ACUTE KIDNEY INJURY | RESPIRATORY INFECTIONS | VERTIGO | CVD RISK

Best Practice

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Issue 46 September 2012

A delicate balance: managing vertigo in general practice

bpac nz
better medicine

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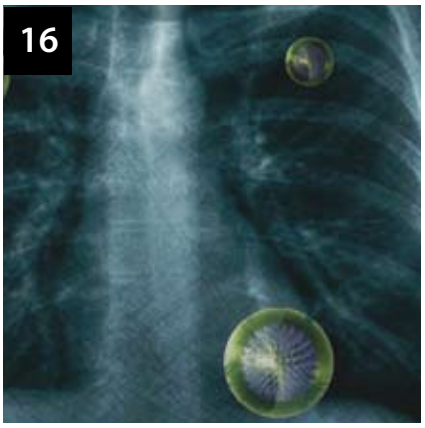
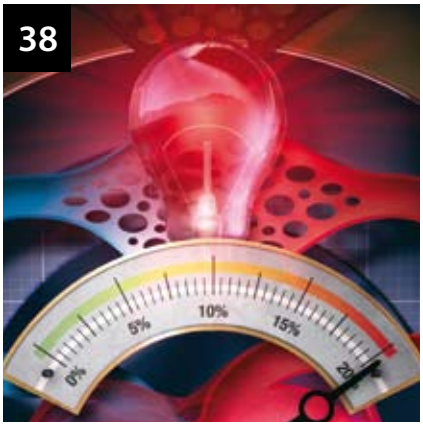
CONTENTS

Issue 46 September 2012



CONTENTS

Issue 46 September 2012



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Funding changes to **diabetes management products** announced

Chronic Care Module

The new ***bestpractice*** **Decision Support** Chronic Care Module combines cardiovascular risk assessment, chronic kidney disease and diabetes management all in one simple intuitive decision support form.

The form highlights the stage of chronic kidney disease and automatically calculates the rate of decline for the last twelve months and the last five years. When the rate of decline is 5 mL per min and 10 mL per minute or greater respectively the module suggests nephrology referral. In the presence of diabetes the module dynamically incorporates the NZGG June 2011 guidance and highlights the risk of diabetes related complications. A goal driven care plan can also be enacted, pre-populated with the information from the module.

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Medicine interactions: using the New Zealand Formulary

The New Zealand Formulary uses **Stockley's Drug Interactions** – one of the world's most comprehensive and authoritative international reference sources on drug interactions.

Medicine interactions usually result from potentiation or antagonism of one drug by another. Medicine selection should aim to minimise interactions. If it is necessary to prescribe a potentially serious combination of medicines, patients should be informed and monitored appropriately.

The New Zealand Formulary (NZF) presents two options for obtaining information about medicine interactions:

1. Stockley's Interaction Alerts
2. British National Formulary (BNF) interaction summaries (synonymous with Appendix 1 in the BNF)

Individual drug monographs in the NZF contain links to the relevant section of Stockley's Interaction Alerts, which rates information according to three categories, and the relevant BNF interaction summary. The alerts give additional information to assist in the management of an interaction. The BNF interaction summaries provide a more general interaction overview. The Stockley's Interaction Alerts database is also used for the NZF interaction checker.

The clinical notes section of the NZF contains links to BNF interaction summaries for the associated drug group or class. This provides an overview for general reference.

It is important that any adverse medicine interactions are reported to the New Zealand Pharmacovigilance Centre: **http://carm.otago.ac.nz**

How to interpret Stockley's Interaction Alerts

The alerts are rated using three separate categories:

- **Action:** this describes whether or not any action needs to be taken to accommodate the interaction. This category ranges from "avoid" to "no action needed".
- **Severity:** this describes the likely effect of an unmanaged interaction on the patient. This category ranges from "severe" to "nothing expected".
- **Evidence:** this describes the weight of evidence behind the interaction. This category ranges from "extensive" to "theoretical".

How to use the interactions checker

To launch the interactions checker, select the interactions tab at the top of the page. Start typing the name of the medicine in the search bar and select the appropriate medicine from the dropdown list. You can enter all of your patient's medicines plus any others you may want to check. Food and herbal products can also be checked (where available within the database). Compound preparations may also be entered.

Once two medicines have been entered, any potential interactions will automatically appear on the screen with an explanation of the interaction (Figure 1). The interactions are sorted by a simple "traffic light" colour-coding system (there is a key to the colours at the top right of the window). A short description of what action should be taken is also displayed. Hovering over the text in the severity or evidence columns gives a description of what these classifications are.

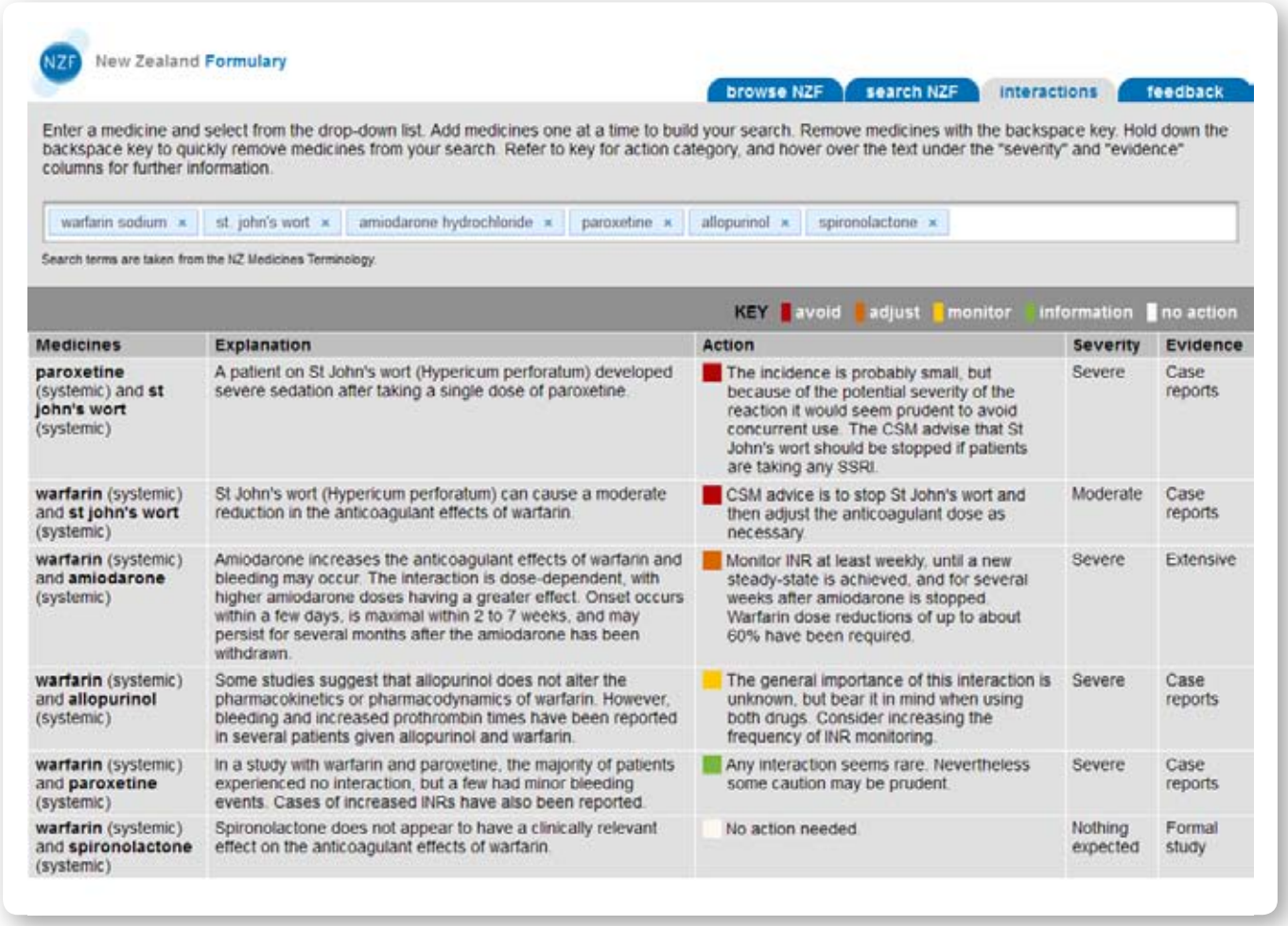


Figure 1: NZF interactions checker screen shot

Definitions of severity and evidence:

Severity	
Severe	Interactions that could totally incapacitate a patient or result in either a permanent detrimental effect or a life-threatening event.
Moderate	Interactions that could result in an effect that may either cause considerable distress or partially incapacitate a patient. These interactions are unlikely to be life-threatening or result in long-term effects.
Nothing expected	Interactions that are unlikely to result in an effect, or for drugs pairs where no interaction occurs.
Evidence	
Theoretical	Based on a theoretical interaction or lack of interaction. This information may have been derived either from in vitro studies involving the drug in question or based on the way other members of the same group act.
Case reports	Based either on a single case report or a limited number of case reports. No trials appear to have been conducted.
Formal study	Based on formal study. This may be one small or medium size study, or several small studies. The studies may or may not be supported by case reports.
Extensive studies	Based on numerous small or medium size studies or several large studies. The information is usually supported by case reports.

Types of interactions

Pharmacodynamic interactions

These are interactions between drugs which have similar or antagonistic pharmacological effects or adverse effects. They may be due to competition at receptor sites, or occur between drugs acting on the same physiological system. They are usually predictable from a knowledge of the pharmacology of the interacting drugs; in general, an interaction demonstrated with one drug is likely to occur with related drugs. N.B. Pharmacodynamic interactions can be compounded by two or more drugs with similar actions.

Pharmacokinetic interactions

These occur when one drug alters the absorption, distribution, metabolism or excretion of another, thus increasing or reducing the amount of drug available to produce its pharmacological effects. Individual variation in metabolic capacity, genotype, organ function and other factors result in a degree of unpredictability and many pharmacokinetic interactions do not affect all patients taking the same combination of drugs. Pharmacokinetic interactions occurring with one drug cannot be assumed to occur with related drugs unless their pharmacokinetic properties are known to be similar.

Relative importance of interactions

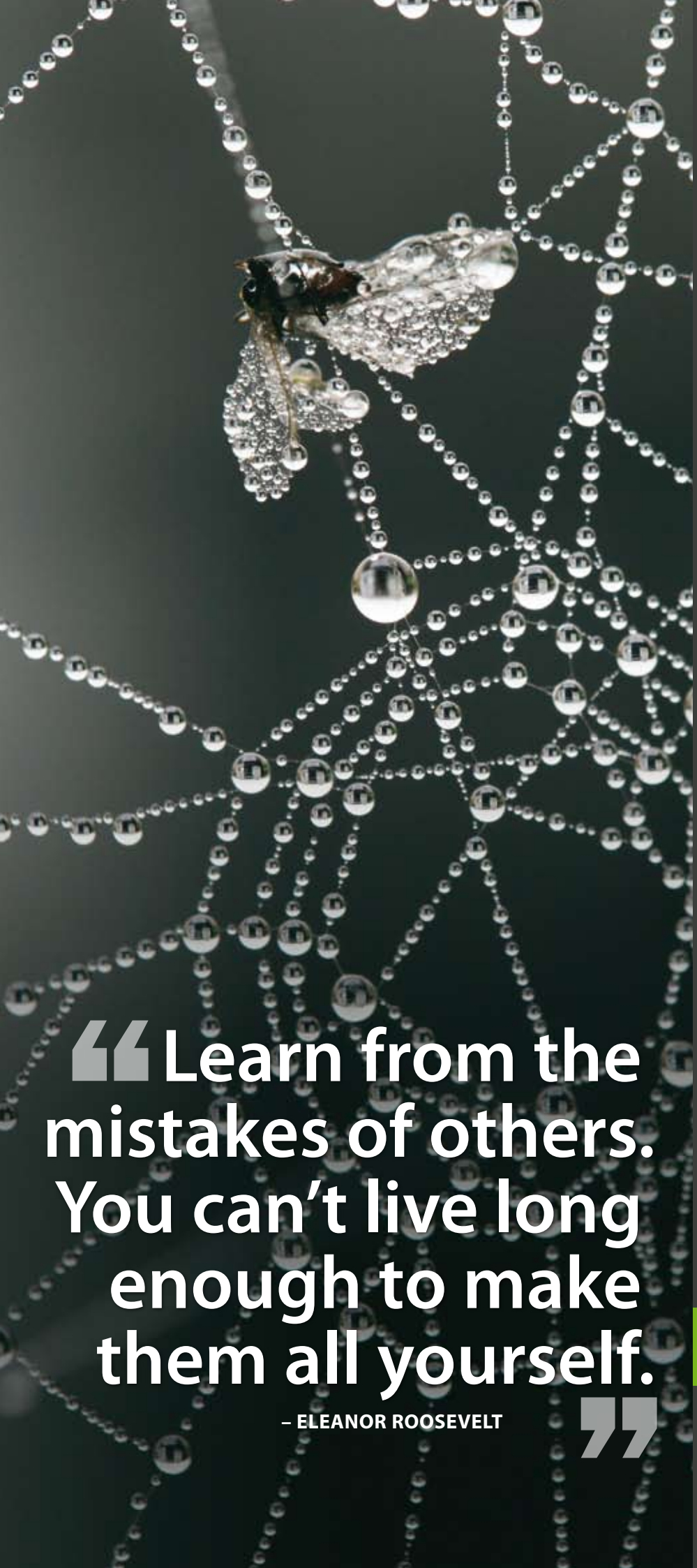
Many medicine interactions are harmless and many of those which are potentially harmful only occur in a small proportion of patients. The severity of an interaction varies from one patient to another. Drugs with a small therapeutic window (e.g. phenytoin) and those which require careful control of dosage (e.g. anticoagulants, antihypertensives and antidiabetics) are most often involved in interactions.

Elderly people and people with impaired renal or liver function are at increased risk of medicine interactions.

Additional interactions information from the BNF

BNF interaction summaries are also provided within the NZF. Interactions shown in **bold** and against a pink background are **potentially serious**; concomitant administration of the medicines involved should be **avoided** (or only undertaken with caution and appropriate monitoring). Interactions that are not in bold type do not usually have serious consequences.

Visit: www.nzformulary.org



“Learn from the mistakes of others. You can’t live long enough to make them all yourself.”

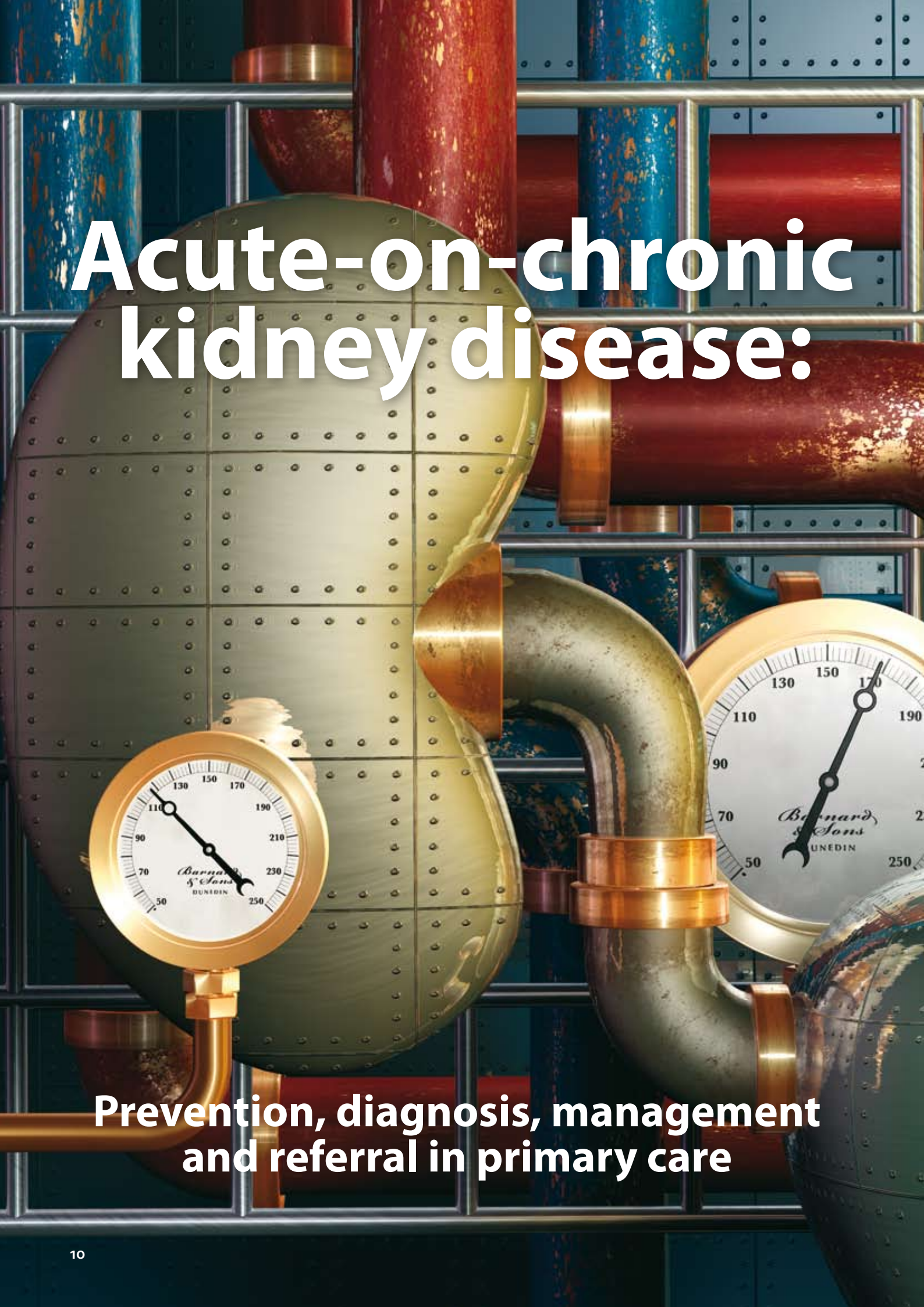
– ELEANOR ROOSEVELT

The bpac^nz Patient Safety Incident Reporting system is an online resource for people working in community health care to report and review patient safety incidents.

Reports are submitted anonymously, to identify factors which have contributed to patient safety incidents and to share solutions to prevent these incidents from occurring again.

Incidents can be reported and cases reviewed at:

www.bpac.org.nz/safety



Acute-on-chronic kidney disease:

Prevention, diagnosis, management and referral in primary care

Acute kidney injury that occurs in a community setting accounts for 1% of all hospital admissions. Most of these patients have pre-existing chronic kidney disease (CKD), managed in primary care. Early identification of patients at increased risk of acute kidney injury can prevent deterioration in renal function. General Practitioners need to be aware of the possibility of acute kidney injury, recognise the causative factors, request and interpret initial investigations and determine the urgency of referral for hospital assessment.

Chronic kidney disease is the major risk factor for acute kidney injury

Acute kidney injury is a medical emergency characterised by a rapid (hours to days) fall in glomerular filtration rate. Most people who experience acute kidney injury have some degree of pre-existing chronic kidney disease (CKD).[1] In a study of over 1700 patients with acute kidney injury requiring dialysis, 74% had an estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73 m^2 in the months prior to admission.[1]Rapidly declining renal function is therefore highly likely to be due to an acute deterioration of CKD, termed acute-on-chronic kidney disease. Acute kidney injury accounts for approximately 1% of all hospital admissions.[2] When severe enough to require dialysis, the associated in-hospital mortality rate can exceed 30%.[1] Prompt diagnosis is important, as in most cases the cause is reversible and early treatment may prevent permanent renal damage.

Who is at risk of developing acute kidney injury?

The prevalence of CKD and acute kidney injury increases with age. Between one-quarter and one-third of all adults aged over 64 years have CKD.[3] The incidence of severe acute kidney injury is more than fifty times higher in people aged over 80 years than in people aged under 50 years.[4]

Diabetes mellitus, hypertension, obesity and proteinuria are independent risk factors for acute kidney injury.[1,5] People with co-existing diabetes mellitus and CKD are at even greater risk of developing acute kidney injury.[1] Older people, who have poor mobility and reduced access to fluids when unwell, have an increased risk of pre-renal injury. When this is combined with polypharmacy, including nephrotoxic medicines, the likelihood of an acute-on-chronic decline in renal function is increased.

Acute kidney injury is now the preferred term

Acute kidney injury is the new consensus term that replaces the term acute renal failure. This term was adopted because the condition is understood to be a continuum of injury, rather than a discrete episode of organ failure defined by arbitrary values. It is also being increasingly recognised that even small impairments to renal function, changes too small to be recognised as organ failure, have a significant effect on patient morbidity and mortality.[6]

What are the causes of acute kidney injury?

Most cases of acute-on-chronic kidney injury occur in the presence of an infection or other concurrent illness. Patients with intrinsic renal disease or low grade chronic obstruction may be largely asymptomatic.

The causes of acute kidney injury can be divided into three categories:

1. Pre-renal causes
2. Intrinsic renal causes
3. Post-renal causes

Pre-renal injury

A reduction in blood flow to the kidney is the most common cause of acute kidney injury.[2] The resulting renal injury is due to the inability to maintain renal blood flow via auto-regulation, and is not due to direct damage to the nephron itself. The defining feature of acute pre-renal injury is that if normal blood flow can be re-established, renal function will often rapidly recover. However, a sustained reduction in renal perfusion increases the risk of intrinsic renal injury (acute tubular necrosis), which may result in irreversible damage to the kidney.

The main causes of pre-renal injury are:[2]

- Hypovolaemia, e.g. as a result of diarrhoea, vomiting, diuretics, osmotic diuresis from poorly controlled diabetes, haemorrhage and traumatic or septic shock
- Decreased effective blood volume, e.g. heart failure or cirrhosis
- Vasoregulation, e.g. medicines such as non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs), or hypercalcaemia

Intrinsic renal injury

Intrinsic renal injury is characterised by direct damage to the nephrons. It is often complex and may be secondary to another illness.[2] The most common cause of intrinsic injury is acute tubular necrosis as a result of pre-renal injury or direct toxicity (hypotension, hypovolaemia, haemolysis, rhabdomyolysis or nephrotoxic medicines, e.g. NSAIDs, lithium or aminoglycosides).[2, 6] The combination of pre-renal injury and acute tubular necrosis accounts for approximately 90% of cases of acute kidney injury.

Medicine-induced interstitial nephritis is the other main form of intrinsic renal disease. There is a high mortality rate associated with intrinsic renal injury, but this is heavily influenced by patient co-morbidity.

Acute glomerulonephritis, particularly as a result of small vessel vasculitis, is an uncommon but important cause of acute kidney injury. Early diagnosis and appropriate treatment prevents end-stage chronic kidney disease.

Post-renal injury

Post-renal injury is caused by a blockage to the flow of urine, resulting in a back pressure to the kidney, causing damage to nephrons. This is obstructive nephropathy and is a relatively uncommon cause of acute kidney injury (5%).[2] Timely diagnosis and treatment can lead to a complete recovery. The most frequent causes of obstructive nephropathy include:[2]

- Urinary tract stones
- Prostatic hypertrophy
- An intra-abdominal process encasing the ureters, e.g. retroperitoneal fibrosis or prostatic and other pelvic malignancy

The site, degree and speed of onset of the obstruction determine the clinical symptoms and signs.

Preventing acute kidney injury

The first focus of primary care is to prevent acute-on-chronic kidney disease from occurring. Patients with CKD, presenting with an acute illness, should have an early assessment of renal function. Blood pressure and volume maintenance, combined with avoidance of nephrotoxic medicines and healthy lifestyle choices, are the recommended strategies for reducing the risk of acute kidney injury in people with CKD.[7] Patients who have had a previous acute decline in renal function should be flagged and identified as being at risk of future kidney injury.

Kidney health checks should be performed regularly

Patients with CKD or risk factors for CKD should have their renal function assessed as indicated by CKD guidelines.

See: "Making a difference in chronic kidney disease", BPJ 22 (Jul, 2009).

Patients with CKD have altered auto-regulation of glomerular blood flow. As a result their creatinine and GFR varies according to their blood pressure. This results in the serum creatinine fluctuating in the absence of clear precipitants. Creatinine often varies by 10–20% in this setting and a repeat creatinine test to exclude further progression is required.

Some medicines should be used with caution

Medicines are reported to contribute to acute kidney injury in approximately 20% of cases.[7] In particular, the triple combination of NSAIDs, ACE inhibitors (or ARBs) and diuretics can cause acute renal injury by interfering with homeostatic mechanisms.[8] This occurs when blood flow to the kidney is compromised, as prostaglandin-mediated afferent arteriolar vasodilation is blocked by NSAIDs, and blood flow from the kidney cannot be reduced, as efferent arteriolar vasoconstriction is prevented by either ACE inhibitors or ARBs. The combined effect is a decline in the glomerular filtration rate which is exacerbated in people who are also taking diuretics.[8] People taking this combination of medicines, particularly those with established CKD and an acute concurrent illness, require regular monitoring of serum creatinine and potassium.

Many other medicines can cause intrinsic acute renal injury, including; furosemide, proton pump inhibitors, beta-lactam antibiotics, aminoglycosides, cyclosporin, sulphonamides, colchicine, phenytoin, lithium and paracetamol (high dose or chronic use).[6,9] Patients who are unwell or develop allergic-type symptoms (allergic nephritis) after starting a new medicine may need their renal function checked.

Acute kidney injury is often associated with acute illness

In primary care, acute-on-chronic kidney disease is often caused by hypovolaemia due to an episode of concurrent illness, e.g. upper or lower respiratory tract infection, urinary tract infection, sepsis or gastrointestinal illness. Maintenance of fluid and electrolyte balance when people are unwell is an important preventative strategy. It may be appropriate for people with CKD and an acute illness to discontinue, or reduce, the dose of potentially nephrotoxic medicines. They should also be advised to avoid taking nephrotoxic medicines, including over-the-counter medicines, e.g. NSAIDs.

Diagnostic procedures may increase the risk of acute kidney injury

People with CKD, particularly in combination with diabetes, are at increased risk of developing acute kidney injury when undergoing procedures requiring radiocontrast media.[6] If contrast-enhanced imaging is required, then consider discontinuing diuretics or any nephrotoxic medicines according to local radiology protocols. Metformin is contraindicated in procedures involving iodine-containing contrast media.[10] Colonoscopy requires bowel preparation which can increase the risk of diarrhoea and volume depletion. When referring people with CKD for colonoscopy, a history of kidney disease should be noted on the referral form and the risk of adverse events discussed with the patient.

Managing acute kidney injury

Acute kidney injury should be considered a medical emergency. If there is a clearly identifiable cause then this should be managed. If the cause of deterioration is not clear, consider discussion with or referral to nephrology services.

Preventative strategies reduce the risk of acute-on-chronic kidney disease and slow the progression of underlying CKD. However, they cannot remove the risk completely or reverse renal damage caused by CKD. Despite sound management, a patient may be found to have an elevated serum creatinine level when assessed as part of routine monitoring, or following investigation of a concurrent illness. If this occurs, the first step is to determine whether the decline in renal function is due to CKD or acute kidney injury – as the management of the two conditions varies.[2]

Distinguishing acute kidney injury from CKD

Previous creatinine measurements are the most useful tool for confirming and assessing the severity of acute kidney injury. The length of time between creatinine measurements will vary from patient to patient and clinical judgement is required to interpret the significance of current levels. The *bestpractice* Decision Support "Chronic Care Module" provides a method to recognise significant rates of change and acute decline in renal function. See Page 5 for further information.

Where there is uncertainty surrounding an assessment of renal function, consultation with a nephrologist is recommended.

Patients who have a single raised serum creatinine and no baseline serum creatinine measurements should be assumed to have acute kidney injury. In the absence of another creatinine result every effort should be made to find a past result. Creatinine levels should be retested to determine the rate of continuing decline. The timeframe for repeat testing depends on the clinical scenario, but should be no longer than 14 days.[9] Where acute decline is suspected and the clinical picture indicates concurrent illness the creatinine should be repeated within 24 hours.

A clinical history may suggest an obvious cause

The findings of the clinical history and physical examination will largely determine whether the patient can be managed in primary care, or if hospital referral is required.

Key points within the history include:

- Any recent acute illness
- Symptoms suggestive of outflow obstruction such as prostate symptoms or abdominal pain in acute obstruction
- A history of abdominal or pelvic malignancy causing obstruction or myeloma causing intrinsic injury from heavy proteinuria
- Systemic symptoms, such as a rash, joint or muscle pain suggesting an underlying systemic disease or vasculitis
- Current prescribed and over-the-counter medicine use or recent contrast radiology
- Pre-existing conditions or a family history of renal disease

Physical examination

Assess whether the patient is dehydrated (e.g. thirst, dry mucous membranes, reduced urinary output, tachycardia) or fluid overloaded (e.g. raised jugular venous pressure, features of pulmonary and peripheral oedema). Look for features of systemic disease, such as fever, skin rashes, joint swelling, iritis or vascular disease, e.g. absent peripheral pulses and cool peripheries. The abdomen should be examined for masses, organomegally, abdominal aortic aneurysm and the bladder palpated and percussed for possible outflow obstruction.[11]

Urinalysis

Where a clinical history and physical examination suggest acute renal injury a urine dipstick test should be performed in order not to miss a renal inflammatory process.[11] Urinalysis that is negative for blood and protein suggests reduced renal blood flow or urinary tract obstruction. However, the later can be complicated by co-existing urinary infection as suggested by the presence of white blood cells or nitrites.[11] Glomerular disease is likely to cause a strongly positive urinalysis for blood and protein (an active urinary sediment).[11] The presence of protein but little or no blood is suggestive of tubular damage or interstitial disease.[11] Macroscopic haematuria may suggest the presence of urinary tract stones or malignancy.

Acute kidney injury – when to refer

Following a diagnosis of acute kidney injury, management should be guided by a discussion with a nephrologist or general medicine physician, especially in the presence of active sediment (positive blood and protein on analysis). An exception to this would be patients with an obvious pre- or post-renal cause of their condition, where the clinician is confident that the patient can be managed in a community setting and treatment will result in a rapid reversal of kidney function, e.g. dehydration following an acute episode of diarrhoea. If there is clinical uncertainty, or an intrinsic renal cause for the condition is suspected, then the patient should be referred to hospital without delay.

Red flags requiring urgent hospital admission include:[7]

- Negligible urine output for 6 hours or < 200 mL over 12 hours
- Serum potassium > 7.0 mmol/L or > 5.5 mmol/L with ECG changes[12]
- Volume overload
- Creatinine concentration > 300 µmol/L or a change of 50% (can be determined using a decision support tool)

Management in the community

Community-based care of patients with acute kidney injury should only be undertaken when the clinician is confident that it can be treated without complications developing, and the patient will be well supported and issues around continuity of care, especially after hours provision, have been addressed. The patient will require daily monitoring of renal function and often assistance from a carer.

The focus of management of acute kidney injury is to:

1. Restore renal blood flow
2. Treat urinary obstructions
3. Review medicine use

Best practice tip: All instances of acute kidney injury should be highlighted in the patient record to prompt future kidney health assessments and prevention of future acute-on-chronic decline. Consider creating a patient alert in the patient management system.

Restoring renal blood flow

Restoration of renal perfusion is the goal in the treatment of pre-renal causes of acute kidney injury. Fluid replacement is the simplest way of achieving this. However, post-renal obstruction first needs to be excluded. Treatment should also target the reason for the volume loss, e.g. diarrhoea or vomiting.

Treating urinary obstructions

Obstruction relief is the goal of treatment in patients with post-renal acute kidney injury. This is necessary to prevent

irreversible kidney damage and for patient comfort.[6] A urethral or suprapubic catheter will relieve obstructions located at the level of the urethra or bladder, respectively.[6] If an obstruction of the upper urinary tract is suspected, then the patient should be referred to an urologist. Once relief of the obstruction has been achieved diuresis may occur, requiring the patient's fluid and electrolyte balance to be monitored.[6]

Medicine review

Patients with acute kidney injury should discontinue non-essential, nephrotoxic medicines, e.g. NSAIDs.[7] Patients with dehydration and pre-renal injury should have their ACE inhibitors, ARBs or diuretics withheld until renal function has recovered. A complete medicine review should also be undertaken either in primary or secondary care as appropriate.

Lifestyle modification to reduce future risk

Following an acute-on-chronic episode all people with CKD should be advised of healthy lifestyle choices that can be made to reduce their risk of a recurrent episode. Salt intake should be limited by using minimal salt when cooking, not adding salt to food and reducing consumption of processed meats and other high-salt food. In the presence of proteinuria, a low protein diet is recommended. Alcohol consumption should be limited Smoking cessation advice in the "ABC" format (ask, brief advice, cessation support) should also be given to all current smokers.

Managing an emergency when hospital admission is delayed

When an acute kidney injury occurs, electrolytes can accumulate and cause life-threatening complications, such as cardiac arrest. Urgent referral to secondary care is recommended for patients with serum potassium > 7.0 mmol/L.[12] An ECG is recommended for patients with serum potassium levels greater than > 5.5 mmol/L with urgent referral if ECG changes are noted (See: "A primary care approach to sodium and potassium imbalance" Best Tests, Sept 2011).[12] If there will be a significant delay in hospital referral, e.g. in an isolated rural setting, then hyperkalaemia should be treated as an emergency.[13] This should be discussed with the on-call renal team at the nearest hospital.

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References

1. Hsu CY, Ordoñez JD, Chertow GM, et al. The risk of acute renal failure in patients with chronic kidney disease. Kidney Int 2008;74(1):101–7.
2. Hilton R. Clinical review: acute renal failure. BMJ 2006;333:786–90.
3. Zhang Q-L, Rothenbacher D. Prevalence of chronic kidney disease in population-based studies: systematic review. BMC Public Health 2008;8:117.
4. Feest TG, Round A, Hamad S. Incidence of severe acute renal failure in adults: results of a community based study. BMJ 1993;306(6876):481–3.
5. Druml W, Metnitz B, Schaden E, et al. Impact of body mass on incidence and prognosis of acute kidney injury requiring renal replacement therapy. Intens Care Med 2010;36(7):1221–8.
6. Khalil P, Murty P, Palevsky P. The patient with acute kidney injury. Prim Care Clin Office Pract 2008;35:239–64.
7. Bellomo R, Kellum JA, Ronco C. Acute kidney injury. Lancet 2012;380(9843):756-66.
8. Fournier J-P, Lapeyre-Mestre M, Sommet A, et al. Laboratory monitoring of patients treated with antihypertensive drugs and newly exposed to non steroidal anti-inflammatory drugs: a cohort study. PLoS ONE 2012;7(3):e34187.
9. National Institute for Health and Clinical Excellence (NICE). Chronic kidney disease: Early identification and management of chronic kidney disease in adults in primary and secondary care. NICE, 2008. Available from: www.nice.org.uk (Accessed Aug, 2012).
10. New Zealand Formulary (NZF). NZF v2.1. NZF, 2012. Available from: www.nzf.org.nz (Accessed Aug, 2012).
11. Brincat S, Hilton R. Acute kidney injury. GPonline, 2008. Available from: www.gponline.com (Accessed Aug, 2012).
12. Guidelines and Audit Implementation Network (GAIN). Guidelines for the treatment of hyperkalaemia in adults. GAIN, Ireland: 2008. Available from: www.gain-ni.org (Accessed Aug, 2012).
13. Griffiths L, Kanagasundaram S. Assessment and initial management of acute kidney injury. Medicine 2011;39(7):390–7.

Bronchiolitis in infants

What is bronchiolitis?

Bronchiolitis is an acute viral infection affecting the bronchioles of the lower respiratory tract. Most cases are caused by Respiratory Syncytial Virus (RSV).

Bronchiolitis is the most common lower respiratory tract infection in infants, and between 2006 – 2010, accounted for almost 15% of all childhood illness-related hospital admissions in New Zealand.[1]

Risk factors associated with bronchiolitis

A New Zealand study of infants aged under two years, who were hospitalised for bronchiolitis during the 2003 – 2005 RSV seasons (Jun – Oct), found that birth between February and July, prematurity and Māori or Pacific ethnicity were risk factors for hospitalisation.[2] Additional factors, which partly explain the increased risk in Māori and Pacific children, included higher maternal smoking rates during pregnancy, the socioeconomic status of the infant's community and low birth weight.[2] Māori and Pacific children are admitted to hospital for acute bronchiolitis at rates approximately three and five times higher respectively, than children of other ethnicities (Figure 1).

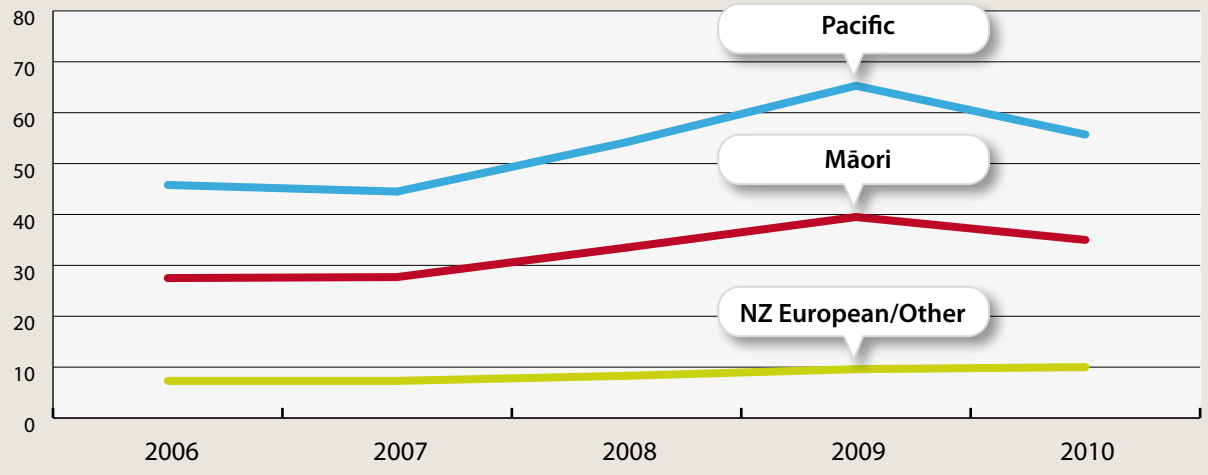


Figure 1: Annual hospital discharge rates for children aged under five years, per1000 patients, by ethnicity

How to diagnose bronchiolitis

Bronchiolitis is diagnosed clinically. Chest x-rays should not be routinely performed. Bronchiolitis is likely if a child aged under 18 months, who has had initial signs and symptoms of an upper respiratory tract infection, presents with the following features:[4]

- Cough
- Tachypnoea
- Inspiratory crepitations
- Wheeze

Dehydration may occur due to difficulties with feeding and losses with tachypnoea.

A low grade fever (less than 39°C) may be present, but if a high fever is present, alternative diagnoses, such as pneumonia, should be considered (Table 1).[4, 5]

Wheeze in children aged under one year should generally be regarded as being due to bronchiolitis or another disorder, and not asthma. However, some infants with suspected bronchiolitis will have a degree of response to a beta-2 agonist. Consider a diagnostic trial of a beta-2 agonist in a child aged over nine months with recurrent wheeze (especially if atopic), after other causes have been excluded.[4]

For further information see: "Diagnosing and managing asthma in children", BPJ 42 (Feb, 2012).

Hospitalisation and overcrowding

Overcrowding and sub-standard housing can negatively impact a family's health.[3] In New Zealand, there is a correlation between household overcrowding and the risk of contracting infectious diseases, such as meningococcal disease.[3] It is probable that overcrowding also increases an infant's risk of developing bronchiolitis.[2]

The Healthy Housing programme has operated in the Counties Manukau DHB since 2001. Through improving access to health care services and providing larger, warmer and better designed houses for over 3000 Pacific families in low socioeconomic areas, the programme has achieved an 11% reduction in acute hospitalisations for children aged 0 – 4 years and a 23% reduction for people aged 5 – 35 years.[3] These results appeared to be most significantly due to reductions in the rates of respiratory infections, asthma and cellulitis.[3]

Table 1: Alternative diagnoses to bronchiolitis[4]

Alternative diagnosis	Distinguishing clinical features
Asthma	Recurrent wheeze
Pertussis	Cough as the main symptom
Pneumonia	Difficult to distinguish, purulent phlegm production may be more prominent
Inhaled foreign body	Sudden onset of symptoms, history of coughing/choking followed by expiratory wheeze, loss of voice or differential air entry on examination
Congestive heart failure	Cardiac murmur, oedema or a history of slow onset of symptoms, failure to thrive
Cystic fibrosis	Repeated and prolonged respiratory infections, failure to thrive

Table 2: Guidance for the assessment and treatment of bronchiolitis in children aged under 18 months.[4, 6]

Mild	Moderate	Severe
■ Normal respiratory rate ■ No or subtle accessory muscle use ■ Normal heart rate ■ Able to feed ■ Oxygen saturation >95%	■ Increased respiratory rate ■ Minor accessory muscle use ■ Increased heart rate ■ Difficulty feeding ■ Minor dehydration, e.g. increased thirst ■ Crepitations ■ Oxygen saturation 90-95%	**The presence of poor respiratory effort, cyanosis or apnoea may indicate life threatening bronchiolitis** ■ Respiratory rate > 60 breaths/minute ■ Moderate/marked accessory muscle use ■ Nasal flare and/or grunting ■ Markedly increased heart rate ■ Feeding < 50% of normal in preceding 24 hours ■ Marked dehydration, e.g. sunken fontanelle, sunken eyes, reduced skin turgor, low urine production, absent tears ■ Toxic appearance ■ Oxygen saturation <90%
Initial treatment		
■ Reassurance and home care	■ Consider referring if child is aged under three months, is not feeding sufficiently, if there is parental distress or if social circumstances are a concern	■ Send to hospital by ambulance ■ Provide oxygen if available
■ Encourage small frequent feeds ■ If nasal congestion, trial saline nasal drops ■ Reassure parents that improvement can be expected within three days, but give instructions to return if there is any concern or if symptoms become severe, e.g. tell them to look for dry nappies, refusal to feed, decreased level of alertness or any breathing difficulties such as nasal flaring, use of accessory (neck) muscles		

N.B. If the child presents with signs or symptoms across categories then management should be according to the most severe symptoms.

Assessment of bronchiolitis

Infants aged under one month with suspected bronchiolitis should be referred to hospital. Table 2 provides guidance on the assessment and treatment of bronchiolitis in primary care.[4, 6] A lower threshold for referral is recommended in the management of infants aged under three months and infants with underlying cardio-respiratory disease.[4]

Socioeconomic factors, e.g. housing quality, should also be considered when managing infants. A lower threshold for admission may be appropriate if the family home environment is considered to be contributing to the condition.

Management of bronchiolitis

A child with mild to moderate bronchiolitis can be managed at home. Pharmacological treatment is not required. It should be explained to parents and caregivers that in the first 72 hours the symptoms may worsen before starting to improve. Instructions should be given on how to identify concerning signs of deterioration, e.g. marked dehydration and respiratory distress, and to access after-hours care if required. This is especially important for Māori and Pacific families, as Māori or Pacific ethnicity is an independent risk factor for hospitalisation for bronchiolitis.[2]

Infants who become dehydrated or who are feeding at below half of the normal amount in a 24 hour period should be assessed and referred to hospital if necessary. High risk-infants, e.g. aged under three months or with underlying co-morbidities, who are examined early in the illness, should be reassessed within 24 hours for signs of deterioration.

Fluids (e.g. breast milk) should be given in small amounts, frequently, to prevent dehydration.

Saline drops may be be trialled if there is nasal congestion.

Steam inhalation is sometimes used for symptomatic relief, however, there is no evidence that it is effective in the treatment of bronchiolitis.[7]

Antibiotics are ineffective as bronchiolitis is viral in origin.

Inhaled bronchodilators are not recommended for the treatment of infants with bronchiolitis and no other history of recurrent wheeze.[8] The respiratory symptoms of bronchiolitis are caused by blockage of airways with mucous, rather than airway narrowing, therefore bronchodilators have little benefit. They do not improve oxygen saturation, reduce the need for hospitalisation or shorten the duration of the illness.[8] Small, short-term improvements in respiratory score following bronchodilation may occur, however, adverse effects include tachycardia and tremor.[8]

There is no evidence to support the use of **oral** or **inhaled corticosteroids** or **ipratropium** for the treatment of bronchiolitis in primary care.

Hospital treatment is also supportive and includes nasal suction, supplemental oxygen, rehydration and maintenance of hydration.

A smoke-free home with a room that is at a comfortable temperature for a lightly clothed adult should be provided for the infant to sleep in. Where possible the infant should avoid close contact with other children to prevent transmission of the disease. Other family members with respiratory symptoms should also avoid close contact in order to reduce the risk of the infant developing a secondary infection. Re-infection is common and hand washing is the best way to prevent RSV transmission.

Post-bronchiolitic wheeze after hospitalisation

Persistent wheeze is experienced by approximately 40% of infants who are hospitalised due to bronchiolitis, continuing up to age five years.[9] Approximately 10% will continue to have wheezing episodes after age five years, but by age 13 years, wheeze will have resolved in most children.[9]

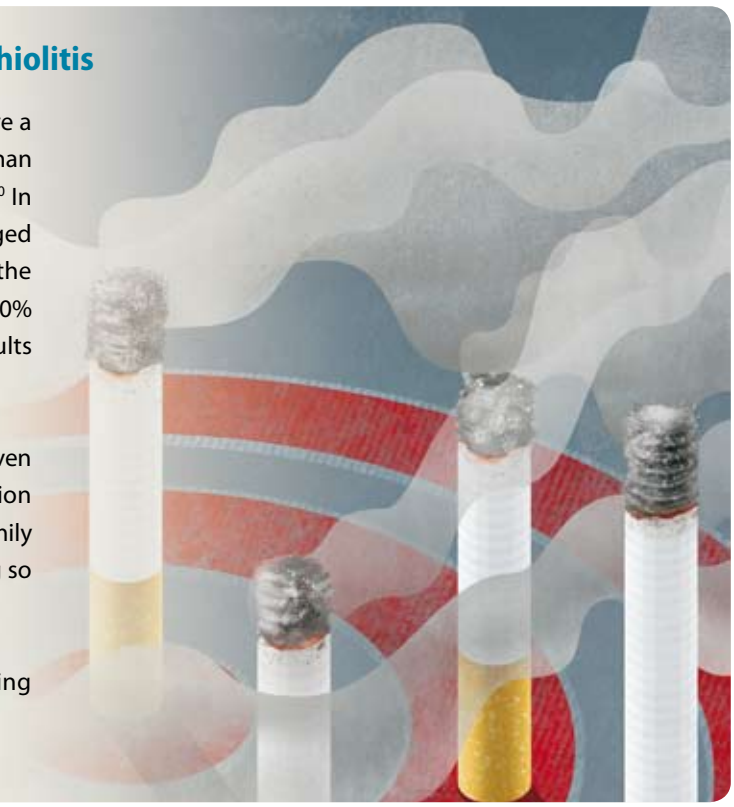
For further information see: "Bronchiolitis update", BPJ 20 (Apr, 2009).

Passive smoking increases risk of bronchiolitis

Infants in a household where both parents smoke have a risk of developing bronchiolitis three times greater than infants in a household where neither parent smokes.[10] In 2009, approximately 20% of people in New Zealand aged 15 – 64 years were current smokers.[11] However, in the same age range, almost 50% of Māori females, over 40% of Māori males and approximately 30% of Pacific adults were current smokers.[11]

It is recommended that smoking cessation advice be given (using the "ABC" method – Ask, Brief Advice, Cessation support) to any family member who is a smoker.[12] If family members must smoke, stress the importance of doing so outside, away from children and never in a car.

For further information see: "Update on smoking cessation" BPJ 33 (Dec, 2010).



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References

1. New Zealand Child and Youth Epidemiology Service. Hospital admissions and mortality with a social gradient in children. The Children's Social Health Monitor New Zealand. Available from: www.nzchildren.co.nz/hospital_admissions.php (Accessed Aug, 2012).
2. Grimwood K, Cohet C, Rich FJ, et al. Risk factors for respiratory syncytial virus bronchiolitis hospital admission in New Zealand. Epidemiol Infect 2008;136(10):1333–41.
3. Jackson G, Thornley S, Woolston J, et al. Reduced acute hospitalisation with the healthy housing programme. J Epidemiol Commun H 2011;65(7):588–93.
4. Turner T, Wilkinson F, Harris C, Mazza D. Evidence based guideline for the management of bronchiolitis. Aust Fam Physician 2008;37(6):6–13.
5. Scottish Intercollegiate Guidelines Network. SIGN 91: Bronchiolitis in children 2006. Available from: www.sign.ac.uk (Accessed Aug, 2012).
6. National Institute for Health and Clinical Excellence. Feverish illness in children: assessment and initial management in children younger than 5 years. 2007. Available from: www.nice.org.uk (Accessed Aug, 2012).
7. Umoren R, Odey F, Meremikwu MM. Steam inhalation or humidified oxygen for acute bronchiolitis in children up to three years of age. Cochrane Database Syst Rev 2011;(1):CD006435.
8. Gadomski AM, Brower M. Bronchodilators for bronchiolitis. Cochrane Database Syst Rev 2010;(12):CD001266.
9. Paediatric Society of New Zealand. Wheeze and chest infection in infants under 1 year. Best practice evidence based guideline. 2005. Available from: www.paediatrics.org.nz (Accessed Aug, 2012).
10. Jones LL, Hashim A, McKeever T, et al. Parental and household smoking and the increased risk of bronchitis, bronchiolitis and other lower respiratory infections in infancy: systematic review and meta-analysis. Respir Res 2011;12:5.
11. Ministry of Health. Tobacco use in New Zealand: Key findings from the 2009 New Zealand tobacco use survey. Wellington: Ministry of Health; 2010. Available from: www.health.govt.nz (Accessed Aug, 2012).
12. Ministry of Health. New Zealand smoking cessation guidelines. 2007. Available from: www.health.govt.nz (Accessed Aug, 2012).

Bronchiectasis:

Rates still increasing among Pacific peoples

What is bronchiectasis?

Bronchiectasis is a lung disease characterised by irreversible bronchial dilation and chronic inflammation, resulting in chronic wet cough. Recurrent cycles of infection and inflammation impair mucociliary clearance, leading to progressive remodelling and scarring of the bronchial walls. Symptoms of bronchiectasis are often present for many years before the diagnosis is made.[1]

Bronchiectasis occurs in both children and adults. Although a relatively uncommon condition, bronchiectasis disproportionately affects Māori and Pacific peoples and people from lower socioeconomic communities. Clinicians need to consider this when diagnosing and treating chronic, wet cough in these patients. Empiric treatment of recurrent chest infections with antibiotics may be considered in some patients with risk factors to help prevent the development of bronchiectasis.

Although bronchiectasis is most often found in children with cystic fibrosis (in developed countries), this article focuses on non-cystic fibrosis bronchiectasis.

What causes bronchiectasis?

Bronchiectasis is commonly caused by recurrent or severe respiratory infections such as pneumonia (both bacterial and viral), tuberculosis, adenovirus, measles and pertussis.[2] It may also be associated with; congenital syndromes and abnormalities, chronic obstructive pulmonary disease (COPD), gastro-oesphageal reflux, smoking and passive smoking, mucociliary dysfunction, immune deficiency, pulmonary fibrosis, post-obstruction (e.g. with an unrecognised foreign body), recurrent aspiration and systemic inflammatory diseases (e.g. rheumatoid arthritis, sarcoidosis). Overcrowding and socioeconomic deprivation are also important contributing factors.[1, 2]

The rate of bronchiectasis in New Zealand adults is not known, however, prevalence increases with age. In the United States, the estimated prevalence of bronchiectasis ranges from 4.2 per 100 000 in people aged 18 – 34 years to 272 per 100 000 in people aged over 75 years.[3]

Pacific children are at increased risk of developing bronchiectasis

In New Zealand, Pacific children aged under 15 years have an estimated incidence rate of bronchiectasis of 17.8 per 100 000.[4] This compares to 4.8 and 1.5 per 100 000 for Māori and New Zealand European children respectively, of the same age.[4] Capital & Coast DHB reported that from 2002 – 2006, the risk of being hospitalised due to bronchiectasis was almost 11 times higher for a Pacific child aged under 14 years and four times higher for a Māori child, than for a New Zealand European child.[5]

Socioeconomic deprivation is associated with an increased risk of developing bronchiectasis. The relative risk of a child living in a decile nine or ten community (most deprived) developing bronchiectasis is over 15 times higher than for a child living in a decile one area (least deprived).[5] Issues such as housing and financial support may need to be considered in the management of the condition.

When to suspect bronchiectasis

The symptoms and signs of bronchiectasis are not diagnostic and may be mild in the early stages of the illness.

Consider bronchiectasis in a child with:[1, 2, 6]

- A chronic wet/productive cough lasting longer than six weeks, especially between viral infections (N.B. if there is suspicion of an inhaled foreign body, the child should have a chest x-ray after two weeks)
- Wheeze that does not respond to treatment
- Partial resolution of severe pneumonia or recurrent pneumonia
- Persistent lung crackles
- Persistent x-ray changes
- Respiratory symptoms with structural or functional disorders of the oesophagus and upper respiratory tract

Other clinical features of bronchiectasis include dyspnoea, chest pain, clubbing, hyperinflation or chest wall deformity and failure to thrive.[1]

Consider bronchiectasis in an adult with a chronic productive cough and:[2]

- A long history of respiratory symptoms
- Large volumes of purulent sputum on a daily basis
- Haemoptysis
- No history of smoking*

* N.B. Although smoking can be a factor in the development of bronchiectasis, it is not a major cause. COPD is a more common respiratory disease in adults, especially among people who smoke.

The prevalence of bronchiectasis increases with age, however, younger age at presentation should increase the clinical suspicion of bronchiectasis, because a diagnosis such as COPD is less likely in a young adult.

Bronchiectasis can co-exist with, or be misdiagnosed as, other chronic respiratory diseases that are more commonly seen in primary care, e.g. asthma, COPD, rhinosinusitis and tracheobronchial infection.[7] Bronchiectasis should be considered in patients being treated for COPD when; management is complicated, there is slow recovery from lower respiratory tract infections, there are frequent exacerbations, there is no history of smoking or sputum is colonised with *Pseudomonas aeruginosa*.[2]

Investigating suspected bronchiectasis

All patients with suspected bronchiectasis should be referred to secondary care for confirmation of the diagnosis, exploration of underlying aetiology and ongoing management and support. High-resolution computed tomography is the current diagnostic gold standard.[1] Several investigations can be performed in primary care before a referral is made.

A sputum sample should be collected for microbiological analysis.[2] This information is important for selecting antibiotics to treat future exacerbations. Ideally, this should be done when the patient is stable and not currently taking antibiotics.

A chest x-ray should be arranged.[2] The presence of dilated and thickened airways (tramlines) is highly suggestive of bronchiectasis. A normal chest x-ray does not exclude bronchiectasis, however, it is useful for excluding other causes of persistent cough. A child with a wet cough lasting longer than two weeks should also have a chest x-ray to exclude the possibility of an inhaled foreign body.

A full blood count and CRP may show non-specific changes, e.g. infection, inflammation, anaemia or polycythaemia.

Immunoglobulins (IgA, IgG and IgM) should be requested to assess basic immune function.

A Mantoux or interferon gamma release assay (IGRA) should be arranged to exclude tuberculosis. IGRA testing, e.g. Quantiferon-TB Gold assay, should be first-line in patients who have previously received a Bacille Calmette-Guérin (BCG) vaccination, as 3 – 5% of people who receive a BCG vaccination as an infant and approximately one-third of those who receive it as an adult will return a false-positive Mantoux result.[8]

Management of bronchiectasis

The goals of management are the prevention and treatment of exacerbations and the prevention of lung function decline, through improved airway secretion clearance and antibiotics. Treatment plans are an important component of this strategy. Children with bronchiectasis require long-term follow-up in secondary care and should be under the care of a paediatrician as the monitoring of nutrition, growth and development are essential. Management of bronchiectasis is similar to cystic fibrosis care. Chest physiotherapy for airway secretion clearance and antibiotics are the mainstays of treatment. It is important that clear information is given so that patients and their families understand the rationale behind the treatment plan.

Smoking should be avoided and homes should be **smoke-free**. Patients can be provided with a "back-pocket" **prescription for antibiotics**, to allow for the prompt treatment of exacerbations.[9] All patients with bronchiectasis should receive an **annual influenza vaccination**. **Pneumococcal vaccination** is also recommended, however, this is unfunded for adults (unless pre/post splenectomy).[10] Pneumococcal vaccination is part of the New Zealand Childhood Immunisation Schedule.

Chest physiotherapy should be performed at home once or twice daily when the patient is well, and should be intensified if cough increases or exacerbation occurs. A physiotherapist should create an individualised airway secretion clearance programme for patients and their families. Family and patient comprehension of this programme is likely to influence compliance. Regular exercise should also be encouraged.[1] There is little evidence to support the routine use of mucolytics in patients with bronchiectasis.[11]

Spirometry can be used at each review (if available) to record any deterioration in lung function.[1] N.B. Spirometry usually cannot be performed in children aged under five years.

Bronchodilators and corticosteroids should not be routinely prescribed for patients with bronchiectasis (particularly children), but may be considered in some cases for adults. Patients with bronchiectasis and co-existing asthma should continue to use beta-2 agonists as prescribed.[1] Inhaled corticosteroids provide only modest benefit to patients with bronchiectasis. Inhaled and oral corticosteroids should only be prescribed where there is an established diagnosis of co-existing asthma.[1]

Managing exacerbations

Prompt use of broad-spectrum antibiotics and increased physiotherapy are essential in treating bronchiectasis exacerbations.[9] The choice of antibiotic should always be guided by sputum culture. If no sputum culture information is available, then a sample should be taken and empiric treatment initiated. *Haemophilus influenzae* should be assumed to be present and amoxicillin administered first-line.[2, 12] Cefaclor, erythromycin or doxycycline in adults are alternatives if the patient is allergic to penicillin.[9] High-dose amoxicillin, e.g. 1 g three times a day or even up to 3 g twice daily, may be considered for adults with severe bronchiectasis who are chronically colonised with *H. influenzae*.[2] Antibiotics are required for at least two weeks.

Follow-up is recommended to assess the effectiveness of any antibiotic treatment. If the patient has not improved, hospital admission for intravenous antibiotics may be required. When sputum culture indicates the presence of *Pseudomonas aeruginosa*, treatment with ciprofloxacin for a maximum of 14 days is indicated.[1]

For children, eradication of *P. aeruginosa* is more aggressive and may require hospital admission, intravenous and nebulised antibiotics. A paediatrician should be consulted following all new *P. aeruginosa* positive cultures.

Hospital admission is indicated for all patients with bronchiectasis who display any of the following clinical features:[2]

- Breathlessness with an elevated respiratory rate and increased effort to breathe in children or respiratory rate > 25 breaths/minute in adults
- Circulatory or respiratory failure or cyanosis
- Temperature ≥ 38°C
- Unable to take oral treatment
- Intravenous treatment required

Socioeconomic circumstances and ability for patients, or parents/caregivers in the case of children, to cope at home may lower the threshold for referral.

Acute haemoptysis may occur during an exacerbation, more often in adults than children. This can be a life-threatening situation and patients who have expelled a volume of blood greater than 25 mL require urgent hospital assessment.[2]

Occasionally patients require surgical resection for symptoms resistant to standard treatments. This is unusual and would be determined in secondary care.

For further information see: "The burden of bronchiectasis in Pacific peoples", BPJ 32 (Nov, 2010).

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References

1. Chang AB, Bell SC, Byrnes CA, et al. Chronic suppurative lung disease and bronchiectasis in children and adults in Australia and New Zealand. Med J Aust 2010;193(6):356–65.
2. Hill AT, Pasteur M, Cornford C, et al. Primary care summary of the British Thoracic Society Guideline on the management of non-cystic fibrosis bronchiectasis. Prim Care Respir J 2011;20(2):135–40.
3. Weycker D, Edelsberg J, Oster J, Tino G. Prevalence and economic burden of bronchiectasis. Clin Pulm Med 2005;12(4):205–9.
4. Twiss J, Metcalfe R, Edwards E, Byrnes C. New Zealand national incidence of bronchiectasis 'too high' for a developed country. Arch Dis Child 2005;90(7):737–40.
5. Captial & Coast District Health Board. Improving child health outcomes in Porirua city. Available from: www.ccdhb.org.nz/Planning/chag/Child%20Health%20Outcomes_final%20version-sandra.pdf (Accessed July, 2012).
6. Al Subie H, Fitzgerald DA. Non-cystic fibrosis bronchiectasis. J Paediatr Child Health 2012;48(5):382–8.
7. ten Hacken NHT, van der Molen T. Bronchiectasis. BMJ 2010;341:c2766.
8. Ministry of Health. Guidelines for tuberculosis control in New Zealand. Wellington: Ministry of Health; 2010. Available from: www.health.govt.nz (Accessed July, 2012).
9. Edwards E. Bronchiectasis - acute respiratory exacerbation. 2008. Available from: www.adhb.govt.nz/starshipclinicalguidelines (Accessed July,2012).
10. Ministry of Health. Immunisation handbook. Wellington: Ministry of Health; 2011. Available from: www.health.govt.nz (Accessed July, 2012).
11. Pasteur MC, Bilton D, Hill AT. British Thoracic Society guideline for non-CF bronchiectasis. Thorax 2010;65(suppl):1-58.
12. Chang AB, Grimwood K, Maguire G, et al. Management of bronchiectasis and chronic suppurative lung disease in indigenous children and adults from rural and remote Australian communities. Med J Aust 2008;189(7):386–93.

Otitis media: a common childhood illness

What is otitis media?

Otitis media is inflammation of the middle ear and the tympanic membrane, which often occurs as a result of an acute upper respiratory tract infection. Generally, it is caused by a viral infection that is then complicated by a secondary bacterial infection.[1] However, the initial infection may also be bacterial.[1]

Otitis media is very common in children and there is a high rate of spontaneous recovery. It has been suggested that it is an unavoidable illness of childhood and part of the natural maturation of a child's immune system.[2] Despite this, suppurative complications can occur, such as perforation of the tympanic membrane, otitis externa and mastoiditis, as well as other sequelae affecting balance, motor control and hearing.[2]

Otitis media covers a spectrum of conditions, of which the most common are acute otitis media and otitis media with effusion (OME). Pneumatic otoscopy or tympanometry if available, are used to aid diagnosis.

What are the risk factors for otitis media?

The main factors influencing the risk of a child developing otitis media are host-related or environmental (Table 1, over page). Children living in communities where overcrowding is common are at increased risk of developing otitis media.[3] Māori and Pacific children are more likely to be affected by otitis media than European children in New Zealand, however, reliable estimates of prevalence by ethnicity are not available.

Acute otitis media

By age three years, 50 – 85% of children will have had acute otitis media.[2] The incidence peaks between age 6 – 12 months, and recurrent acute otitis media is common, affecting 10 – 20% of children by age one year.[2]

Diagnosis of acute otitis media

Children with acute otitis media have rapid onset of pain and/or fever. The most useful symptom for diagnosis is otalgia (ear pain). Children may also display symptoms of an upper respiratory tract infection, abnormal ear tugging, otorrhoea (discharge from the ear), hearing loss, irritability and not settling at night (pain increases when supine).[5]

On otoscopic examination, the tympanic membrane will:

- Bulge due to effusion with a loss of normal landmarks
- Show areas of intense erythema and/or a yellow colouration
- Show a loss of translucency and be dull or opaque
- Display reduced mobility

Bulging, opacity and immobility are all highly predictive of acute otitis media.

Management of acute otitis media

Reassurance is an important aspect of the management of otitis media. Symptom relief with analgesics and watchful waiting is recommended, as approximately 80% of children with

acute otitis media have spontaneous resolution within two to 14 days.[2] Paracetamol is the first-line analgesic. Ibuprofen is known to reduce inflammation and pain associated with acute otitis media, however, it should not be given if the child displays signs of dehydration or has concurrent asthma (NSAIDs can potentially worsen asthma symptoms). There is no evidence that antihistamines or decongestants provide benefit.[7]

Antibiotics are not routinely required in the treatment of uncomplicated acute otitis media. However, a recent study suggests that empiric antibiotic treatment of acute otitis media in infants can reduce symptoms and decrease the likelihood of persistent infection.[8]

Antibiotic treatment should be considered in children:[7, 9]

- Aged under six months
- Aged under two years, with bilateral acute otitis media or severe illness
- With acute otitis media and perforation
- With systemic symptoms, e.g. fever
- Who have not improved following 48 hours of watchful waiting

When antibiotics are required, first-line treatment is amoxicillin 40 mg/kg/day in two to three divided doses (up to 1.5 g daily), for five days. Treat for seven to ten days in a child aged under two years, with an underlying medical condition, perforated eardrum or chronic or recurrent infections.[7, 10] N.B. Some clinicians are now recommending using amoxicillin 80 mg/kg/day as it is more effective in treating resistant *Streptococcus pneumoniae* strains, however, current guidelines reflect the lower dose. Co-trimoxazole, cefaclor and erythromycin are alternative antibiotics to amoxicillin, although erythromycin would be trialled last as it has poor activity against *Haemophilus influenza*, which is a common pathogen associated with otitis media.[1, 10]

Delayed ("back pocket") prescribing may be appropriate in some cases. Providing a prescription with advice to only use it if the symptoms persist for more than 24 – 48 hours can reduce antibiotic use without reducing family satisfaction with treatment.[7] This can also be written on the prescription in order to allow pharmacists to provide counselling on delayed use when parents request immediate dispensing of antibiotics. Alternatively, the prescription could be dated in advance. The family should be asked to return if the child's condition worsens despite the use of antibiotics, or if they have concerns about the child's health.

Table 1: Patient and environmental factors influencing a child's risk of developing otitis media[4]

Patient-related factors	
Age	Otitis media peaks between ages 6 - 24 months and between ages 4 – 5 years
Reduced breast feeding	Breast feeding for at least three months reduces the rates of acute otitis media by 13%
Premature birth	May increase the risk of otitis media
Use of a dummy (pacifier)	After age 11 months dummy use increases the risk of children developing acute otitis media by 24%
Environmental factors	
Attendance at early childhood care	The most significant environmental factor and directly related to the number of children at each centre
Overcrowded homes and/or a large number of older siblings	As with daycare, relating to increased close contact between siblings
Winter	Upper respiratory tract infections are more common in winter and viruses are often associated with acute otitis media
Passive smoking	Shown to increase the risk of otitis media in children

Otitis media with effusion (OME)

OME is defined as the presence of fluid in the middle ear without signs or symptoms of an infection.[1] It can occur spontaneously, as part of rhinosinusitis, or following an episode of acute otitis media. It is the most frequent cause of balance disorder and acquired conductive hearing loss in childhood.[4, 11]

Following acute otitis media, OME is present in approximately half of patients at one month, 20% of patients at two months and 10% of patients at three months.[12] A study which included tympanometry as part of an assessment of routine development in over 1000 Pacific children, found that at age two years one in four were affected by OME.[11]

Most children with OME will improve spontaneously within three months.[7] There is concern that persistent hearing loss due to OME can affect language development, although no causal relationship has been clearly established.[7]

Diagnosing otitis media with effusion

A diagnosis of OME can be confirmed if pneumatic otoscopy or tympanometry shows reduced or absent tympanic membrane mobility.[5] Other signs include abnormal colouring of the tympanic membrane, e.g. yellow or amber, opacity not due to scarring (which may be present in patients with a history of inner ear issues) or the presence of air bubbles.

Management of otitis media with effusion

Watchful waiting is recommended as most cases of OME resolve spontaneously.[7] Antibiotics provide little or no long-term benefit for children with OME.[13]

Interpretation of tympanometry

Tympanometry allows assessment of middle ear function by measuring tympanic membrane compliance (mobility).

Tympanograms vary between patients, however, there are three broad types of trace that can be used for diagnostic purposes:

1. **The type "A" trace** reflects a normal middle ear mechanism.
2. **The type "B" trace** lacks a sharp peak, suggesting decreased mobility of the tympanic membrane, which may be due to the presence of fluid within the middle ear. Cerumen occluding the ear and grommets can also cause a type "B" trace. In adults, other causes should be considered including tympanic membrane scarring, tympanosclerosis, cholesteatoma and a middle ear tumour.
3. **The type "C" trace** indicates negative pressure within the middle ear space and correlates with a retracted tympanic membrane. This suggests eustachian tube dysfunction or the aspiration of fluid from the nasopharyngeal space into the middle ear, which may result in acute otitis media.

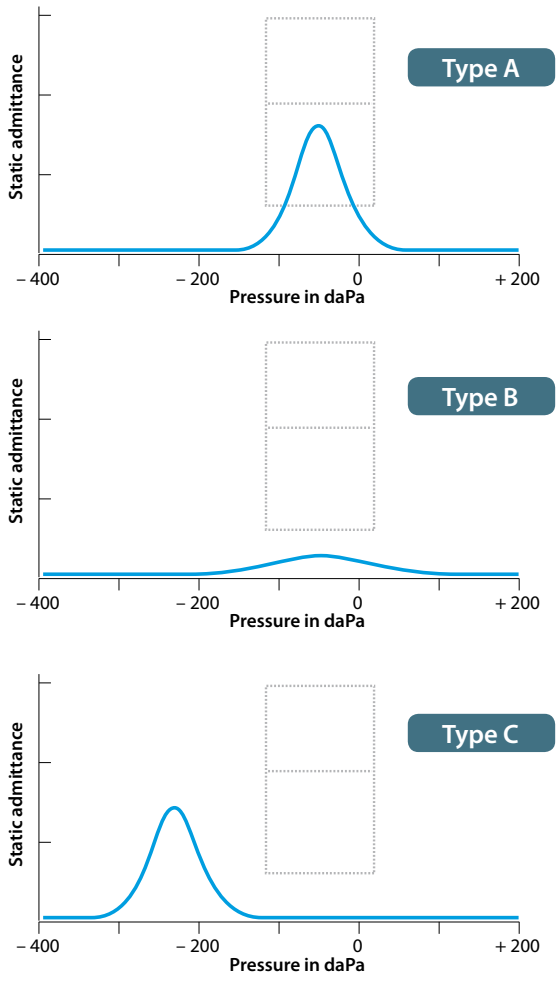

Figure 1: Type "A", "B" and "C" tympanograms adapted from Onusko et al, 2004[6]

Reducing the risk of otitis media-related complications

In New Zealand, there is limited epidemiological data regarding complications associated with otitis media. In Australia, indigenous children, particularly those living in isolated rural communities, are most at risk of complications.[7]

The two most significant modifiable risk factors for otitis media are dummy (pacifier) use after the age of 11 months and passive smoking.[4] Smoking rates are higher amongst Māori and Pacific peoples compared to Europeans in New Zealand, therefore reducing smoking rates is likely to reduce the rate of otitis media in Māori and Pacific children. Breast feeding is thought to protect against the development of otitis media.[4] Teaching and encouraging young children to blow their noses may also assist in routinely clearing the eustacian tubes.

Referral to an otolaryngologist should be considered in children who have recurrent acute otitis media. This is defined as more than three episodes in a six month period, or four times in one year.[14] Grommets have been shown to reduce the incidence of recurrent acute otitis media in the six months following insertion (see "Grommets – indications, outcomes and complications").[15] Referral is also indicated for children with OME and hearing loss lasting longer than three months that has been confirmed by otoscopy or tympanometry.[14]

To confirm hearing loss, a hearing test should be arranged for all children with bilateral OME that persists for longer than three months.[1]

The Kidz First hearing and vision testing service provides a free mobile service to children with high needs within the Counties Manukau region. There are also community-based

Grommets – indications, outcomes and complications

The association between persistent hearing loss caused by OME and adverse language and behavioural outcomes is controversial. Grommets (tympanostomy tubes) are often inserted to restore hearing in children with chronic OME. A Cochrane review found that grommet insertion could improve hearing loss associated with OME in children, however, this effect was limited to six to nine months post surgery, by which time natural resolution also led to improved hearing in non-surgically treated children.[16] The review concluded that a policy of watchful waiting for three months in children with bilateral OME and a hearing impairment is justified, as during this period, approximately 50% of children will experience spontaneous resolution.[16] This guidance is in line with United Kingdom NICE referral guidelines for the surgical management of OME in children.[17] It is also supported by another Cochrane review, which found no clinically significant benefits to language and behaviour outcomes of screening and early treatment of OME in the first four years of life.[18]

Grommet insertion is also used to prevent recurrent acute otitis media. A Cochrane review found that grommet insertion was effective in reducing the frequency of recurrent acute otitis media in the first six months following surgery.[15]

Otorrhoea (discharge) will be experienced by one in four children at some stage while the grommets are in place.[19] In approximately 4% of infants, this complication will develop into chronic suppurative otitis media (CSOM), the most severe form of otitis media.[19] Grommet insertion is a significant cause of CSOM in children, but it can also develop as a complication of acute otitis media with perforation.[20]

A diagnosis of CSOM can be made when there is discharge through a perforated tympanic membrane, which persists for two to six weeks.[1] Topical quinolones are the first-line treatment, as there is a small risk of ototoxicity associated with the use of non-quinolone topical antibiotics such as aminoglycosides.[1] Ciprofloxacin with hydrocortisone ear drops (not subsidised) are effective in treating CSOM. The external auditory canal should be cleaned using suction prior to administration to allow the antibiotic to penetrate affected tissues. Children who fail to respond to topical antibiotics should be referred for more intensive treatment.

clinics in this region. Further information is available from: **www.healthpoint.co.nz**. Similar services also exist in other regions. Local DHBs should be contacted for details.

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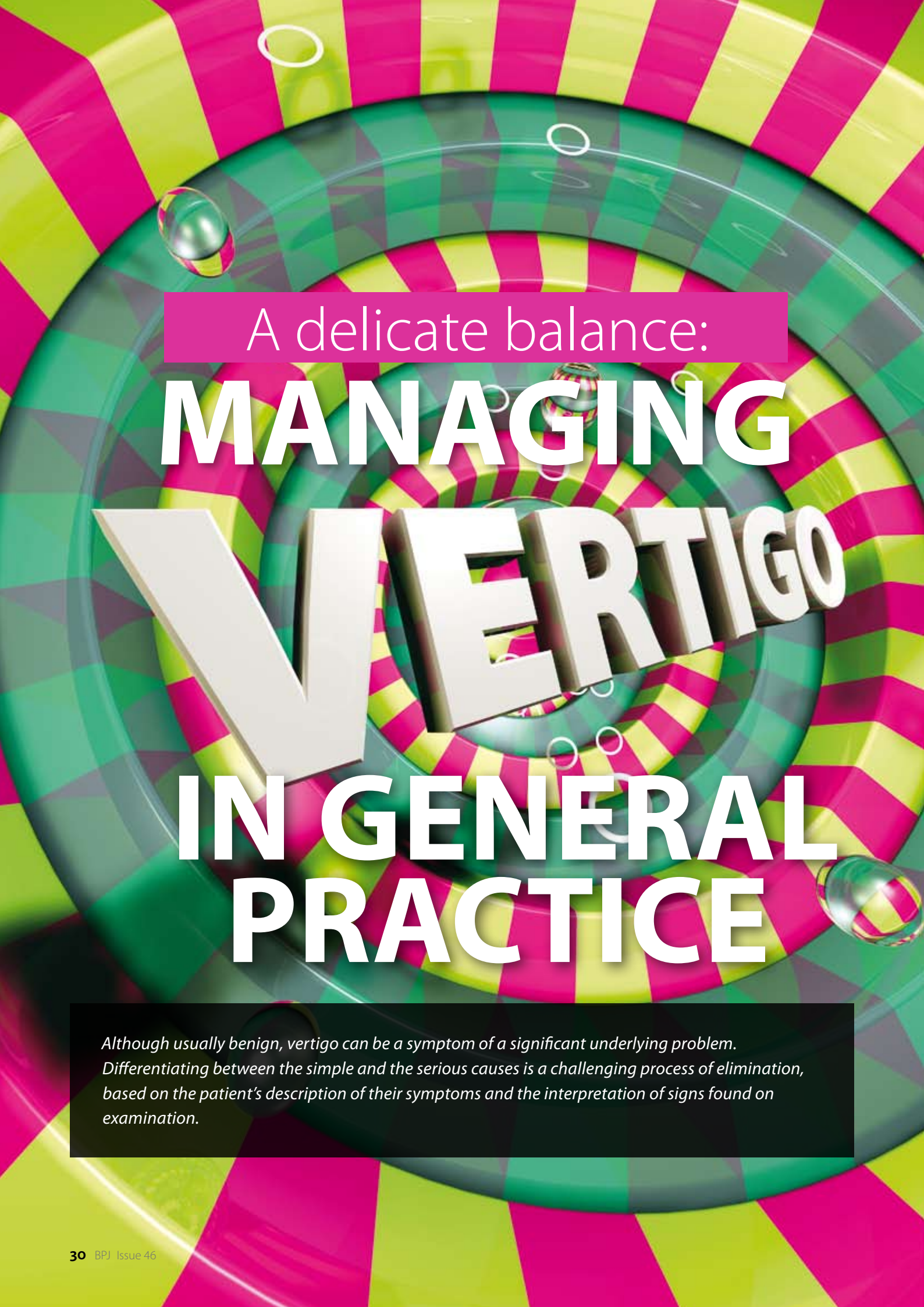
Vaccination can protect against otitis media

On 1 July 2011, two new pneumococcal vaccines that could potentially reduce the health burden of otitis media replaced Prevenar (PCV7) on the New Zealand immunisation schedule.[21] The routine immunisation schedule now includes Synflorix which covers ten pneumococcal serotypes and may provide protection against non-typeable *Haemophilus influenzae* (NTHi). NTHi is one of the main bacterial pathogens associated with acute otitis media.[21] Prevenar 13 is available for children at high risk of pneumococcal disease, e.g. immunodeficient or with cochlear implants. These children should also receive the pneumococcal polysaccharide vaccine Pneumovax 23 after age two years.

For further information see: "Pneumococcal vaccine for adults: Pneumovax 23", BPJ 35 (Apr, 2011).

References

1. Morris PS, Leach AJ. Acute and chronic otitis media. Pediatr Clin North Am 2009;56(6):1383–99.
2. Rovers MM, Schilder AGM, Zielhuis GA, Rosenfeld RM. Otitis media. Lancet 2004;363(9407):465–73.
3. Jackson G, Thornley S, Woolston J, et al. Reduced acute hospitalisation with the healthy housing programme. J Epidemiol Commun H 2011;65(7):588–93.
4. Kong K, Coates H. Natural history, definitions, risk factors and burden of otitis media. Med J Aust 2009;191(9):S39–43.
5. Shaikah N, Hoberman A, Kaleida P, et al. Diagnosing otitis media - otoscopy and cerumen removal. N Engl J Med 2010;(20):e62.
6. Onusko E. Tympanometry. Am Fam Physician 2004;70(9):1713–20.
7. Morris P, Leach A. Managing otitis media: an evidence based approach. Aust Prescr 2009;32:155–9.
8. Hoberman A, Paradise JL, Rockette HE, et al. Treatment of acute otitis media in children under 2 years of age. N Engl J Med 2011;364(2):105–15.
9. Diagnosis and management of acute otitis media. Pediatrics 2004;113(5):1451–65.
10. New Zealand Formulary (NZF). NZF v2.1. NZF, 2012. Available from: www.nzf.org.nz (Accessed Aug, 2012).
11. Paterson JE, Carter S, Wallace J, et al. Pacific Islands families study: the prevalence of chronic middle ear disease in 2-year-old Pacific children living in New Zealand. Int J Pediatr Otorhinolaryngol 2006;70(10):1771–8.
12. Nash E. Otitis media. InnovaiT 2009;2(3):172–7.
13. Gasper K. Clinical inquiries: are antibiotics effective for otitis media with effusion? J Fam Pract 2003;52(4):321–3.
14. Harris C. Childhood ENT disorders: when to refer to specialists. Aust Fam Phys 2002;31(8):1–5.
15. McDonald S, Langton Hewer CD, Nunez DA. Grommets (ventilation tubes) for recurrent acute otitis media in children. Cochrane Database Syst Rev 2008;(4):CD004741.
16. Browning GG, Rovers MM, Williamson I, et al. Grommets (ventilation tubes) for hearing loss associated with otitis media with effusion in children. Cochrane Database Syst Rev 2010;(10):CD001801.
17. National Institute for Health and Clinical Excellence. Surgical management of otitis media with effusion in children: NICE clinical guideline 60. Available from: www.nice.org.uk/cg60 (Accessed Jul, 2012).
18. Simpson SA, Thomas CL, van der Linden MK, et al. Identification of children in the first four years of life for early treatment for otitis media with effusion. Cochrane Database Syst Rev 2007;(1):CD004163.
19. Kay DJ, Nelson M, Rosenfeld RM. Meta-analysis of tympanostomy tube sequelae. Otolaryngol Head Neck Surg 2001;124(4):374–80.
20. van der Veen EL, Schilder AGM, van Heerbeek N, et al. Predictors of chronic suppurative otitis media in children. Arch Otolaryngol Head Neck Surg 2006;132(10):1115–8.
21. Walls T, Best E, Murdoch D, Mills N. Vaccination to prevent otitis media in New Zealand. N Z Med J 2011;124(1340):6–9.



A delicate balance:

MANAGING VERTIGO IN GENERAL PRACTICE

Although usually benign, vertigo can be a symptom of a significant underlying problem. Differentiating between the simple and the serious causes is a challenging process of elimination, based on the patient's description of their symptoms and the interpretation of signs found on examination.

Vertigo and the vestibular system

Our sense of orientation and balance depends on input from the visual and proprioceptive systems and the inner ear, integrated in the brainstem vestibular nuclei and the cerebellum (Figure 1). In the inner ear, otolith organs in the vestibule detect vertical and non-rotational movement (orientation in relation to gravity), and the ampullary receptors in the semicircular canals detect rotation of the head. When the head rotates, the receptors on one side are stimulated and those on the opposite side are inhibited. The eyes attempt to keep a visual fixation on the environment with quick movements in the opposite direction, called the vestibulo-ocular reflex (VOR). At the same time, the vestibular nuclei send impulses to the limb and trunk muscles to contract and preserve balance. Dysfunction of any of these structures can cause disorders of balance and the sense of orientation, often leading to vertigo.

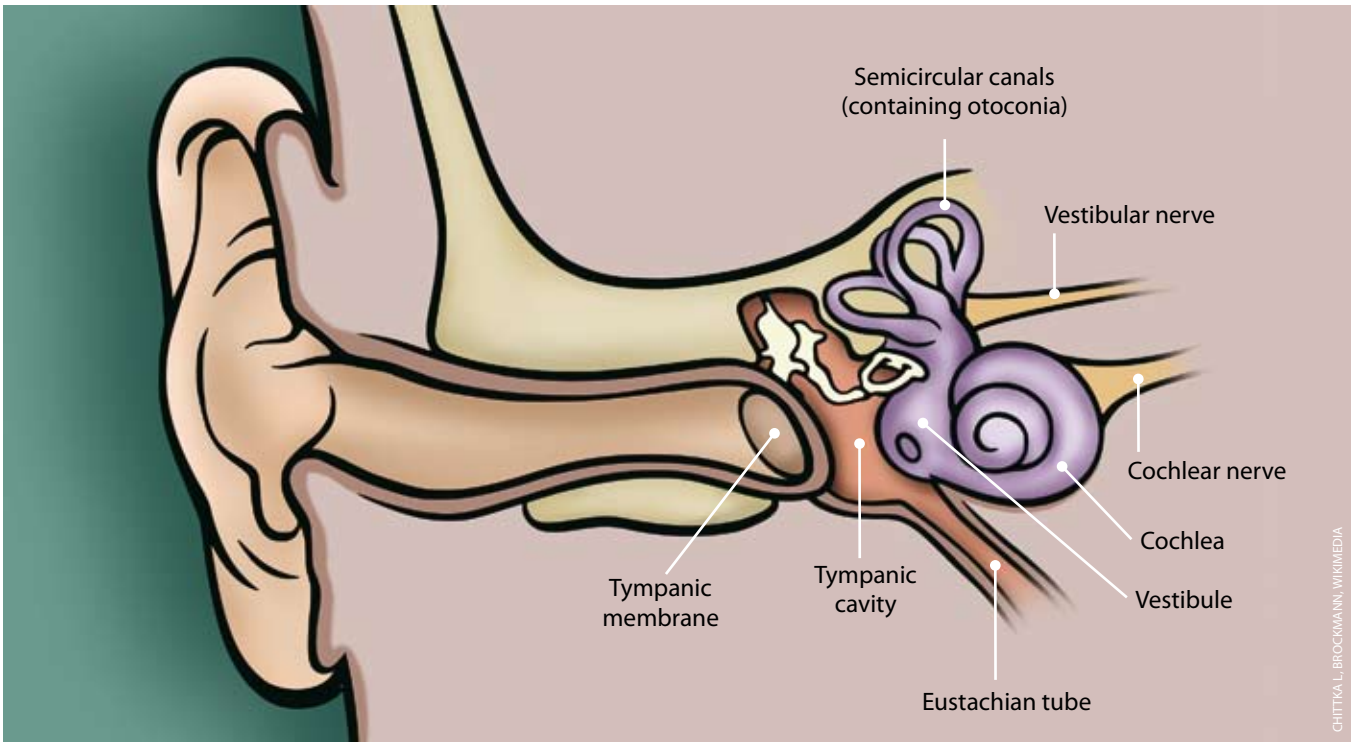


Figure 1: The inner ear

Table 1: Causes of vertigo (adapted from Kuo et al, 2008)

Peripheral vertigo	Central vertigo	Other
Common		
Acute vestibular failure (vestibular neuritis) Benign paroxysmal positional vertigo (BPPV) Ménière's disease	Vestibular migraine	Psychogenic vertigo
Rare		
Cholesteatoma Herpes zoster oticus Labyrinthitis	Cerebellopontine angle tumour (acoustic neuroma) Transient ischaemic attack (brainstem or cerebellum) Multiple sclerosis	Medicines

Vertigo is a symptom, not a diagnosis

Vertigo is a symptom, not a diagnosis. Peripheral vertigo arises from dysfunction of the vestibular labyrinth (semicircular canals) or the vestibular nerve.[1] Most patients presenting with vertigo in general practice have symptoms due to peripheral causes, such as benign paroxysmal positional vertigo (BPPV), vestibular neuritis, Ménière's disease or vestibular migraine (Table 1).[2] A small number of people with vertigo will have a significant, serious underlying condition, usually arising from a central cause such as stroke or a tumour, and will require urgent referral. It is vital not to miss such cases, and wherever there is doubt, refer.

Key questions to consider in a patient presenting with vertigo

When a patient presents with vertigo, assessment can be based on the following three judgments:

1. Are the symptoms being described most likely to be vertigo, dizziness or disequilibrium?
2. If it is vertigo, is the cause suspected to be central, peripheral or other?
3. Depending on the cause, what is the most appropriate management?

Is it vertigo or something else?

The first step should be to determine whether the patient's symptoms are due to vertigo, dizziness or disequilibrium. Ask the patient to explain the sensation in detail.

Vertigo is a sensation of motion (usually whirling) either of the body or the environment, caused by asymmetric dysfunction of the vestibular system.

Dizziness is a sense of spatial disorientation without a false sense of motion, often described as light-headedness, generalised weakness and feeling faint. When intense, it is usually called presyncope. Presyncopal dizziness usually has a cardiovascular cause and may be accompanied by other symptoms indicative of hypotension or anaemia, such as pale skin or "clamminess". Common aetiologies include arrhythmias, cardiomyopathy, hypovolaemia or orthostatic hypotension.[3]

Disequilibrium is a sense of being off-balance without dizziness or vertigo, particularly when walking. The patient may describe feeling as though the floor is tilted or that they are floating. It can originate in the inner ear or other sensory organs, from muscle and joint weakness or in the central nervous system. Disequilibrium is generally caused by bilateral dysfunction of the vestibular system, rather than unilateral dysfunction as seen in vertigo. Causes of disequilibrium include ototoxic loss of vestibular function, head trauma, cerebrovascular disease and progressive loss of vestibular function due to age or spinocerebellar degeneration, osteoarthritis or multiple sclerosis.[3]

Patients with dizziness or disequilibrium may require further testing, and a serious underlying cardiovascular cause or neurological disorder should always be considered. Further management of dizziness or disequilibrium is not covered in this article.

What is the cause of the vertigo?

A history is important for defining the aetiology of vertigo

Once the patient's symptoms have been established as vertigo, specific features are used to help determine the cause. The duration of each episode of vertigo is an important indication of the likely aetiology:[1,4]

- Seconds – likely to be psychogenic
- Less than one minute – likely to be BPPV
- Minutes – likely to be vascular/ischaemic
- Hours – likely to be Ménière's disease or vestibular migraine
- Hours to days – likely to be vestibular neuritis, central causes possible, e.g. stroke, vestibular migraine, multiple sclerosis
- Recurrent with headaches, photophobia and phonophobia – likely to be vestibular migraine

The patient should be asked about specific factors associated with onset of the vertigo. The most important is head position: is it triggered by lying down, rising, turning over in bed, looking up or stooping? Or does it start when they are upright and still?

Is there a recent history of a head injury, even if trivial?

Are they taking any new medicines, such as regular aspirin or phenytoin?

Are there other symptoms associated with the vertigo such as tinnitus, hearing loss or aural fullness (pressure) in one ear?

Are most episodes accompanied by headache? Is there a history of migraine?

An examination should help to confirm the cause

The examination should include:

Cardiovascular

- Heart rate and rhythm, with ECG if indicated by clinical findings: this is important for ruling out an underlying cardiac cause for the symptoms
- Blood pressure, standing and supine (three minutes for each position) – a significant drop in blood pressure, e.g. ≥20 mmHg systolic, when moving from supine to standing suggests presyncope rather than vertigo
- Auscultation of the neck – the presence of a carotid bruit particularly if there are neurological abnormalities on examination, may raise the suspicion of a central disorder, e.g. TIA or stroke

Otoscopic examination of the ears

Relevant findings include are signs of inflammation, infection, secretion or malodour, and signs of cholesteotoma or herpes zoster vesicles.

Best practice tip: When examining the ear with otoscopy, if there are any deposits at the top of the ear drum, it is more likely to be a cholesteotoma than wax build-up.

A focused neurologic examination

Initially, perform an assessment of the eyes, gait, balance and co-ordination and hearing. Further examination may be required, depending on the clinical picture, to detect any neurological signs that may point towards a central cause, e.g. motor or sensory changes in the face or upper limbs and tests of cerebellar function. If the vertigo is due to a peripheral cause, there should be no abnormal neurological signs other than nystagmus (see "Defining nystagmus") and possibly hearing loss.

- Examination of the eyes – e.g. presence of nystagmus, papilloedema
- Head impulse test – a test for the presence or absence of the vestibulo-ocular reflex (VOR) and a sign of unilateral vestibular dysfunction (see "Head impulse test for VOR", over page). An abnormal test significantly increases the likelihood of a peripheral cause.
- Assessment of gait, balance and co-ordination - Romberg tests, heel-toe tests and cerebellar testing, e.g. disdiadochokinesia, finger-nose tests. Poor balance or gait or cerebellar signs are significant red flags for a central cause.
- Basic initial hearing tests – N.B. a pure tone audiogram (to document fluctuating hearing in the affected ear) is essential for a symptom-based diagnosis of Ménière's disease and may indicate the presence of an acoustic neuroma. This will require referral for audiometry.

Specific positional testing

A Dix-Hallpike positional test (see "Performing a Dix-Hallpike test", Page 35) is essential for all patients presenting with, or with a history of, vertigo who do not have spontaneous nystagmus while upright.

Defining nystagmus

Nystagmus is the involuntary, rapid and repeated movement of the eyes. Nystagmus from a peripheral cause is usually horizontal (across the eye) with a slow component to the symptomatic side (affected ear) and a fast component (VOR) to the opposite side. The direction of the nystagmus is defined by the fast component, i.e. left, right, up or down-beating. In general, nystagmus that fatigues with time, goes away with fixation (e.g. asking the patient to stare at your finger), starts after a short delay and does not change direction with gaze is due to a peripheral cause. Vertical nystagmus is usually a sign of an underlying central lesion. The upward torsional nystagmus of BPPV is the only exception.

Red flags in vertigo diagnosis

Certain signs or symptoms indicate a possible serious underlying cause, for which the patient will require referral to hospital. Red flags include:[6]

- Vertigo that continues for several days
- Nystagmus that is down-beating and continuing
- Unremitting headache and nausea
- Ataxia, cerebellar signs
- Progressive hearing loss
- Signs of suppurative labyrinthitis – bulging, erythematous tympanic membrane, fever, balance disturbance

Symptomatic treatments for benign vertigo

In many cases, patients can be reassured that their symptoms are not associated with a serious cause and are satisfied to take a "wait and see" approach.

Antiemetics, e.g. prochlorperazine and cyclizine, are likely to be helpful to patients with spontaneous acute vertigo with nausea. They are generally well tolerated, but are associated with adverse effects such as drowsiness, dry mouth and blurred vision, so should be used with caution, particularly in elderly people. They should not be prescribed long-term.[9]

Benzodiazepines are not recommended, as they are likely to provide short-term symptomatic benefit, but they interfere with the natural central compensation in vestibular conditions and prolong vertigo.[10]

Based on the suspected cause, what is the most appropriate management?

Vertigo associated with a central disorder

The following features are indicative of a possible central cause:

- Recurrent or persistent vertigo
- Gait or movement abnormalities
- Constant nausea
- Poor performance on tests of cerebellar function, e.g. dysdiadochokinesis and heel-toe testing

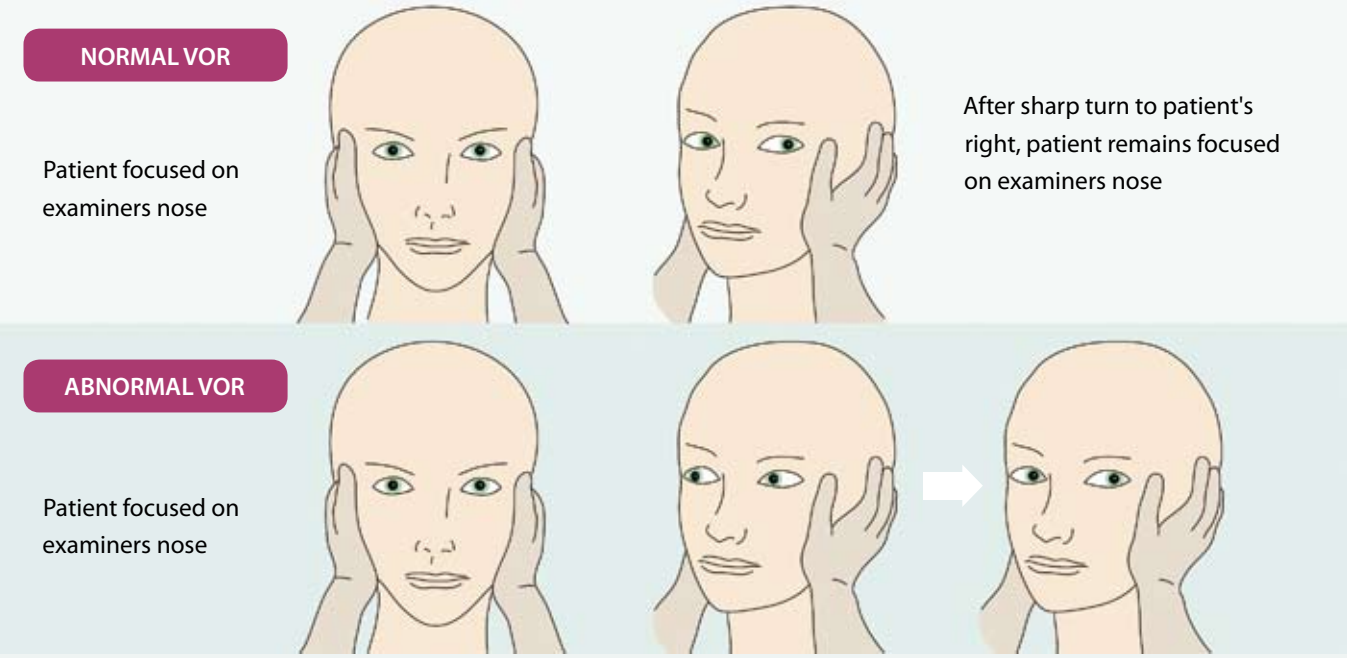
Anyone with a suspected central disorder should be urgently referred to hospital. Common causes of central-disorder vertigo include stroke, multiple sclerosis, vertebrobasilar ischaemia and tumours.

Benign paroxysmal positional vertigo (BPPV)

A positive Dix-Hallpike test is diagnostic of posterior canal BPPV. Horizontal canal BPPV is a less common form which can

Head Impulse Test for VOR

The Head Impulse Test is used to indicate the presence or absence of normal VOR.[5] It is commonly used in secondary care, but is a simple test that can also be carried out in the general practice setting. N.B. use with caution in people with cervical spine disease. The patient should be asked to sit upright and to stare at the examiner's nose without blinking. The examiner should turn the patient's head sharply and unpredictably to one side. The test is abnormal if the eyes make corrective saccades (rapid movement of both eyes to maintain focus on a point) to re-fix on the examiner's nose. This indicates a peripheral cause of the vertigo. In the illustration thrusting of the head to the right induces corrective saccades.



be seen as horizontal, direction-changing nystagmus as the patient's head is turned from side to side while supine.[4]

BPPV has a life-time prevalence of 2.4%, and most patients presenting to general practice with vertigo will have BPPV.[3, 6] BPPV occurs when otoconia (Figure 1, Page 31) in the vestibule of the inner ear become dislodged and enter the semicircular canals, usually the posterior canal.[7] In middle-aged and elderly people there is often no obvious cause, but in younger people it is usually due to head trauma – even mild trauma may be sufficient to displace otoconia.[8] BPPV may also be secondary to vestibular neuritis.

Treatment: Perform an Epley canalith repositioning procedure (See "Epley canalith repositioning procedure", over page). The success rate for the Epley procedure is approximately 70% on the first attempt, and almost 100% on successive manoeuvres.[8]

When there is no response to repeated repositioning manoeuvres, or atypical or ongoing nystagmus or nausea is present, a central cause should be suspected.[7] When there is unusual horizontal or down-beating nystagmus referral to an otolaryngologist is recommended.

Ménière's disease

Recurring episodes of vertigo, usually lasting for several hours, associated with fluctuating hearing, tinnitus and aural fullness is suggestive of Ménière's disease, an incapacitating disorder of the inner ear.[11] Ménière's disease is caused by an excess of cochlear endolymph (endolymphatic hydrops) which eventually "refluxes" into the semicircular canals to cause vertigo. As the vertigo episodes continue, hearing may decline to a "flat" sensorineural loss at 60 dB.[11] Ménière's disease usually occurs in people aged over 40 years, but in one-third of people it starts after age 60 years.[12]

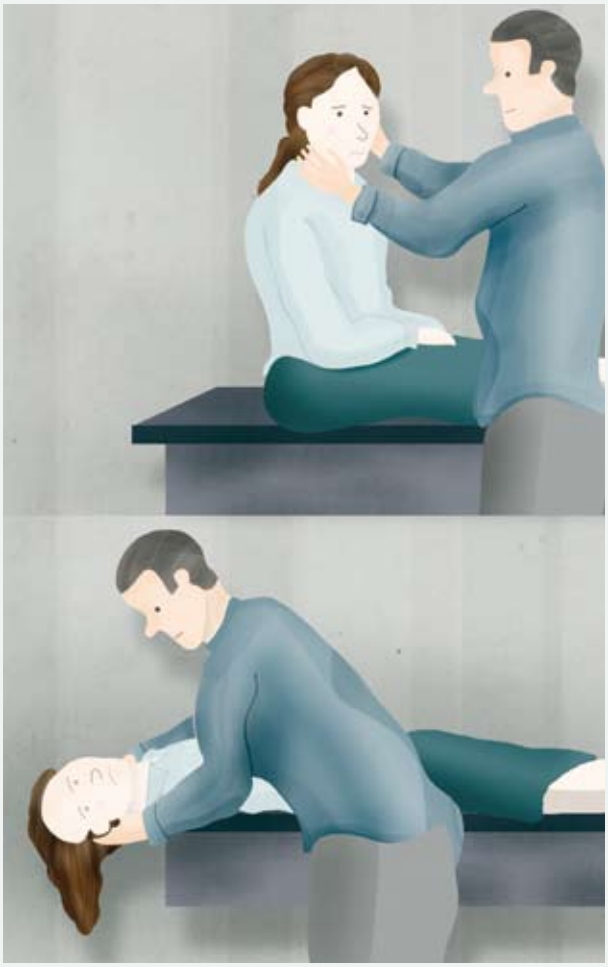
Diagnosis: Patients with suspected Ménière's disease should be referred for further investigation and confirmation of the diagnosis. Diagnosis is based on the classical symptoms and a pure tone audiogram test.[11] A MRI scan to exclude retrocochlear pathology is usually required. Some otolaryngology departments offer a specific electrophysiological test for hydrops. N.B. Ménière's disease and vestibular migraine can be easily confused.

Treatment: Currently there is no treatment which can reverse the hydrops and the hearing loss. The goal of management is symptom control.

Performing a Dix-Hallpike test

The Dix-Hallpike test is the diagnostic test for posterior canal BPPV. Before performing the test, warn the patient that the test is likely to trigger vertigo or nausea. The test can be performed on an examination table or bed. Older patients may find it easier to lie back with their shoulders on a pillow. The patient should be instructed to keep their eyes open. While still upright, turn the patient's head 45 degrees to one side, then lie them back with their neck extended over the head of the table/bed or pillow. A positive test must comprise a voluntary report of acute vertigo, and a delayed up-beating (towards the forehead) and torsional nystagmus which is anticlockwise if the right ear is affected and clockwise if the left ear is affected. The nystagmus should cease within 30 seconds. Sit the patient up.

Repeat the test on the opposite side. Ideally, test the suspected normal ear first and the suspected symptomatic ear second. If there is no nystagmus it is not BPPV.



Adapted from BMJ Best Practice[4]

The only oral medicine with some evidence of efficacy in controlling the vertigo episodes is betahistine.[13] The recommended maximum daily dose of betahistine is 48 mg/day (in divided doses), however, evidence suggests that significant benefit is derived from doses greater than this.[13] Diuretics have been used for Ménière's disease, but are not recommended as there is a lack of evidence of benefit, and adverse effects are possible, particularly in older people.[14]

Until recently, surgical treatment was the only alternative to pharmacological management.[9] However, intratympanic gentamicin is now being used by otolaryngologists.[15] Diluted gentamicin is placed in the middle ear through a myringotomy. The gentamicin is then absorbed into the inner ear. This does not treat the underlying pathology, but disables the semicircular canal receptors causing the vertigo episodes. A single treatment usually results in cessation of vertigo for several years.

Vestibular neuritis

A single, severe episode of vertigo, lasting at least 48 hours is suggestive of vestibular neuritis.[16] The principal signs are horizontal nystagmus and an abnormal head impulse test, which indicates a unilateral vestibulopathy. If the head impulse test is normal, cerebellar infarction should be suspected. Vestibular neuritis is thought to be caused by reactivation of the herpes simplex virus in the vestibular nerves.[1]

Treatment: Offer symptomatic treatment if required, e.g. if nausea is present, and consider referral if symptoms do not resolve or if there is any doubt about the diagnosis.

BPPV may occasionally develop in the affected ear after vestibular neuritis, due to inflammatory disruption of otoconia. Patients should be warned of the possibility of this complication and instructed to return for repositioning treatment if they experience positional vertigo.

Labyrinthitis

Labyrinthitis is an older term which is often misused for vestibular neuritis. However, a patient with acute otitis media who presents with vertigo, balance disturbance and hearing loss may have a viral or bacterial true labyrinthitis.[17] IV antibiotic treatment is usually required.[9]

Treatment: Refer to hospital.

Epley canalith repositioning procedure

This procedure can be performed in patients with BPPV. The goal of repositioning is to return the otoconia to their original position on the utricle. The procedure is successful in approximately 70% of patients on the first attempt, and approaches 100% effectiveness on successive manoeuvres. Repositioning is safe, but should be used with caution in people with cervical spine disease, unstable cardiovascular disease, suspected vertebrobasilar disease and high-grade carotid stenosis.

Tilt the patient back (neck extended over the end of the table/bed) with their head turned 45 degrees to the symptomatic side (as in a Dix-Hallpike test) and hold them in that position for one minute. Then turn their head to the opposite side at 45 degrees. Next, ask the patient to turn their hips and trunk (or assist them) until they are looking down at the floor at 135 degrees (180 degrees from the initial Dix-Hallpike position), so that the upper section of the posterior canal is vertical. After one minute ask the patient to sit up quickly with their head tilted toward the treated ear.

Older people may find it more comfortable to lie back over a pillow, rather than hanging their head over the end of the table.

A repeat Dix-Hallpike test should be done and if there is no response, treatment has been successful. However, it cannot be guaranteed that the repositioning has been successful. Younger patients can be instructed to test themselves at home in two days by lying back over a cushion on the floor. Older patients should be asked to return and be retested.

If repeated repositioning procedures are unsuccessful or if there is unusual and continuing nystagmus and nausea, referral is indicated.

Vestibular migraine

Recurrent, fluctuating vertigo that occurs with a throbbing headache, photophobia or transient visual symptoms is likely to be migraine related.[1] This occurs most frequently in people with a personal or family history of migraine.

Treatment: Treat as for migraine, if vertigo persists, reconsider the initial diagnosis (it is easily confused with Ménière's disease) and consider referral to an otolaryngologist.

Medicine-related vertigo

Many recreational drugs, most commonly alcohol, can cause short-term vertigo. Prescribed medicines almost never cause vertigo. However, many medicines, e.g. antihypertensives, can cause a fluctuating disequilibrium and this cause should always be considered.

Treatment: Trial cessation of the suspected medicine where possible.

Follow-up is essential for all people with vertigo

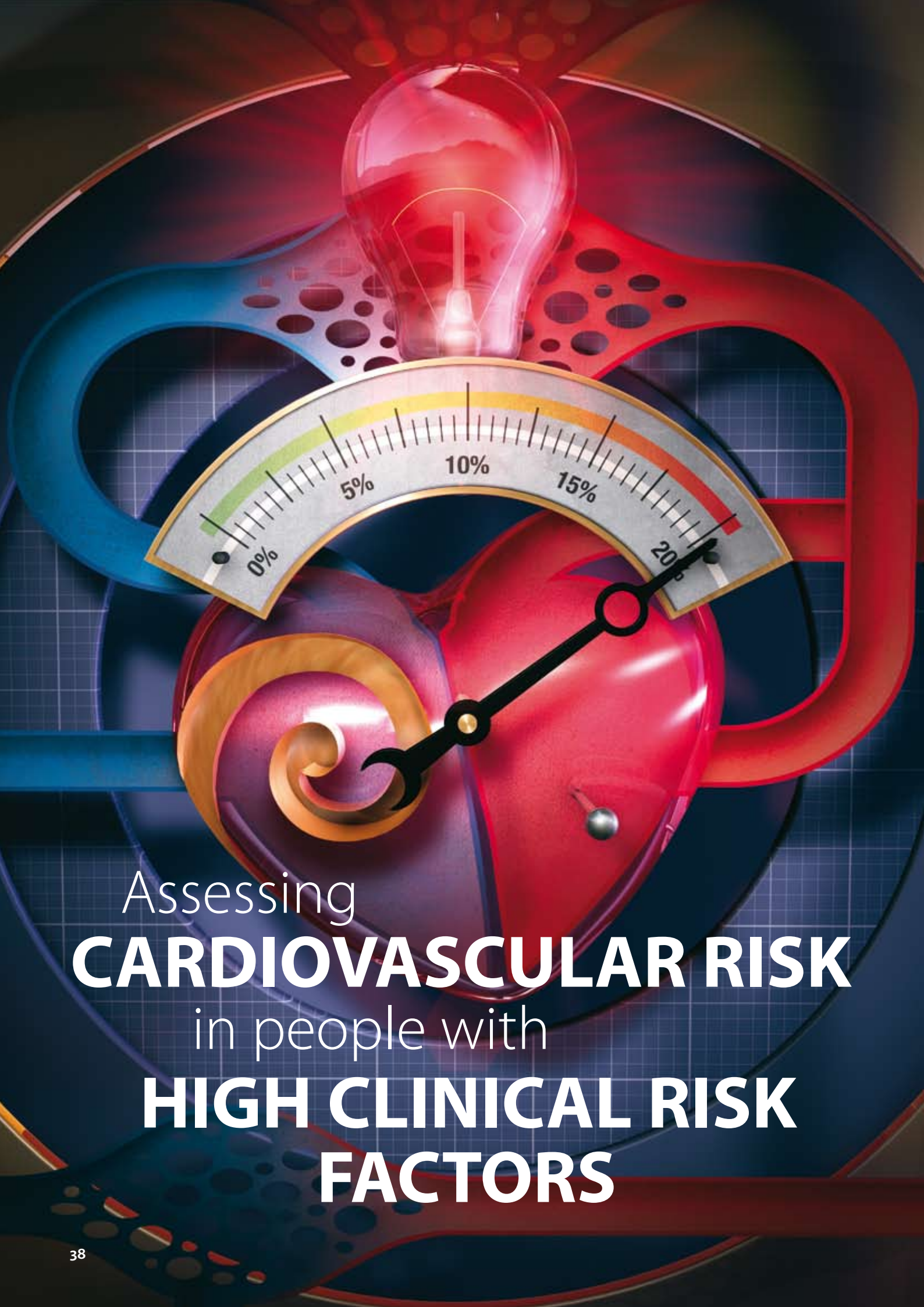
Continuing or worsening symptoms in people with vertigo may indicate an incorrect initial diagnosis or the possibility of a serious aetiology. Patients with vertigo should be instructed to return if their symptoms persist unexpectedly.

After otolaryngological diagnosis of a persisting and non-fluctuating peripheral vestibular disorder, vestibular rehabilitation may be beneficial.[18] This is a movement and exercise-based treatment offered in hospital physiotherapy departments and in some private clinics.

ACKNOWLEDGEMENT Thank you to **Professor Jeremy Hornibrook**, Otolaryngologist and Head and Neck Surgeon, Christchurch Hospital for expert guidance in developing this article.

References

1. BMJ Best Practice. Assessment of dizziness. BMJ; 2011. Available from: http://bestpractice.bmj.com/best-practice/monograph/71.html (Accessed Aug, 2012).
2. Hanley K, O'Dowd T. Symptoms of vertigo in general practice: a prospective study of diagnosis. Brit J Gen Prac 2002;52(483):809–12.
3. Mayo Foundation for Medical Education and Research. Dizziness. Mayo Clinic; 2010. Available from: www.mayoclinic.com/health/dizziness/ (Accessed Aug, 2012).
4. BMJ Best Practice. Benign paroxysmal positional vertigo. BMJ; 2011. Available from: http://bestpractice.bmj.com/best-practice/monograph/73.html (Accessed Aug, 2012).
5. Halmagyi G, Curthoys I. A clinical sign of canal paresis. Arch Neurol 1988;45(7):737–9.
6. Kuo C, Pang L, Chang R. Vertigo: Part 1 - assessment in general practice. Clin Pract 2008;37(5):341–7.
7. Hornibrook J. Benign paroxysmal positional vertigo (BPPV): history, pathophysiology, office treatment and future directions. Int J Otolaryng 2011;2011:835671.
8. Hilton M, Pinder D. The epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. Cochrane Database Syst Rev 2010;(2):CD003162.
9. Kuo C, Pang L, Chang R. Vertigo: Part 2 - management in general practice. Clin Pract 2008;37(5):341–7.
10. Bhattacharyya N, Baugh RF, Orvidas L, et al. Clinical practice guideline: Benign paroxysmal positional vertigo. Otolaryng Head Neck 2008;139(5, Suppl 4):S47–81.
11. American Academy of Otolaryngology-Head and Neck Foundation, Inc. Committee on hearing and equilibrium guidelines for the diagnosis and evaluation of therapy in Ménière's disease. Otolaryng Head Neck 1995;113(3):181–5.
12. BMJ Best Practice. Ménière's disease. BMJ; 2011. Available from: http://bestpractice.bmj.com/best-practice/monograph/155.html (Accessed Aug, 2012).
13. James A, Burton M. Betahistine for Ménière's disease or syndrome. Cochrane Database Syst Rev 2010;(1):CD001873.
14. Burgess A, Kundu S. Diuretics for Ménière's disease or syndrome. Cochrane Database Syst Rev 2009;(3):CD003599.
15. Pullens B, van Benthem P. Intratympanis gentamicin for Ménière's disease or syndrome. Cochrane Database Syst Rev 2011;(3):CD008234.
16. Strupp M, Brandt T. Chapter 25 - Vestibular neuritis. Vertigo and imbalance: Clinical neurophysiologyof the vestibular system. Elsevier; 2010. p. 315–32. Available from: http://www.sciencedirect.com/science/article/pii/S1567423110090258 (Accessed Aug, 2012).
17. BMJ Best Practice. Labyrinthitis. BMJ; 2012. Available from: http://bestpractice.bmj.com/best-practice/monograh/72.html (Accessed Aug, 2012).
18. Yarley L, Donovan-Hall M, Smith H, et al. Effectiveness of primary care-based vestibular rehabilitation for chronic dizziness. Annal Int Med 2004;141(8):598–605.



Assessing **CARDIOVASCULAR RISK** in people with **HIGH CLINICAL RISK FACTORS**

Cardiovascular risk assessment tools automatically adjust risk to greater than 20% for people with high risk factors, e.g. a prior cardiovascular event or diabetes with overt nephropathy. This is leading to a blurring of the concept of primary and secondary prevention and in some cases, patients are not receiving the intensive interventions required as the perception is that their risk is always high and cannot be reduced. Although "high risk" people have a permanent risk of at least approximately 20%, many also have modifiable factors which increase their risk well beyond this level, and it is this risk that can be reduced.

Cardiovascular risk and the New Zealand guidelines

A person's cardiovascular risk (i.e. the risk that they will experience a cardiovascular event) is determined by a combination of modifiable and non-modifiable factors. New Zealand cardiovascular risk charts use the Framingham equation to incorporate the most significant of these factors into individualised five-year absolute cardiovascular risk assessments.[1] This approach allows for more accurate stratification of cardiovascular risk than can be achieved using clinical perception alone.[2] It also provides an important opportunity for clinicians to engage with patients over the issue of cardiovascular health.

Table 1: Cardiovascular risk factors and cardiovascular risk over time for a 61-year-old, European male with a myocardial infarction

Risk factor	Presentation	6 month follow-up	1 year follow-up
Smoker	Yes	Recently quit	No
Blood pressure	165/98	145/90	130/80
Total cholesterol	6.7	5.2	4.3
Triglycerides	2.1	1.7	1.4
HDL cholesterol	0.86	0.95	1.1
LDL cholesterol	4.9	2.7	1.9
Total chol/HDL ratio	7.8	5.5	3.9
Risk assessment			
Framingham risk (progress to target)	33%	22%	8%
Actual persisting clinical risk	>20%	>20%	>20%

Assessing people with a high clinical risk

In New Zealand, it is recommended that five-year cardiovascular risk should guide treatment decisions for variables such as blood pressure and lipid levels. However, in very high risk groups, the five-year risk is assumed to be above 20% for life, and the use of risk charts is not advised. This applies to people with a clinical history of:[1]

- Previous cardiovascular events: angina, coronary artery bypass grafting, ischaemic stroke, myocardial infarction, percutaneous coronary intervention, peripheral vascular disease, transient ischaemic attack
- Some genetic lipid disorders: familial hypercholesterolaemia, familial combined dyslipidaemia, familial defective apolipoprotein B and genetically very low HDL levels (some types)
- Diabetes with overt nephropathy
- Diabetes with other renal disease causing renal impairment

There may be a misconception that cardiovascular risk in these patients cannot be reduced, resulting in less aggressive treatment of risk factors. Although people in high risk groups have a cardiovascular risk of at least 20%, Framingham study-based tools can still play an important role in conveying the potential reduction of risk that improved risk factor management can provide to individuals, as well as in assessing progress made towards target levels. Emphasising this benefit to patients is likely to improve compliance with treatment.[3, 4, 5]

For example, Table 1 (previous page) shows risk calculation for a 61-year-old, European male, who has had a myocardial infarction and is followed up for one year.

In Table 1, the use of the Framingham study-based cardiovascular risk equation is helpful in conveying the potential benefit of risk factor management. The high cardiovascular risk at presentation illustrates the severity of the situation and the decreasing risk, as targets are approached, provides tangible progress and further motivation for the patient.

The Heart Foundation provides an online "heart forecast" tool, designed for health professionals to use with patients to demonstrate their current and future risk. Although this tool is not strictly designed for use in people at high risk, e.g. prior cardiovascular event, this is still a tangible way to show a patient how their risk changes when lifestyle factors change. The tool is available from: **www.heartfoundation.org.nz** Keyword search = heart forecast.

Modifying risk in people with cardiovascular disease

People who have had a prior cardiovascular event have a risk level approximately 20% higher than those with no prior event, however, this risk increases progressively with poor risk factor control.[5] Having a prior cardiovascular event significantly increases the risk of having another event and it is this group of patients who gain the most from preventative interventions. A New Zealand study found that in a group of over 35, 000 primary care patients, 10% had a prior cardiovascular event, but this group accounted for approximately 40% of the cardiovascular events among the cohort.[5]

Individual risk factors such as lipid profile, blood pressure and smoking status should be used as treatment targets for people with known cardiovascular disease.[1] These factors should be assessed every three to six months.[1] Intensive lifestyle changes that improve physical fitness and promote weight reduction should also be recommended.[1]

Treating to target

Most patients who have had a prior cardiovascular event will have had medicines initiated in secondary care. The role of the primary care team is to ensure that the patient is concordant with their medicines, to adjust doses as required and to recommend lifestyle changes to reduce cardiovascular risk. As a rule, the greater an individual's cardiovascular risk, the more aggressive the treatment should be.[1]

Statin treatment is recommended first-line for dyslipidaemia.[1] In some cases, a fibrate may be considered in combination with a statin, e.g. in people with high triglyceride levels or low HDL-cholesterol levels. Table 2 shows the New Zealand cardiovascular guidelines optimal lipid targets for people with known cardiovascular disease, diabetes or a cardiovascular risk calculated to be over 15%.

For further information see: "An update on statins", BPJ 30 (Aug, 2010).

Antihypertensive medicines are indicated for all patients with an average blood pressure ≥ 170/100 mm Hg. The recommended blood pressure targets are:[1, 6]

- < 140/85 mm Hg for people without clinical cardiovascular disease
- < 130/80 mm Hg for people with diabetes or cardiovascular disease
- < 125/75 mm Hg if estimated kidney protein loss is

Table 2: Lipid targets for people with known CVD, adapted from NZGG (2011)[1]

Lipids	
LDL cholesterol*	< 2.0 mmol/L
HDL cholesterol	≥ 1.0 mmol/L
Total cholesterol (TC)	< 4.0 mmol/L
TC : HDL ratio	< 4.0
Triglycerides	< 1.7 mmol/L

*LDL cholesterol is the primary lipid indicator for management of cardiovascular risk

greater than 1 g in 24 hours (i.e. urine protein/creatinine >100 mg/mmol or urine albumin/creatinine > 70 mg/mmol)

Glycaemic control in people with type 2 diabetes is important for preventing microvascular complications, e.g. retinopathy, nephropathy, neuropathy. Macrovascular complications, e.g. coronary artery disease, stroke and peripheral vascular disease, may also be reduced if glycaemic control begins early,[7] along with management of other risk factors. Some research has found that the macrovascular benefits provided by metformin are independent of its blood glucose lowering effect,[8] however, evidence is inconclusive at this stage.

A HbA_{1c} level of 50 – 55 mmol/mol is recommended, or a target as individually agreed.[7] When setting an HbA_{1c} target, it is important to consider the age of the patient, their motivation, and the risks and consequences of hypoglycaemia and potential weight gain if treatment is intensified.[7] In younger people, tighter glycaemic control should be considered due to an increased lifetime risk of experiencing diabetes related complications.[7]

For further information see: "HbA_{1c} targets in people with type 2 diabetes" BPJ 30 (Aug, 2010).

Lifestyle interventions

After a cardiovascular event, motivational interviewing ("Motivational interviewing", BPJ 17, Oct, 2009) can be used to establish goals for lifestyle changes that are in keeping with a person's readiness to improve their health.[1] Involving the patient's family (whānau) in this conversation, with patient consent, can also be beneficial. Many general practices have

The PHO Performance Programme

The PHO Performance Programme aims to improve health outcomes and reduce disparities for all people using primary care health services in New Zealand. Financial payments are used as incentives to improve PHO performance as measured against indicators. Ischaemic CVD detection and CVD risk assessment are two of the seven funded indicators for chronic conditions in New Zealand.

The target for ischaemic CVD is for 90% of enrolled people aged between 30 – 79 year with ischaemic CVD to have been identified and coded in their patient notes.[12] The denominator (i.e. what the results are compared against) is calculated by adjusting the national prevalence of ischaemic CVD to account for the age, gender and ethnicity of individual PHO populations.[12]

The target for CVD risk assessment is for 80% of enrolled and eligible people to have had their CVD risk assessed and recorded in their patient notes within the last five years.[12] The denominator for this indicator is the number of enrolled people in the PHO who are eligible for a CVD risk assessment:[12]

- Māori, Pacific and Indian subcontinent males aged 35 – 74 years
- Māori, Pacific and Indian subcontinent females aged 45 – 74 years
- Males of all other ethnicities aged 45 – 74 years
- Females of all other ethnicities aged 55 – 74 years

For further information see: "Ischaemic cardiovascular disease", BPJ 36 (Jun, 2011) and "Cardiovascular disease risk assessment", BPJ 37 (Aug, 2011).

nurse-led clinics that allow time to assist with education around lifestyle interventions.

Physical activity is essential for people at high risk of a cardiovascular event, however, advice should be tailored to individual circumstances. In general:

- The recommended activity level for an adult is at least 30 minutes of moderate to vigorous activity per day, e.g. brisk walking
- Exercise-based cardiac rehabilitation can reduce mortality by one fifth to one third[9]
- People with CVD should include five minutes of warm-up and cool-down in their exercise sessions[10]
- Vigorous physical activity, e.g. aerobics, fast cycling, running or swimming, is not recommended for people with impaired left ventricular function, severe coronary artery disease, recent myocardial infarction, significant ventricular arrhythmias or stenotic valve disease[1]
- Following angina, coronary artery bypass grafting, myocardial infarction or percutaneous coronary intervention, patients should be referred to a cardiac rehabilitation programme[1]

Smoking cessation is strongly encouraged in any person who continues to smoke after a cardiovascular event. The following general advice applies:

- Nicotine replacement therapy (NRT) approximately doubles a smoker's chance of quitting,[1] bupropion also approximately doubles a smoker's chance of quitting and varenicline (available under Special Authority) approximately triples this chance
- NRT can be safely used by people with cardiovascular disease, however, in the acute phase following myocardial infarction or stroke, oral NRT should be prescribed in preference to patches as nicotine levels can be reduced more rapidly if an adverse event occurs[1]
- Bupropion can be safely used in people with cardiovascular disease
- People with established cardiovascular disease using varenicline may have a slightly increased risk of experiencing a cardiovascular event, however, the risk is likely to be greater if they remain a smoker.[1] Varenicline has also been associated, in rare cases, with neuropsychiatric adverse effects.
- Nortriptyline is effective for smoking cessation, but it is contraindicated in the acute phase following myocardial infarction, as it can affect cardiac conductivity[11]

Best practice tip: Through discussion, find out what motivates or interests your patient, to improve their health; such as children (tamariki), grand children (mokopuna), family (whānau), pets, gardening or bowls. Encourage patients to have support – take a friend, find a "buddy" who wants to quit smoking as well. Consider establishing a buddy system through the general practice clinic.

References

1. New Zealand cardiovascular guidelines handbook: A summary resource for primary care practitioners. 2nd ed. Wellington: New Zealand Guidelines Group; 2009.
2. Mosca L, Linfante AH, Benjamin EJ, et al. National study of physician awareness and adherence to cardiovascular disease prevention guidelines. Circulation 2005;111(4):499–510.
3. Stafford L, Jackson HJ, Berk M. Illness beliefs about heart disease and adherence to secondary prevention regimens. Psychosom Med 2008;70(8):942–8.
4. Mann DM, Allegrante JP, Natarajan S, Halm EA, Charlson M. Predictors of adherence to statins for primary prevention. Cardiovasc Drugs Ther 2007;21(4):311–6.
5. Kerr A, Broad J, Wells E, et al. Should the first priority in cardiovascular risk management be those with prior cardiovascular disease? Heart 2009;95:125–9.
6. Kidney Health New Zealand. Chronic kidney disease (CKD) management in General Practice. Available from: www.kidneys.co.nz (Accessed Aug, 2012).
7. New Zealand Guidelines Group. Guidance on the management of type 2 diabetes 2011. Wellington: New Zealand Guidelines Group; 2011. Available from: www.nzgg.org.nz (Accessed Aug, 2012).
8. Bailey CJ. Metformin: effects on micro and macrovascular complications in type 2 diabetes. Cardiovasc Drugs Ther 2008;22(3):215–24.
9. Heran BS, Chen JM, Ebrahim S, et al. Exercise-based cardiac rehabilitation for coronary heart disease. Cochrane Database Syst Rev 2011;(7):CD001800.
10. Thompson PD, Franklin BA, Balady GJ, et al. Exercise and acute cardiovascular events placing the risks into perspective: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism and the Council on Clinical Cardiology. Circulation 2007;115(17):2358–68.
11. Ministry of Health. New Zealand smoking cessation guidelines. Wellington: Ministry of Health; 2007. Available from: www.health.govt.nz (Accessed Aug, 2012).
12. DHBNZ. PHO Performance Programme. Indicator definitions. Version 5.5. 2012. Available from: www.dhbnz.org.nz (Accessed Aug, 2012).

Guidance for stopping alendronate

Dear Editor,

A patient recently asked me "How long should I stay on alendronate?" A very good question that had me struggling. A local rheumatologist has stopped alendronate in several of my patients after five years. "A practical guide to stopping medicines in older people" (BPJ 27, Apr 2010) mentioned alendronate as a medication that could be considered for possible cessation in the elderly, but gave no clear guide on who and when. My limited reading suggests that a holiday from alendronate should be considered after five years in most and ten years in the rest, as alendronate has an ongoing effect after its cessation, the maximal bone strength is attained at three years and additional risk of atypical fracture occur after that duration. I am unsure what to do here? This is an expensive medication and its unnecessary use would be good to eliminate. Wonder if you can answer my questions? What is the optimal duration of alendronate treatment, in whom should we stop it and what monitoring is required?

Brian Scrimshaw
General Practitioner, Wanganui

Treatment with a bisphosphonate, such as alendronate, has proven benefits in terms of the prevention of bone loss and the reduction of fractures in males or post-menopausal females with osteoporosis. However, alendronate is associated with adverse effects such as oesophagitis, oesophageal ulcers and strictures, as well as a very small increased risk of osteonecrosis of the jaw and atypical femur fractures.[1] Therefore the benefits vs. risks of alendronate treatment must be carefully weighed up and regular review should take place.

There is currently a lack of evidence to form a consensus on the optimal length of alendronate treatment and when, if ever, it should be stopped, and for how long. Many clinicians recommend that alendronate should be interrupted periodically. In theory, this is to allow recovery of bone turnover, which is suppressed during treatment, but it is unknown whether this suppression contributes to the rare adverse effects associated with alendronate.[1] The beneficial effect of alendronate remains for three to five years after ceasing treatment.

In a patient who has taken alendronate for five years and whose bone density is no longer in the osteoporotic range, discontinuing alendronate is a reasonable approach. The patient is likely to have substantial residual anti-resorptive activity during this period. N.B. this can be checked through the measurement of serum P1NP, with a value < 35 µg/L indicative of significant inhibition of bone resorption, however, this test is not usually carried out in general practice. Bone density and fracture risk can be re-evaluated (using DEXA scan) after two years off treatment, and alendronate resumed in patients with a 10-year hip fracture risk greater than 3% (calculated using FRAX).

If patients are still at high risk after five years of alendronate treatment (bone mineral density remains low, fragility fracture has occurred), the risk of stopping treatment is likely to exceed the risk of continuing.[1]

In a recent perspective article in the New England Journal of Medicine, the authors concluded the following, based on the limited evidence about long-term alendronate use:[2]

- Patients with bone density T-scores of −2.5 or below at the femoral neck, after three to five years of treatment, benefit the most from continuation
- Patients with bone density T-scores between −2.5 to −2.0 and an existing vertebral fracture, after three to five years of treatment, may also benefit from continuation
- Patients with bone density T-scores above −2.0 at the femoral neck, after three to five years of treatment, are unlikely to benefit from continuation

The authors also note that reduced doses may be considered if alendronate is continued beyond five years.[2]

ACKNOWLEDGEMENT Thank you to **Professor Ian Reid**, Professor of Medicine and Endocrinology, Faculty of Medical and Health Sciences, University of Auckland for expert guidance in preparing this answer.

We value your feedback. Write to us at:
Correspondence, PO Box 6032, Dunedin
or email: editor@bpac.org.nz

References

1. Khosla S, Bilezikian J, Dempster D, et al. Benefits and risks of bisphosphonate therapy for osteoporosis. J Clin Endocrinol Metab 2012;97:2272-82.
2. Black D, Bauer D, Schwartz A, et al. Continuing bisphosphonate treatment for osteoporosis – for whom and for how long? N Engl J Med 2012;366(22):2051-3.

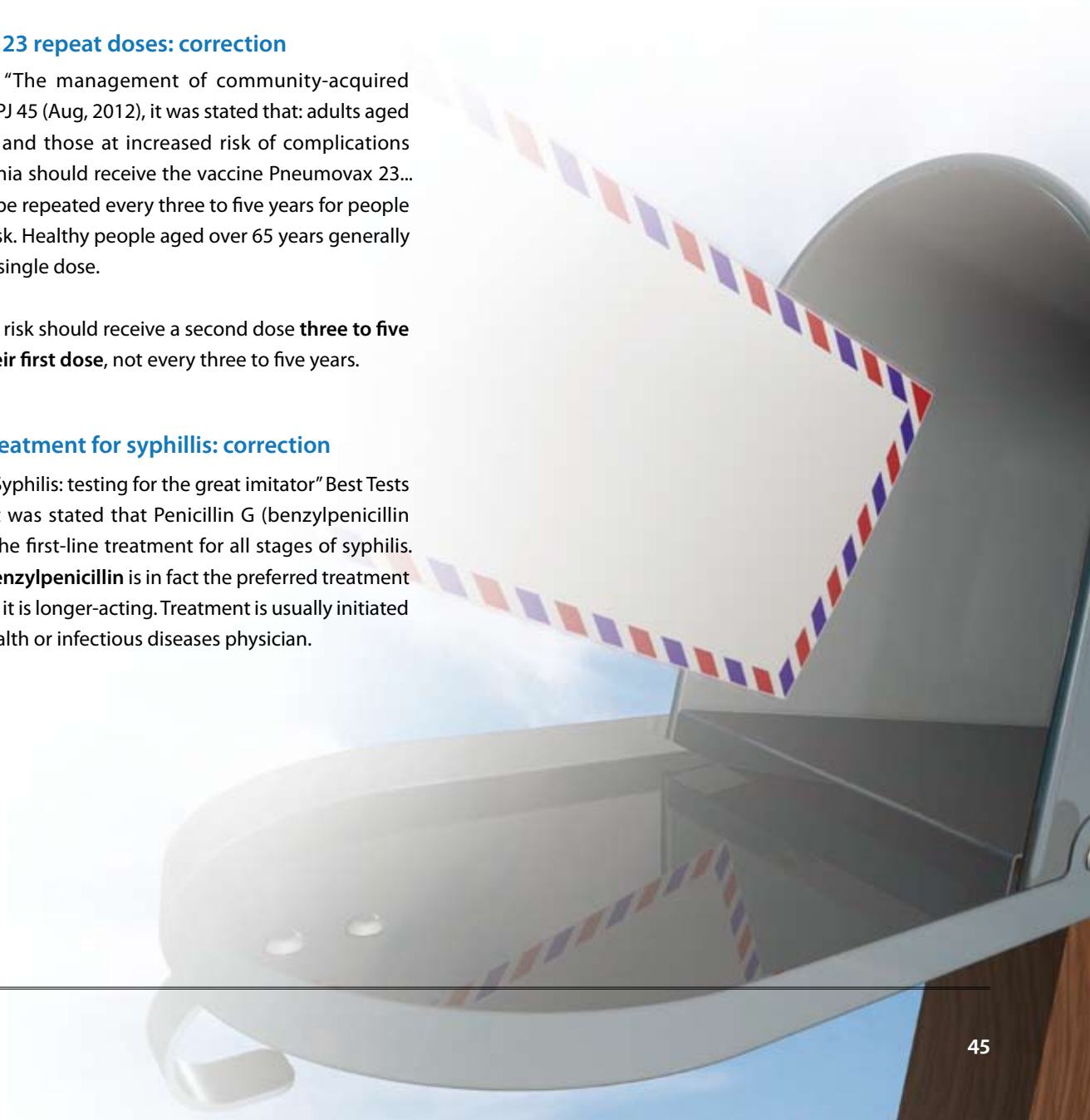
Pneumovax 23 repeat doses: correction

In the article "The management of community-acquired pneumonia" BPJ 45 (Aug, 2012), it was stated that: adults aged over 65 years and those at increased risk of complications from pneumonia should receive the vaccine Pneumovax 23... Doses should be repeated every three to five years for people at increased risk. Healthy people aged over 65 years generally only require a single dose.

People at high risk should receive a second dose **three to five years after their first dose**, not every three to five years.

Antibiotic treatment for syphillis: correction

In the article "Syphilis: testing for the great imitator" Best Tests (June 2012), it was stated that Penicillin G (benzylpenicillin sodium) was the first-line treatment for all stages of syphilis. **Benzathine benzylpenicillin** is in fact the preferred treatment at all stages, as it is longer-acting. Treatment is usually initiated by a sexual health or infectious diseases physician.



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