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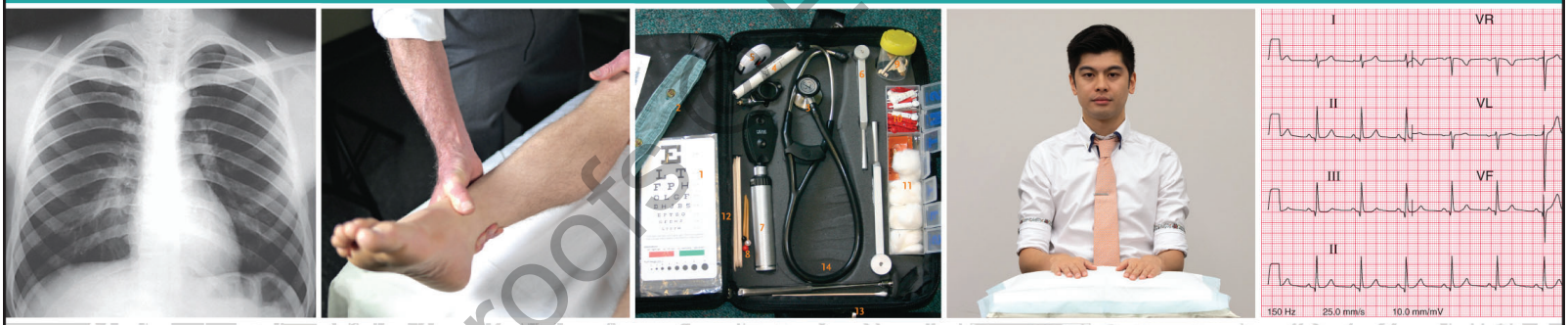
Talley & O'Connor's

Examination Medicine

A guide to physician training

10TH EDITION

NICHOLAS J TALLEY & SIMON O'CONNOR



ELSEVIER

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A Guide to Physician Training

10TH EDITION

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Foreword

They dared to be doctors: Elizabeth Blackwell, Elizabeth Garrett Anderson was published in 1965. Elizabeth Garrett Anderson was the first woman in England to qualify as a physician in the 1860s, and Elizabeth Blackwell became the first woman to earn a medical degree in the United States in 1849.

Their journey was fraught with challenges, impeded by prejudice and misconceptions. Yet they persevered against all odds, treating thousands of patients. Dr Anderson founded the London School of Medicine for Women, which later became the Royal Free Hospital School of Medicine and is now part of University College London's medical school.

They dared to be doctors was a gift from my father. In the 1990s, as a young teenager growing up in Mauritius, Dr Blackwell and Dr Anderson inspired me to dare to be a doctor too despite my family's modest means, thanks to my dear father, who deeply valued education for his children. A pivotal moment in my life was winning a medical scholarship to the University of Melbourne. I continue to dare to break barriers and challenge preconceived norms as I am equally humbled and proud to belong to one of the most noble of professions.

Physicians are the healers of souls and bodies. Physicians are the poets of medicine whose verses are written in the lives they touch, healing not just the body but also the spirit and mind. Through the seasons of sickness and health, they remain steadfast, a beacon of guidance in the tempest of uncertainty, nurturing life with the delicate balance of science and empathy.

To this day, the journey of physicians remains as challenging as those of Drs Blackwell and Anderson, almost 200 years ago. A career in medicine requires resilience, extraordinary discipline and devotion.

Talley and O'Connor's Examination Medicine: A Guide to Physician Training is an integral part of the journey of every Australian and New Zealand physician. It is the book that helps ease the load, a trustworthy friend and ally in the challenging world of medicine.

Talley and O'Connor, affectionately known as "The Bible", is an essential text for every physician trainee, particularly during their summative examination year. This book is meticulously studied by each conscientious trainee, almost guaranteeing success in the short-case examinations. It serves as an invaluable guide for conducting clinical examinations of each body system in a clear, concise and logical approach, providing interpretations of each sign, list of possible diagnoses and their prevalence in the Australian and New Zealand context.

First written by Professor Nicholas Talley and Dr Simon O'Connor during their registrar years, the book offers a unique ground-level perspective from trainees. Now in its 10th edition, the book retains its grounded approach to clinical examination through the minds of true clinicians. It emphasises the importance of skills and clinical acumen in diagnosing and assessing the severity of diseases. Over the years, the authors have diligently kept the book vibrant and updated, now including an e-book and videos.

Talley and O'Connor remains an integral part of the rigorous journey of physician trainees preparing for clinical examinations, helping them reach the pinnacle of physician skills and build confidence in their ability to qualify as internal medicine physicians. To this date, I still have my own copy of *Talley and O'Connor*, and when faced with an

unusual clinical sign or preparing to act as an FRACP examiner, I revisit the book to refresh my knowledge. I know I can always rely on *Talley and O'Connor* to provide the gold standard of internal medicine examinations.

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Preface to the 10th edition

Practising medicine of the highest standard is both art and science; physicians are meant to think and think deeply. Use this book as a help for your (examination) preparation, not something to be learned by heart.

***Examination Medicine* (2014)**

Welcome to the 10th edition of *Examination Medicine*! It seems extraordinary that it's now 40 years ago that we set out to write a textbook that aimed to help and guide trainees pass the Royal Australasian College of Physicians (RACP) Part One clinical examination. Today, these barrier examinations are still high stakes and difficult. There are other postgraduate examinations that are of a high standard, such as the Practical Assessment of Clinical Examination Skills – PACES – examination for the Membership of the Royal College of Physicians of the United Kingdom – MRCP(UK). However, in our view the RACP examination stands out globally as one of the most rigorous and challenging barrier clinical examinations.

Today, most candidates pass the RACP written and clinical examinations because they come very well trained and prepared. Passing requires mastering clinical skills including history taking, excellence in physical examination under pressure, an ability to maturely synthesise all available clinical data and the expertise to diagnose and sensibly manage very complex medical patients at the high level expected of those progressing to advanced training. There is currently no required summative exit examination for subspecialty advanced training (as occurs internationally) so the Fellow of the Royal Australasian College of Physicians (FRACP) Part One is the last formal barrier clinical examination many trainees will ever do.

We are delighted *Examination Medicine* remains a popular book, not just among physicians sitting the Fellowship but also among trainees sitting long- and short-case examinations in many specialist Colleges, from emergency medicine to psychiatry. We also know from the sales and feedback that senior medical students and overseas medical graduates use and recommend the book, although our undergraduate textbook *Clinical Examination* is more aimed at them. (However, note that the advanced sections in *Clinical Examination* will be useful for those sitting their postgraduate barrier examinations too.)

Medical knowledge now doubles at an astonishing rate, estimated as every few months. Disease management continues to advance rapidly in many fields, with new tests and treatments, although for most chronic diseases cure remains out of reach (for now). Global mega-trends in medicine are reshaping, or will soon reshape, clinical practice. This includes (to name a few):

- the ascendancy of artificial intelligence (with an ever-increasing capacity to replace some of what we do!)
- the increasing opportunities presented by telemedicine, virtual reality, new bedside tools and wearable devices
- advances in personalised medicine, including tissue engineering and gene therapies
- the explosion of new vaccine research

- the rise of an ageing population and chronic diseases globally (although it is estimated by 2100 that the global population, having peaked earlier, will dramatically decline).

Yet we as senior clinicians appreciate how important the skills we acquired as basic physician trainees – the same skills needed to still pass the RACP examinations today – remain essential now and into the foreseeable future.

The secret to success in the clinical examinations, and practising excellent medicine in the 21st century, is to gain clinical experience by the bedside and to constantly practice and drill clinical skills, guided by your senior colleagues and role models. A mature clinical approach is also needed to pass the FRACP Part One clinical examination. This requires you to understand each patient's unique personal and social environment, and complex medical problem-solving must be considered in this context; we have written the book with this key principle in mind. For these reasons, in our view formal barrier clinical examinations remain irreplaceable, as they uniquely drive candidates to achieve the required high standards and over time gain mastery.

In this edition, like in previous editions, we have updated the long cases and added new material into the short cases, as well as kept all chapters as current as possible. The book has again undergone expert peer review to help us ensure clarity and coverage and to hopefully avoid errors. We thank the peer reviewers who have helped us update and revise this edition.

We welcome any feedback for the next edition! We wish you every success in your examinations and in your medical career.

Authors' statement

Professor Nick Talley AC is a gastroenterologist, educator and researcher, a local RACP examiner and past president of the RACP. Dr Simon O'Connor, a cardiologist, has been a longstanding member of the RACP Senior Examination Panel (SEP) until retiring from clinical practice in 2024.

Examination Medicine, first published in 1986, is not an RACP publication, nor is it endorsed by the RACP. Trainees should directly consult the College website to obtain up-to-date information about all policies and procedures as these are subject to regular change.

Acknowledgements

The authors and publishers wish to thank all the peer reviewers who have provided feedback or updated content for this new edition, and all past editions. This edition is dedicated to you.

We are especially grateful to Allergist and Immunologist Professor Brad Frankum OAM, BMed (Hons), FRACP for his revisions and updates to this edition.

We also wish to thank the following people for their assistance with the preparation of *Talley and O'Connor's Examination Medicine* 10th edition.

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The authors displaying the 1st and 9th editions of *Examination Medicine* in 2025.

Chapter 11

The renal long case

When the patient dies the kidneys may go to the pathologist, but while he lives the urine is ours.
Thomas Addis (1881–1949)

Chronic kidney disease

Chronic kidney disease (CKD) by itself is not a particularly common main problem for the long case. However, it is a difficult and important topic and kidney disease is often present in that very common long case: the obese person with diabetes with hypertension and vascular disease.

Patients receiving renal replacement therapy (RRT) (dialysis or transplant) are also commonly seen in the exam.

Keep in mind the current CKD classification and the causes of and risk factors for progression of CKD (Tables 11.1–11.3). CKD is classified based on the eGFR and the level of proteinuria and helps to risk stratify patients. The patient will usually know that he or she has renal disease. Methodical questioning to establish the diagnosis, cause, management, disease course and complications is necessary.

Fast facts on the eGFR

- The estimated glomerular filtration rate (eGFR) is a useful tool for communicating to patients. It conveys the level of renal function. One can simplify it as a percentage of global kidney function. The normal range is 80–120 mL/min (thus the average is 100 mL/min). This means that if the eGFR is 30 mL/min, patients can be told they have 30% of normal renal function.
- CKD patients often know their eGFRs and many can tell you their CKD stage.
  - The measurement tends to underestimate normal or near-normal renal function.
  - The serum creatinine should be stable for a number of days for an accurate eGFR reading, so eGFR is *not* helpful for assessing acute kidney injury (AKI).
  - There is controversy about its interpretation in elderly people. An eGFR of more than 45 mL/m^2 in an old person without proteinuria is not associated with an adverse renal prognosis.
  - For black populations in the United States, the laboratory reading should be multiplied by 1.2.

Table 11.1 Stages of chronic kidney disease

Stage	Description	eGFR (mL/min/1.73m²)	Albuminuria stage# and risk of progression^
G1	Normal or high kidney function with kidney damage*	≥ 90	A1: Low risk A2: Moderate risk A3: High risk
G2	Mild reduction in kidney function with kidney damage*	60–89	A1: Low risk A2: Moderate risk A3: High risk
G3a	Mild to moderate reduction in kidney function	45–59	A1: Moderate risk A2: High risk A3: Very high risk
G3b	Moderate to severe reduction in kidney function	30–44	A1: High risk A2: Very high risk A3: Very high risk
G4	Severe reduction in kidney function	15–29	A1–A3: Very high risk
G5	Kidney failure (end-stage kidney disease)	< 15 or on dialysis	A1–A3: Very high risk Likely requires dialysis or transplant
G5D	Kidney failure treated with dialysis	Variable	Patient on dialysis treatment
T	Kidney transplant recipient	Variable	Monitor for transplant function

eGFR = estimated glomerular filtration rate
#Albuminuria categories (urine albumin-to-creatinine ratio, uACR):
• A1: Normal to mildly increased (< 3 mg/mmol)
• A2: Moderately increased (3–30 mg/mmol)
• A3: Severely increased (> 30 mg/mmol)
^Risk of CKD progression: Risk is determined by both eGFR and albuminuria together. A patient with normal eGFR (G1–G2) but severe albuminuria (A3) is at HIGH risk, not low risk.
*Kidney damage markers: albuminuria, abnormalities of urine sediment or electrolytes, histological or structural(imaging) abnormalities, or history of kidney transplantation

Table 11.2 Causes of CKD (people who are on dialysis or have had a transplant)

1. Diabetes mellitus: 40%
2. Glomerulonephritis: 18%
3. Hypertension: 11%
4. Hereditary (including polycystic kidneys): 7%
5. Tubulointerstitial: 8%
6. Analgesic nephropathy
7. Uncertain

Table 11.3 Risk factors for progression of CKD

1. Low birth weight (fewer than normal nephrons to start with)
2. Hypertension
3. Acute kidney injury
4. Proteinuria
5. Smoking
6. Hyperuricaemia
7. An increase in glomerular pressure (pregnancy, obesity, diabetes)

  - This correction is not valid for Australian Aboriginal and Torres Strait Islander people. The estimation of eGFR by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula, which is in common use, seems accurate for Aboriginal and Torres Strait Islander and Māori populations.
  - The measurement of eGFR is not accurate in pregnancy.
  - In people with low muscle mass the number may be an overestimate of renal function.
- The eGFR helps to determine when preparation for dialysis should begin (but gives no information about the cause of the renal dysfunction). For example, the Initiating Dialysis Early and Late (IDEAL) study suggested that dialysis should commence when the eGFR is 7–10 mL/min.[1]
- Arteriovenous fistulas for haemodialysis are not usually ready to use for at least 3 months after the surgery. Nephrologists prepare for vascular access surgery when the eGFR is about 15–20 mL/min.
- A pre-emptive renal transplant is sometimes performed when the eGFR is about 15 mL/min.
- It is important to note that eGFR falls with ageing. The rate of fall is slower for females (≈0.7 mL/min/year) than for males (≈1 mL/min/year). The falls start at approximately the age of 35. Hence, when assessing an 85-year-old man, you could expect that his eGFR should be about 50 mL/min. If he has had a nephrectomy, he should have about 25 mL/min eGFR.
- If there is discrepancy between the calculated and expected eGFR, the nephrologist or astute candidate will suspect the presence of causes of reduction of eGFR other than ageing.
- The eGFR is not accurate in patients with limb amputation because of their loss of muscle mass.
- The majority of kidney donations are from deceased donors (76%); the rest are from live donors.
- Chronic haemodialysis can be performed in hospital (22%), in a satellite unit (54%) or at home (24%).
- About 20% of dialysis patients are having peritoneal dialysis (PD).

The history

In many cases, the abnormal renal function has been detected on routine blood tests before symptoms have occurred.

QUESTIONS REGARDING SYMPTOMS, DIAGNOSIS AND AETIOLOGY

1. **Early symptoms** of renal failure are most often non-specific but may include:
   - nocturia
   - lethargy
   - loss of appetite
   - fluid retention (ankle oedema, dyspnoea, orthopnoea).

   Severe CKD (GFR < 10–20 mL/min/1.73 m^2) can cause:
   - pericarditis
   - serositis
   - pruritus
   - encephalopathy

[1]B A Cooper, P Branley, L Bulfone et al. A randomized, controlled trial of early versus late initiation of dialysis. *The New England Journal of Medicine* 2010 Aug 12; 363(7):609–19.

- gastrointestinal bleeding
- uraemic neuropathy.

These symptoms and signs are rare because treatment is almost always begun with RRT in order to prevent them.

2. **Precipitation factors.** Patients are sometimes diagnosed following an episode of haematuria or loin pain. A first episode of overt renal failure may have been precipitated by a further insult, such as:
   - use of drugs such as NSAIDs, trimethoprim or, less commonly now, aminoglycoside administration
   - use of radiocontrast injections
   - infection
   - use of angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs) (if there is bilateral renal artery stenosis)
   - dehydration
   - anaemia.
3. **Asymptomatic patients.** Many patients are asymptomatic and have a family history. Sometimes haematuria or proteinuria has been detected during a routine or insurance medical examination or during pregnancy.
4. **Glomerulonephritis diagnosis.** If the patient has had a diagnosis of glomerulonephritis (Table 11.4), determine whether there is a history of:
   - proteinuria
   - haematuria
   - oliguria
   - oedema
   - sore throat
   - sepsis
   - rash
   - haemoptysis
   - renal biopsy.

Run through the various causes listed in the Hint box below with the patient or ask "Have you been told what the cause of your kidney trouble is?" This question may save a lot of time and trouble.

Table 11.4 Classification of glomerulonephritis (GN)

PRIMARY	
Diffuse	**Focal**
1. Minimal change disease (MCD): most common cause of nephrotic syndrome in children 2. Membranous GN (Box 11.2) 3. Proliferative • Post-streptococcal (and after other infections) • Mesangiocapillary • Crescentic • Mesangioproliferative	1. IgA nephropathy (Box 11.1) 2. Focal segmental glomerulosclerosis (FSGS): most common cause of nephrotic syndrome in adults (Box 11.3); *note: MCD and primary FSGS increasingly viewed as spectrum of immune-mediated NS*
GLOMERULONEPHRITIS AS PART OF A SYSTEMIC DISEASE	
1. Systemic lupus erythematosus (SLE) 2. Granulomatosis with polyangiitis (GPA) 3. Polyarteritis nodosa (PAN) 4. Anti-glomerular basement membrane (Goodpasture) 5. Henoch-Schönlein purpura	6. Infective endocarditis 7. Cryoglobulinaemia ± hepatitis C 8. Myeloma 9. Diabetes mellitus 10. Haemolytic uraemic syndrome (HUS)

Box 11.1 IgA nephropathy – associations

- HIV infection
- Chronic liver disease
- Inflammatory bowel disease
- Coeliac disease

Box 11.2 Causes of membranoproliferative glomerulonephritis

1. Hepatitis C
2. Autoimmune diseases
3. Indolent infections (malaria, syphilis)
4. Essential cryoglobulinaemia
5. Malignancies
6. Drugs: penicillamine, NSAIDs, anti-TNF drugs
7. Mercury or gold poisoning

NSAID = non-steroidal anti-inflammatory drug; TNF = tumour necrosis factor.

Box 11.3 Causes of focal segmental glomerulosclerosis

- Primary
- Familial
- HIV infection
- Morbid obesity
- Heroin use
- Reflux nephropathy

T&O'C HINTS

Ask yourself after you have taken the history: "What is the cause of this patient's CKD?" Consider the following.

1. **Glomerulonephritis.** Has the patient had a kidney biopsy? Was there any specific change in therapy following the biopsy? For example, in IgA nephropathy, if there are significant chronic changes and reduced kidney function, data suggest there is little point in starting high-dose steroids. On the other hand, if there are no severe chronic changes (eGFR > 80 mL/min and proteinuria < 0.5 g/day) a 6-month trial of prednisolone (≈1 mg/kg/day) may be indicated to attempt to induce a remission, if supportive therapy with ACEIs or ARBs hasn't helped.
2. **IgA nephropathy.** This is associated with intermittent macroscopic haematuria, synpharyngitic haematuria (typically following soon after a mild upper respiratory tract infection) or persistent microscopic haematuria. Even so, it is important to consider other causes of haematuria (e.g. bladder transitional cell carcinomas, kidney stones).
3. **Diabetic nephropathy.** This is now the most common cause of CKD in Australia. These patients don't usually undergo a renal biopsy. However, it may be indicated in patients with diabetes where non-diabetic renal disease is suspected; for example,

those without micro- and macrovascular complications of diabetes, or where the duration of diabetes is short.

4. **Hypertensive nephropathy.** This is an unusual diagnosis. However, most patients with CKD are hypertensive and improving blood pressure (BP) control is often a mainstay of therapy aimed at slowing the rate of progression of CKD of any cause. Because of its place in therapy, detailed knowledge of the patient's BP and its management is crucial. Trials have suggested that angioplasty for atheromatous renal artery stenosis is no more effective than medical therapy; similarly, despite early enthusiasm, renal artery sympathectomy (denervation) has not proven to be effective in most patients with resistant hypertension.
5. **Analgesic nephropathy.** This is now a truly rare condition but patients very occasionally turn up at exams.
6. **Family history.** Don't forget to ask about a family history of kidney disease. Clearly, it is important not to miss polycystic kidney disease. However, diabetes, hypertension, reflux nephropathy and various forms of glomerulonephritis (GN) can also have an inherited basis. This can be important even when discussing the possibility of living related donors for kidney transplantation, for example.
7. **Interstitial nephritis.** Acute interstitial nephritis can be a result of drug allergy, an immune reaction or an infection (Table 11.5). It is an important cause of chronic interstitial nephritis and CKD.
8. **Unknown cause.** This group of patients usually present with chronic changes in their kidneys or small shrunken kidneys that cannot be safely biopsied. Serological tests for causes of kidney diseases are negative.

Table 11.5 Causes of acute interstitial nephritis

DRUG-INDUCED ACUTE INTERSTITIAL NEPHRITIS (DI-AIN) Proton pump inhibitors, penicillin, NSAIDs, gadolinium contrast material
IMMUNE Transplant rejection, autoimmune nephritis
INFECTIONS TB, bacterial pyelonephritis, leptospirosis
TOXINS Mushrooms, myeloma light chains

Ascertain treatment details (e.g. antihypertensives, immunosuppressives, antiplatelet therapy, dialysis).

T&O'C HINT

Distinguish the nephrotic syndrome (Table 11.6) from the nephritic syndrome (Table 11.7) and the types of glomerulonephritis (GN) (see Table 11.4).

- **Nephrotic** – protein leakage across the glomeruli, "PALE":
  - **P**roteinuria (severe protein loss in urine > 3.5 g/24 hours, urine is frothy with fatty casts)

Continued

- **A**lbumin low (hypoalbuminaemia < 30 g/L)
- **L**ipids high (hyperlipidaemia)
- o**E**dema.

- **Nephritic** – inflammation or injury within the glomeruli allowing protein, red blood cells and white blood cells into the renal tubule; "PHARAOH":
  - **P**roteinuria (mild, < 3.5 g/24 hours)
  - **H**aematuria (prime characteristic; "smoky" or "coca-cola" coloured urine)
  - **A**zotemia (raised urea/creatinine; mildy reduced eGFR)
  - **R**BC casts
  - **O**liguria
  - **A**ntibodies
  - **H**ypertension

Table 11.6 The nephrotic syndrome

CLINICAL FEATURES
1. Proteinuria (> 3.5 g/24 h)
2. Hypoalbuminaemia (serum albumin < 30 g/L)
3. Oedema
4. Hyperlipidaemia (increased LDL and cholesterol levels)

CAUSES
1. **Primary (80%)**
   Idiopathic membranous glomerulonephropathy is the most common cause in adults over 40 years of age. Other primary causes include focal glomerular sclerosis, membranoproliferative glomerulonephritis and minimal change nephropathy
2. **Secondary**
   Systemic disease: diabetes mellitus (the most common by far), SLE, Hodgkin's disease (minimal change), solid tumours (membranous), amyloid, multiple myeloma
   Infection: hepatitis B (membranous), HIV (IgA nephropathy, collapsing focal sclerosis), infective endocarditis
   Drugs: D-penicillamine, probenecid, non-steroidal anti-inflammatory drugs, heroin

Note: Renal vein thrombosis is a complication and rarely a cause of the nephrotic syndrome.

Table 11.7 Causes of nephritic syndrome

ABNORMALITY	CAUSE OF NEPHRITIC SYNDROME (%)	COMPLEMENT (C) FINDINGS
IgA nephropathy	25	Normal
Lupus (SLE)	20	Low C3 and C4
Pauci-immune crescentic GN	20	Normal
Membranoproliferative GN	10	Low C3 or C4 or both
Thrombotic microangiopathy	5	Low C3 sometimes
Postinfectious GN	5	Low C3
Anti-glomerular basement membrane disease (Goodpasture)	3	Normal
C3 glomerulonephropathy	< 1	Low C3

1. **Diabetic nephropathy.** Ask about other complications and therapy. An ACEI or ARB (but not both) is preferred for all cases with diabetic nephropathy. The creatinine should be monitored after treatment is begun. An increase in serum creatinine of less than 30% is acceptable and may indeed indicate a degree of renal protection; reduced glomerular pressure increases the creatinine but protects the kidneys in the long run. A rise in creatinine of more than 30% usually means the drug should be stopped. Patients with very low eGFRs (< 20 mL/min) should have the drug stopped, but if there is no improvement in eGFR it can usually be restarted.
2. **Polycystic kidney disease (PKD).** Ask about family history (the condition is usually autosomal dominant [APKD]), how the disease was diagnosed, haematuria, polyuria, loin pain, hypertension, headache, subarachnoid haemorrhages and visual disturbance (intracranial aneurysm).

   Also ask about deafness and a history of persistent haematuria (hereditary nephritis – Alport's syndrome).

   Has the patient been treated with tolvaptan? This drug is PBS approved and has been shown to slow disease progression by reducing cyst growth.
3. **Reflux nephropathy.** Ask about childhood renal infections, cystoscopy, operations, treatment (e.g. regular antibiotics) and enuresis.
4. **Hypertensive nephropathy.** Ask about how the disease was diagnosed, duration and control of hypertension, treatment and compliance with medication, renal angiography and family history.
5. **Connective tissue disease.** Think especially of systemic lupus erythematosus (SLE) and scleroderma.
6. **Long-term prognosis.** Find out whether the patient is aware of this. If he or she is not yet on dialysis, has this been discussed? Is the patient likely to be eligible for dialysis or the transplant list? For some patients, conservative therapy (i.e. no dialysis) may be recommended. In some cases a pre-emptive transplant may have been discussed.
7. **Timeframe.** Ask when the *underlying condition* was *diagnosed* and how it is being *treated*. The progression to end-stage kidney disease may be rapid or very prolonged.

T&O'C HINT

Autosomal dominant polycystic kidney disease

The extrarenal manifestations of autosomal dominant polycystic kidney disease (ADPKD) include:

- liver cysts/hepatomegaly
- pancreatic cysts
- splenic cysts
- thyroid cysts
- seminal vesicle cysts
- intracranial cerebral aneurysms
- hypertension
- diverticular disease
- hernias (can occur as a result of the increased size of intra-abdominal organs; it may be a problem for use of peritoneal dialysis (PD) and an indication for nocturnal PD).

QUESTIONS REGARDING MANAGEMENT

See Table 11.18.

1. Conservative management
   a. Ask about:
      - follow-up
      - medications (e.g. ACE, SGLT-2 inhibitor, NSAIDs)
      - diet; low potassium and avoidance of high protein but not otherwise restricted because of the risk of weight loss associated with loss of appetite
      - salt and water allowance
      - investigations performed (particularly renal biopsy)
      - whether erythropoietin has been given subcutaneously (or IV in dialysis patients) in an attempt to elevate the haemoglobin
      - medications to control potassium – sodium polystyrene sulfonate (Resonium) or more recently patiromer.
   b. Has the patient been advised to restrict protein intake? There is controversy about the value of protein restriction in delaying end-stage renal failure. Patients with nephrotic syndrome should be much less restricted. The concern about protein restriction is that it leads to more rapid loss of muscle mass without much delay in end-stage renal failure.
   c. Has potassium restriction been recommended? Potassium accumulates in patients with severe CKD and intake is often restricted to 70 mmol/day (Table 11.9). Has the patient been told about food that should be avoided because of its potassium content?
   d. What effect have the disease and the dietary and other restrictions had on the patient's quality of life and his or her family?

Table 11.8 Principles of management of CKD

1. Delay progression (Box 11.4)
2. Fluid intake and diet
3. Anaemia (CKD 4): iron therapy first (IV in advanced CKD and dialysis), then erythropoietin (new oral formulations are being tried)
4. Acidosis (CKD 5)
5. Phosphate/calcium/bones (CKD 4–5)
6. Cardiovascular risk reduction
7. Consider vascular access (usually when eGFR 20 mL/min)
8. Plan treatment in conjunction with patient and family. Avoid starting dialysis late. Be prepared for the next steps.

Box 11.4 Delaying progression of CKD

1. Control BP
2. Review medications (NSAIDs and sometimes ACEIs and ARBs)
3. Stop smoking
4. Avoid AKI (dehydration, infection, major surgery, consider suspending ACEI or ARB treatment)
5. Inhibit renin angiotensin system (ACEI, ARB)
6. Manage proteinuria (ACEI, BP)
7. Add SGLT-2 inhibitor (dapagliflozin: reduces proteinuria, delays decline in GFR)

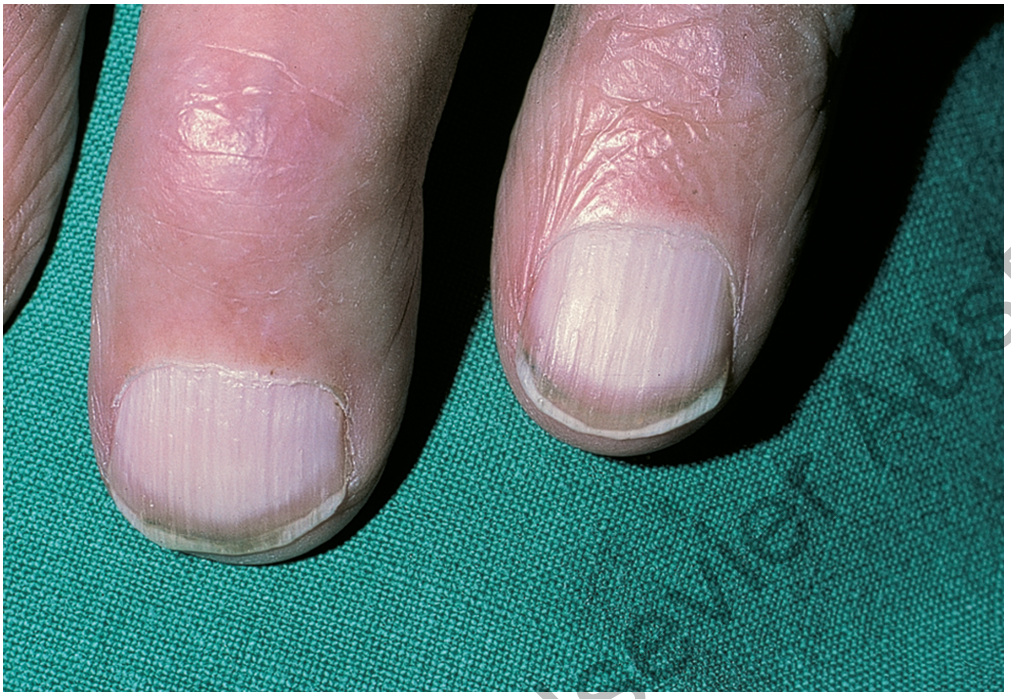

Figure 11.1 Terry's nails in chronic kidney disease. There is proximal pallor with distal brownish colour.

G M White, N H Cox (eds). *Diseases of the skin: a color atlas and text*, 2nd edn. St Louis, Mosby, Elsevier, 2006, with permission.

nodes (lymphoma, cytomegalovirus or other infections if the patient is immunosuppressed), ascites (cannot be diagnosed in presence of PD) and femoral bruits and pulses.

8. Urine. For blood, protein, specific gravity, pH, glucose, urine microscopy and examination of the urinary sediment for casts.
9. Legs. Oedema, bruising, pigmentation, scratch marks, peripheral neuropathy, vascular access and myopathy.
10. Back. Bone tenderness and sacral oedema.
11. Fundoscopy. Laser treatment for proliferative retinopathy.

Investigations

1. Determine renal function.
   a. Glomerular filtration rate (GFR): creatinine clearance (creatinine clearance levels of <10 mL/min are considered indications for dialysis) and plasma creatinine/urea level; the eGFR is routinely calculated by laboratories and the patient may know these results.
   b. Tubular function: plasma electrolyte levels, urine specific gravity and pH, glycosuria, serum potassium, serum phosphate and uric acid, aminoaciduria, serum calcium and plasma albumin levels.
   c. Urine analysis and urinary protein excretion (protein-to-creatinine ratio), 24-hour urinary protein.
2. Determine renal structure.
   a. Ultrasound: renal size and symmetry, signs of obstruction; small kidneys (suggest chronic disease) and the presence or absence of ureteric jets – indicating patent ureters.

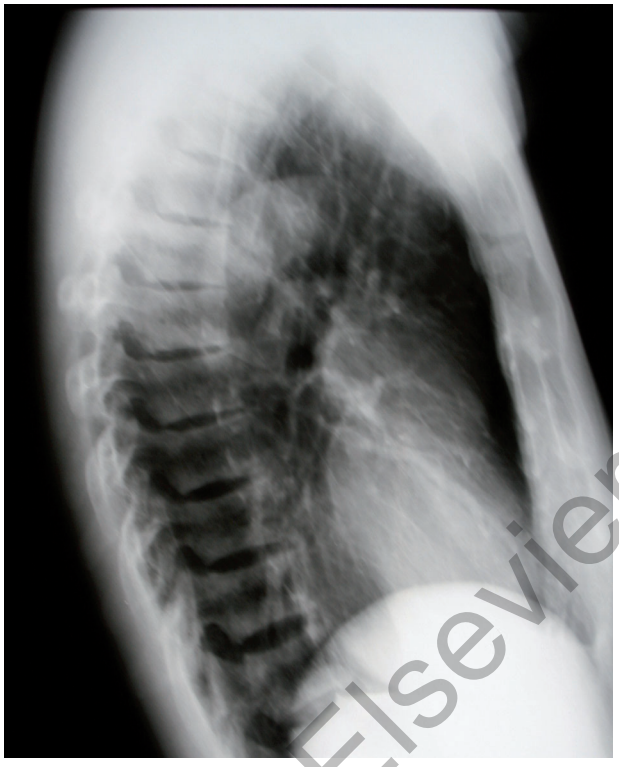

Figure 11.2 Spine X-ray of a patient with CKD showing alternating dense and radiolucent bands – "rugger jersey spine".

Figure reproduced courtesy of The Canberra Hospital.

4. Normalise the calcium and phosphate levels with diet, phosphate binders or calcitriol. Non-calcium-based phosphate binders (e.g. sevelamer) improve mortality compared with calcium-based ones. Treatment of secondary hyperparathyroidism with the drug cinacalcet has not been shown to improve mortality or risk of vascular events in dialysis patients.
5. Restrict dietary protein. However, although this may delay slightly the need for dialysis, it leads to wasting and protein malnutrition. It is no longer universally recommended.
6. Assess and treat sexual dysfunction.
7. Dialyse when indicated (see below).
8. Consider transplantation.

WHEN TO START DIALYSIS

It is very important to consider that patients may not be suitable for dialysis or may have unrealistic expectations of the benefits of dialysis. Remember that there is no benefit in starting dialysis early, and that patients over 75 years of age who have significant co-morbidities have reduced survival with dialysis compared with conservative treatment. Patient and family education about dialysis options and non-dialysis management is necessary in a timely manner for all patients with worsening CKD.

Not all end-stage CKD patients should be dialysed. Consider:

1. age and co-morbidities
2. conservative treatment

Table 11.15 Causes of renal allograft rejection

HINT: consider surgical problems, thrombophilia or SLE.

TIMEFRAME	CAUSE	POSSIBLE REASONS
Very early (hours to days)	Renal artery or vein thrombosis	Surgical problems Thrombophilia or SLE
	Ureteric leak	Small bladder
	Delayed graft function	Long graft ischaemia time, older donor, elevated tacrolimus level
	Hyperacute rejection	HLA mismatch Previous transplant Pre-formed anti-HLA antibodies
Early (weeks)	Acute rejection, non-adherence to treatment or inadequate immunosuppression	HLA mismatch Previous transplant Pre-formed anti-HLA antibodies
Months	Renal artery stenosis, BK virus infection and nephropathy	Use of ureteric stent, intense immunosuppression, disease of donor kidney, damage to graft during harvesting
Years	Chronic allograft injury (usually mediated by antibodies)	Insufficient immunosuppression, non-adherence Previous acute rejections
At any time	Cyclosporin or tacrolimus toxicity	High doses, serum levels not monitored Concurrent use of P450 cytochrome-inhibiting drugs
	Infection	
	Recurrence of original kidney disease (e.g. focal glomerulosclerosis), minimum change GN (early) IgA nephropathy or membranous GN (later)	Recurrence in a previous transplant

GN = glomerulonephritis; HLA = human leucocyte antigen; SLE = systemic lupus erythematosus.

Possible lines of questioning

1. Did *this* patient have complications during renal transplant surgery? How were they managed?
2. Has *this* transplant patient had rejection episodes? How were they diagnosed and treated?
3. What changes to immunosuppressive treatment have been required during the period since *this* patient's transplant? Is the current regimen optimal?
4. What complications of immunosuppressive treatment have occurred?

Chapter 15

The short case

You see, but you do not observe.
Sir Arthur Conan Doyle (1859–1930)

The short case is a test of the candidate's ability to examine a patient smoothly, confidently and accurately. There is rarely the opportunity to go back and repeat the examination. It takes a long time to get used to being watched critically while examining. This is why it is important to practise short cases of every conceivable type so that the physical examination is performed automatically in the correct way. While proceeding, the candidate should be consciously synthesising the results, not trying to remember what to do next.

There is always considerable discussion at examiners' meetings about the usefulness of the short-case exam. It is obviously an artificial situation unlike normal clinical practice where more history is almost always available from the patient before the examination begins. It is also true that complete examinations of this type are rarely performed in practice. In most specialties physicians use short cuts when they examine patients. However, working out a satisfactory short cut relies on knowing how to perform a complete examination.

The other objection to the routine of the short case is that many of the clinical signs examiners expect candidates to find are of doubtful value. Perhaps only signs with good evidence of usefulness should be looked for. The problem with this approach is that examiners examining outside their own specialty are unlikely to have clear views on the objective value of signs and are more likely to expect candidates to do the examination they are used to seeing. It would be a courageous candidate who refused on principle to look for signs he or she considered to be of no value. Perhaps in the future patients will be assessed for only evidence-based signs. This might make the short case quite brief.

In fact, the real point of the short case is to make candidates practise and become familiar with (and expert in) systematic examination techniques that form the basis of the short cut focused examinations they use throughout their careers.

Although examiners have expectations about the form of the short-case examination, there is no single correct method and what is presented in Chapter 16 is only suggested as an approach that should be used as a basis for your own method.

Presentation

Before presenting the findings, listen closely to the examiners' instructions. Candidates will often be asked "What did you find?", at which point they are expected to describe the relevant signs first and then comment on possible causes. Sometimes candidates will be asked "What is your diagnosis?", at which point they are expected to give a diagnosis or differential diagnosis first and then list the signs supporting the contention.

T&O'C HINT

Formulate your diagnosis and differential diagnosis *based on the individual in front of you.*

Using a formulaic presentation of your findings might give you more time to think, but can be intensely irritating for the examiner if this is the fourth time they have heard "Mr Smith is an elderly man lying comfortably in bed", and especially if the patient is no older than the examiners and is obviously breathless and not comfortable.

One useful method of presentation is to first repeat the examiners' introduction briefly, and then give the relevant findings, followed by the provisional diagnosis. For example: "I was asked to examine Mr Jones, a 60-year-old man who has had problems with dyspnoea. On examination of his cardiovascular system, I found ..." When describing the signs it is probably easiest to present them in the order they were looked for (e.g. for the cardiovascular system describe pulse rate, then blood pressure, then jugular venous pressure). It is important to state all the positive signs and the important negative ones. Be definite about each sign mentioned, or do not mention the sign at all. There is no place for expressions such as "slightly asymmetrical" or "minor".

T&O'C HINT

In neurological examinations, don't rush to undertake sensory testing, which is often frustrating and less reliable. Leave the sensory examination to the end if at all possible.

Confidence is critical to success in the short cases. Do not lose confidence if you make a minor error; just continue – the examiners may not even have noticed.

A short differential diagnosis is usually expected, even if the diagnosis is obvious. For example, a patient with fasciculation plus upper and lower motor neurone signs in the legs and no sensory loss almost certainly has motor neurone disease, but a non-metastatic manifestation of carcinoma must be considered. Always mention common diseases before rare ones and always consider the patient's age and sex. Never reel off any old list; the differential diagnosis must be tailored to the particular patient. Sometimes patients will have signs of two different problems. This should not be ignored. For example, a patient with proximal muscle weakness as a result of polymyositis may have unrelated Dupuytren's contractures.

After presentation of the signs, a few minutes or more are set aside for discussion. The examiners are not encouraged to take the candidate back for a second look at a

sign, as this can be extremely unsettling for the candidate and perhaps not fair. However, this does happen occasionally and it is best to think of it as a genuine second chance.

T&O'C HINT

A redirect represents a genuine second chance – grab hold of the opportunity. There are no tricks in the examination.

Understanding the role of the examiners

From the examiners' point of view, the candidate who is completely wrong presents a problem. This can occur because he or she has not read the stem properly; for example, when a request to examine the lower cranial nerves leads a candidate to begin to test visual acuity. Sometimes the examination depends on a spot diagnosis. For example, for an obvious acromegalic patient the stem might be: "This man has noticed some changes in his hands. Have a look at his face, examine the hands and go on from there". The risk here is that the acromegaly is not recognised and the candidate decides the diagnosis is, say, rheumatoid arthritis. The examination and discussion will then have nothing to do with what the examiners had expected and prepared for.

If the candidate's mistake is recognised early on, the examiners may attempt to redirect the examination. This can be surprisingly difficult. Some candidates persist in continuing the way they began, despite strong hints or even direction from the examiners. This is presumably because they think an attempt is being made to trick them. This never happens.

T&O'C HINT

If your diagnosis is completely incorrect, a good discussion won't usually help you.

Sometimes the examination seems to be going well and then the candidate comes out with a completely wrong diagnosis. This makes the examiners' prepared discussion unusable. In this case the examiner will probably attempt to continue the discussion along the lines the candidate has begun. For example, the candidate appears to have examined a patient with small muscle wasting of the hands satisfactorily but then, against all the evidence, decides the problem is rheumatoid arthritis. The examiners may ask what was found that led to the diagnosis and were there any alternative possibilities, but if no alternatives can be extracted from the candidate then they will allow a discussion of rheumatoid arthritis.

This problem usually occurs when a candidate has decided on a diagnosis before looking at the patient. Deciding that because the stem was "'examine the hands', therefore this 'can only be rheumatoid'" must be avoided.

One examiner will have introduced the patient and repeated the stem. This is likely to be the lead examiner. In many cases that examiner will conduct the discussion. There may or may not be an opportunity for the other examiner to ask some questions at the end. This may be a sign that the first one has run out of questions. This doesn't really tell you whether things are going very well or very badly.

Investigations

Relevant X-rays or an ECG may be shown to the candidate. Some diagnostic and therapeutic aspects are likely to be discussed in the short case. If a candidate has done well in a case and there are a few minutes left for extra questions, the score can only improve.

T&O'C HINT

If you have done well, in the last few minutes of discussion your score can only go up, not down. So don't worry if the depth becomes overwhelming; press on talking about the problems in a mature, sensible fashion.

Short-case selection

Consider the short-case lottery as you prepare for the exam:

- cardiovascular ≈ 25%
- neurology ≈ 25%
- respiratory ≈ 15%
- rheumatology ≈ 10%
- gastroenterology ≈ 6%
- renal ≈ 5%
- haematology ≈ 5%
- endocrine ≈ 3%.

The average frequencies of cardiovascular short cases are as follows.

- Most common: mitral regurgitation, aortic stenosis, aortic regurgitation, hypertrophic cardiomyopathy, aortic stenosis/aortic regurgitation (each about 10%).
- The rest:
  - pulmonary hypertension/tricuspid regurgitation
  - prosthetic valves, mitral valve prolapse, mitral stenosis/mitral regurgitation
  - pulmonary regurgitation.

The organising team is asked to find a variety of cases for each candidate, but when a patient drops out or is rejected by the examiners these arrangements can go astray. One candidate got three cases of interstitial lung disease in her exam! However, all types still crop up and a candidate must try to prepare for most possibilities. It is also true that the more straightforward the case, the higher is the standard of examination that will be expected, and vice versa. Trick cases are deliberately avoided.

Understanding the examiners' thinking

The value of some traditional clinical signs is now being questioned as *evidence-based* approaches to clinical examination help establish the validity and utility of signs. There is much work still to be done in this area, but an understanding of the value of signs is increasingly important. A tactful approach may be needed with the examiners to prevent any resentment at the candidate's failing to look for a traditional sign that is a particular favourite of theirs.

SIX GOLDEN RULES FOR THE SHORT CASES

1. Do *everything* properly when you examine the patient: *never* take short cuts.
2. *Think* and *synthesise* as you examine the patient: be alert.
3. *Never* make up signs and *never* ignore signs because they don't fit neatly together.
4. Always *be sure of your facts* when presenting: it's better to say that you don't know than to guess.
5. Always show consideration for the patient and *never* cause the patient pain.
6. Wash your hands before and after the case.