacement
- Abdominal rooftop incision (hemihepatectomy for hepatocellular carcinoma)

Evidence of complications

- **Endocrine:** 'bronze diabetes' (e.g. injection sites), hypogonadism and testicular atrophy
- **Cardiac:** congestive cardiac failure
- **Joints:** arthropathy (pseudo-gout)

Discussion

Inheritance

- Autosomal recessive on chromosome 6
- **HFE** gene mutation: regulator of gut iron absorption
- Homozygous prevalence 1:300, carrier rate 1:10
- Males affected at an earlier age than females – protected by menstrual iron losses

Presentation

- Fatigue and arthritis
- Chronic liver disease
- Incidental diagnosis or family screening

Investigation

- ↑ Serum ferritin
- ↑ Transferrin saturation

- ↓ Total iron-binding capacity
- Liver biopsy (diagnosis + staging)
- Genotyping

And consider:

• Blood glucose	Diabetes
• ECG, CXR, ECHO	Cardiac failure
• Liver ultrasound, α-fetoprotein	Hepatocellular carcinoma (HCC)

Treatment

- Regular venesection (1 unit/week) until iron deficient, then venesect 1 unit, 3–4 times/year
- Avoid alcohol
- Surveillance for HCC

Family screening (1st degree relatives aged > 20 years)

- Iron studies

If positive:

- Liver biopsy
- Genotype analysis

Prognosis

- 200 × increased risk of HCC if cirrhotic
- Reduced life expectancy if cirrhotic
- Normal life expectancy without cirrhosis + effective treatment

Liver transplantation in haemochromatosis

- Only 50% 1-year survival
- High mortality: cardiac + infectious complications

Splenomegaly

This man presents with tiredness and lethargy. Please examine his abdominal system and discuss your diagnosis.

Clinical signs

General

- Anaemia
- Lymphadenopathy (axillae, cervical and inguinal areas)
- Purpura

Abdominal

- Left upper quadrant mass that moves inferomedially with respiration, has a notch, is dull to percussion and you cannot get above nor ballot
- Estimate size
- Check for hepatomegaly

Extra points

• Lymphadenopathy	Haematological and infective
• Stigmata of chronic liver disease	Cirrhosis with portal hypertension
• Splinter haemorrhages, murmur etc.	Bacterial endocarditis
• Rheumatoid hands	Felty's syndrome

Discussion

Causes

- Massive splenomegaly (>8 cm):
  - Myeloproliferative disorders (**CML** and **myelofibrosis**)
  - Tropical infections (**malaria**, visceral leishmaniasis: kala-azar)
- Moderate (4–8 cm):
  - Myelo/lymphoproliferative disorders
  - Infiltration (Gaucher's and amyloidosis)
- Tip (<4 cm):
  - Myelo/lymphoproliferative disorders
  - Portal hypertension
  - Infections (EBV, infective endocarditis and infective hepatitis)
  - Haemolytic anaemia

Investigations

- Ultrasound abdomen

Then if:

- **Haematological:**
  - FBC and film
  - CT chest and abdomen

  - Bone marrow aspirate and trephine
  - Lymph node biopsy
- **Infectious:**
  - Thick and thin films (malaria)
  - Viral serology

Indications for splenectomy

- Rupture (trauma)
- Haematological (ITP and hereditary spherocytosis)

Splenectomy work-up

- Vaccination (ideally 2/52 prior to protect against encapsulated bacteria):
  - Pneumococcus
  - Meningococcus
  - *Haemophilus influenzae* (Hib)
- Prophylactic penicillin
- Medic alert bracelet

Renal enlargement

This woman has been referred by her GP for investigation of hypertension. Please examine her abdomen.

Clinical signs

Peripheral

- Blood pressure: **hypertension**
- Arteriovenous fistulae (thrill and bruit), tunnelled dialysis line
- Immunosuppressant 'stigmata', e.g. cushingoid habitus due to steroids, gum hypertrophy with ciclosporin

Abdomen

- Palpable kidney: ballotable, can get above it and moves with respiration
- Iliac fossae: scar with (or without!) transplanted kidney
- Ask to dip the urine: proteinuria and haematuria
- Ask to examine the external genitalia (varicocele in males)

Extra points

- Hepatomegaly: polycystic kidney disease
- Indwelling catheter: obstructive nephropathy with hydronephrosis
- Peritoneal dialysis catheter/scars

Discussion

Causes of unilateral enlargement

- Polycystic kidney disease (other kidney not palpable or contralateral nephrectomy – flank scar)
- Renal cell carcinoma
- Simple cysts
- Hydronephrosis (due to ureteric obstruction)

Causes of bilateral enlargement

- Polycystic kidney disease
- Bilateral renal cell carcinoma (5%)
- Bilateral hydronephrosis
- Tuberous sclerosis (renal angiomyolipomata and cysts)
- Amyloidosis

Investigations

- U&E
- Urine cytology