BEST PRACTICE

21

JUNE 2009

Low back pain
Antibiotics
Influenza

bpac nz
better medicine

We would like to acknowledge the following people for their guidance and expertise in developing this edition:

Mr Chris Hoffman, Wellington

Dr Rosemary Ikram, Christchurch

Dr John MacVicar, Christchurch

Dr Susan Taylor, Auckland

Assoc. Professor Mark Thomas, Auckland

Dr Nikki Turner, Auckland

Dr Neil Whittaker, GP Reviewer, Nelson

Best Practice Journal (BPJ)

ISSN 1177-5645

BPJ, Issue 21, June 2009

BPJ is published and owned by bpac^{nz} Ltd
Level 8, 10 George Street, Dunedin, New Zealand.

Bpac^{nz} Ltd is an independent organisation that promotes health care interventions which meet patients' needs and are evidence based, cost effective and suitable for the New Zealand context.

We develop and distribute evidence based resources which describe, facilitate and help overcome the barriers to best practice.

Bpac^{nz} Ltd is currently funded through contracts with PHARMAC and DHBNZ.

Bpac^{nz} Ltd has five shareholders: Procare Health, South Link Health, IPAC, the University of Otago and Pegasus Health.

Contact us:
Mail: P.O. Box 6032, Dunedin
Email: editor@bpac.org.nz
Free-fax: 0800 27 22 69

www.bpac.org.nz

This magazine is printed on an environmentally responsible paper managed under the environmental management system ISO 14001, produced using Certified ECF pulp sourced from Certified Sustainable & Legally Harvested Forests.

CONTENTS

CONTENTS

CONTENTS

Essentials

All web links in this journal can be accessed via the online version.

www.bpac.org.nz

www.bpac.org.nz keyword: funding

The Funding Maze

A Clinical Pathologist's Perspective

Contributed by Dr Rosemary Ikram, Clinical Microbiologist, Medlab South Ltd

The role of a Clinical Pathologist has always been a fascinating one. We develop expertise in testing; when, where and how this should occur, these days called best practice. We ensure quality is maintained which is monitored by International Accreditation New Zealand (IANZ) and advise referrers on the best treatments and preventative measures. For a Clinical Microbiologist this includes immunisation and prevention of healthcare associated infection.

In recent years the dogma related to efficiency has become the perceived wisdom in the pathology sector. Efficiency has always been an essential component of operating a community laboratory service. Even before the vogue of bulk funding pathology services New Zealand had the cheapest community pathology tests when compared to the USA, Canada, Australia and the UK. This gap will have increased considerably with bulk funding.

So what? You say. This is all good and the money saved can be ploughed back into other health sectors. In my area of expertise there are PHO programmes and funding for antenatal HIV screening, screening for Chlamydia infection, increasing uptake of immunisation and quality initiatives in infection control. These are all projects which many colleagues have discussed for years. So why don't we just get on with it and stick to our knitting? Believe me we would love to do just that.

Firstly there is the antenatal HIV screening. This is an important programme. Nobody would consider that funding HIV positive pregnant women and treating them, to prevent transmitting this infection to their infants is a bad idea. This has already been piloted. Now is the time for the rest of us to start. Each DHB has someone to coordinate this. We need to discuss it, in my case I have three DHBs to liaise with. We ask where the funding for doing the tests is coming from, nobody knows. It is difficult to believe that a programme so long in gestation has not allocated funds for the testing.

This is not an isolated instance. In their recent programme bpac encouraged more screening for *Chlamydia trachomatis*. Agreed it is important to do this, but who is going to fund the extra tests? If the funding is not forthcoming then the only way forward in the short term, is for the laboratories to charge the patient which will decrease the number of patients screened, and jeopardise the programme's success.

It is difficult to believe that these programmes are planned without allocating funding for the tests. It is absolutely impossible to imagine that there is an expectation that the testing be squeezed into the already lean bulk funded pathology contracts. The increased number of tests will be considerable.

The PHO Performance management programme also has a similar disconnect. Influenza vaccination uptake by the "at risk" population is a performance indicator. Only patients who are vaccinated by the general practice can be counted. This means that if a patient is vaccinated while in hospital it will not "count" and therefore reduce the chance of the local PHO reaching its target and claiming the accompanying funding. Therefore a measure which is aimed at improving vaccination coverage, is in conflict with a measure which is in itself known to do this. This indicates a lack of overall appreciation of factors which can influence vaccination rates.

Infection control initiatives are also suffering from a similar syndrome. Hand Hygiene New Zealand is introducing a programme to all DHBs. Hand hygiene has to be good, and so say all of us who have been running programmes for years. The New Zealand programme involves "the five moments of hand hygiene". These "moments" are to be audited by "platinum" and "gold" auditors who have to be flown around the country to train, and then spend hours auditing. This programme has been imported from healthcare systems with more health dollars than New Zealand. Will it succeed? The jury is out, but it is well recognised that continued success of such programmes relies on the benefits being maintained.

All the above programmes are laudable and could result in positive health outcomes. Some aspects such as funding and communication are neglected which can jeopardise the outcome. More consultation with all stakeholders in the planning stages of these programmes would improve their implementation and credibility. After all some of us have been advising, testing and educating on these issues for years.

The views expressed in this article are the personal views of the author and should not be assumed to reflect a particular organisation.

Acute low back pain

Key reviewers:
Mr Chris Hoffman, Orthopaedic Surgeon, Mana Orthopaedics, Wellington

Dr John MacVicar, Medical Director, Southern Rehab, Christchurch

Key concepts:

- Acute low back pain is common and most patients will recover fully within three months
- Serious causes are rare and can be excluded with careful history and examination
- Radiological studies are not required for acute low back pain in the absence of red flags
- An exact diagnosis is often not possible, nor needed for management
- Patients' beliefs and attitudes warrant as much attention as the anatomical and pathological aspects of their condition
- Fear about pain is a major determinant of disability and possible chronicity
- Management should include reassurance, education and helping the patient stay active
- Adequate analgesia is important to allow the patient to stay active

www.bpac.org.nz keyword: lowbackpain

Acute low back pain is common and often relapsing

Low back pain is discomfort, muscle tension or stiffness localised to the area around the lumbar spine. Back pain may radiate to the groin, buttocks or legs as referred somatic pain and may be associated with lumbar radicular pain such as sciatica.

In any given year approximately one third of adults will suffer from low back pain and one third of these will seek help from a health practitioner.[1] Most people with low back pain self-treat with over-the-counter medications and lifestyle changes.[2]

Low back pain is described as acute if present for less than six weeks, sub-acute between six weeks and three months, and chronic if it continues for longer than three months.

Low back pain varies in severity and associated disability. Most episodes of acute, non-specific low back pain resolve within two weeks. 70–90% of patients will recover fully from an acute episode within three months.[3, 4] However, subsequent relapse is common and many individuals will have recurring episodes of acute low back pain.

Only a small group will go on to suffer from chronic pain and disability.

Acute low back pain can be separated into three categories

The aim of the history and examination is to separate people with acute low back pain into three categories. Those with:[5]

- Serious pathology (red flags – see box)
- Radicular nerve involvement
- Non-specific back pain (this is a diagnosis of exclusion)

Red Flags:

- Trauma
- Unrelenting pain, or pain worse at night (supine)
- Age <20 years, or new back pain age >50 years
- History of cancer
- Systemic symptoms
- IV drug use
- Immunosuppression or steroids
- Widespread or progressive neurological deficit

Serious causes of acute low back pain are rare and include:[6]

- Osteoporotic or trauma related vertebral fracture (4%)
- Cancer involving the lumbar spine (0.66%)
- Inflammatory disease such as ankylosing spondylitis (0.3%)
- Spinal osteomyelitis associated with IV drug use, urinary tract infection or skin infection (0.01%)

Key history for acute low back pain

It is important to determine:

- Onset and duration of pain
- Site and radiation
- Precipitating and relieving factors
- Severity and functional impact
- Any neurological deficit
- Any symptoms of systemic illness

Onset and duration

Patients may recall a specific event that triggered their acute low back pain, however it can frequently occur for no apparent reason, or after ordinary activity.

A history of trauma, such as a fall or motor vehicle accident, may indicate vertebral fracture or sacro-iliac joint problems.

Pain that develops slowly may indicate serious pathology.

Site of the pain and radiation

Many people have pain only in their back. If there is associated leg pain it may be somatic referred pain or radicular (neurogenic) pain.

For people who present with back and leg pain, determine which pain is dominant. One way to check this is to ask, "Which pain would you like to be rid of first?"[7]

When the leg pain is dominant it is more likely to be radicular in origin. Radicular pain is often described as shooting or stabbing, like an "electric shock" and may be associated with pins and needles or numbness. Somatic referred back pain is usually dull in nature, "like a toothache" (Table 1). Both types of pain may co-exist.

Precipitating and relieving factors

Typically non-specific back pain feels better at rest and worse with activity. The opposite occurs with the inflammatory arthritides such as ankylosing spondylitis. Patients with disc disorders may find prolonged sitting or forward flexion aggravates symptoms. Leg dominant pain that resolves with flexion and sitting and worsens with extension may be claudicant pain from spinal stenosis (if normal lower limb pulses).

Severity and functional impact

What effect is the pain having on activities or sleep? Severe unremitting pain, especially if sleep is disturbed, is a red flag. A numerical or functional scale to assess the severity of the back pain and to help monitor progress may be useful.

Neurological deficit

Ask about any change in gait, perineal sensation, sexual function, micturition or defaecation.

Symptoms of systemic illness

Ask about any symptoms of systemic illness such as weight loss, fatigue, night sweats or fever.

Table 1: Distinguishing features of lumbar radicular and somatic referred pain[8]

	Radicular pain	Somatic referred pain
Distribution	Entire length of lower limb BUT below knee>above knee	Entire length of lower limb BUT proximal>distal
Pattern	Narrow band Travelling	Wide area with indistinct boundaries Static
Quality	Shooting, lancinating, like an electric shock	Dull, aching, like an expanding pressure
Depth	Deep as well as superficial	Deep only. No cutaneous quality

Base the examination on the history

The history will guide the extent of the examination. Examination aims to identify any serious pathology (very rare), and differentiate between patients with radicular pain (a few) and those with non-specific low back pain (the majority).

A minimal musculoskeletal examination for acute low back pain consists of:

- Observing posture, gait and general demeanour
- Checking for any structural abnormality or tenderness
- Assessing range of motion

A neurological examination is required if the patient has pain in the leg or if the history suggests any neurological symptoms such as paraesthesia, weakness or sphincter dysfunction.[9]

See page 17 for a five minute back examination with neurological assessment.

Symptoms and signs of lumbar radicular irritation:

- Leg pain greater than back pain
- Narrow band of pain radiating into foot or lower leg
- Numbness and paraethesias in dermatomal distribution
- Diminished leg reflexes
- Positive straight leg raising test (L4-S1 nerve roots)
- Positive femoral stretch test (L2-L4 nerve roots)
- Leg pain exacerbated by coughing, sneezing or Valsalva manoeuvre

A more general examination should be considered if the picture is atypical (see box).

Atypical causes of back pain[10]

Consider referred visceral pain presenting as low back pain, such as:

- Gastrointestinal disease (e.g. inflammatory bowel disease, pancreatitis, diverticulitis)
- Renal disease (e.g. renal stones, pyelonephritis)
- Abdominal aortic aneurysm
- Gynaecological disease (e.g. pelvic inflammatory disease)

Consider other disorders such as fibromyalgia and herpetic neuralgia.

Table 2: Red flags and what to do (Adapted from WeMeRec 2008)[12]

▪ Major trauma or minor trauma with osteoporosis **Consider plain x-ray of lumbar spine**
▪ Unrelenting pain, pain worse at night (supine) ▪ Age <20 years, or new back pain age >50 years ▪ History of cancer ▪ Systemic symptoms e.g., fever, weight loss ▪ IV drug use ▪ Immunosuppression or steroids **Consider urgent investigation (CBC, CRP, Alk P, Ca^{2+}, PSA, x-ray) and referral**
▪ Sphincter disturbance e.g. recent bladder dysfunction (retention, overflow incontinence) ▪ Gait disturbance: severe and/or progressive neurological deficit in lower extremities ▪ Saddle anaesthesia: diminished sensation over the buttocks, posterior-superior thighs and the perineal region in the "saddle" distribution **Possible cauda equina REFER IMMEDIATELY FOR EMERGENCY ASSESSMENT (see page 12)**

Investigation of acute low back pain

Investigation depends on which category of low back pain the patient falls into and is divided into possible serious and non–serious conditions (non-specific back pain and back pain with radicular nerve involvement).

Investigation of serious conditions

Serious conditions are detected with red flags and investigated and referred as appropriate (Table 2).

Investigation of non-serious conditions

95% of low back pain is not serious. Most acute low back pain is likely to be a functional problem of the musculoskeletal system and is termed non-specific low back pain (previously known as mechanical pain).[11] Approximately one in twenty people with acute low back pain will have radicular pain.

Most patients with back pain do not require radiological investigations[13]

Lumbar x-ray

X-ray of the lumbar spine is not required for non-specific back pain and lumbar radicular pain in patients aged 20 to 50 years.[5, 14] In this situation x-rays do not provide extra information and often confound the picture with false positive findings such as spondylolisthesis, which occurs as often in people with and without acute low back pain. It also exposes the patient to relatively high doses of radiation (approximately one hundred and fifty times the dose of a chest x-ray).

An x-ray may provide reassurance for a doubtful patient, although the demonstration of incidental asymptomatic abnormalities may cause anxiety.

Lumbar x-ray is of benefit in younger patients with suspected ankylosing spondylitis (anteroposterior, lateral and oblique views), rare spinal developmental disorders and in older patients with suspected osteoporotic collapse. X-rays should be considered in all patients who have had recent trauma irrespective of age.

If serious pathology is suspected an x-ray of the lumbar spine should be obtained but not relied upon as even an advanced tumour may not show on the films. A plain x-ray will only show pathology once 50% of bone destruction has occurred. If underlying disease is suspected, check bloods for CBC, CRP, Alk P, Ca^{2+}, PSA and arrange referral for bone scan or MRI.

MRI

MRI is not usually appropriate for patients with predominant back pain and is best reserved for the investigation of radicular leg pain, that is not settling with standard treatment, or as an alternative to isotope bone scan in cases of possible serious pathology. Similar to lumbar x-ray, false positives are common.

References:

1. Andersson G. Epidemiology of low back pain. Acta Orthop Scand Suppl 1998;281:28-31.
2. Jarvik J, Deyo R. Diagnostic evaluation of low back pain with emphasis on imaging. Ann Intern Med 2002;137:586-97.
3. European Commission Cost B13 Working Group. European guidelines for the management of acute non-specific low back pain in primary care. Available from: www.backpaineurope.org (accessed April 2009).
4. McGuirk B, King W, Govind J, et al. Safety, efficacy, and cost effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. Spine 2001;26(23):2615-22.
5. Koes B. Diagnosis and treatment of low back pain. BMJ 2006;332:1430-4.
6. Yelland M. Diagnostic imaging for back pain. Am Fam Prac 2004;33(6):415-9.
7. Wai E, Howse K, Pollock J, et al. The reliability of determining "leg dominant pain". Spine J 2008 [Epub ahead of print].
8. Govind J. Lumbar radicular pain. Aust Fam Phys 2004;33(6):409-12.
9. Australian Acute Musculoskeletal Pain Guidelines Group. Evidence-based management of acute musculoskeletal pain: a guide for clinicians. National Health and Medical Research Council 2004.Available from: www.nhmrc.gov.au (accessed April 2009).
10. Deyo R, Weinstein J. Low back pain. N Engl J Med 2001;344:363-70.
11. Hollingworth W, Todd C, King H, et al. Primary care referrals for lumbar spine radiography: diagnostic yield and clinical guidelines. Br J Gen Prac 2002;52:475-80.
12. Welsh Medicines Resource Centre. Management of acute low back pain. WeMeRec Bulletin 2008. Available from: www.wemerec.org (accessed April 2009).
13. Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007;147:478-91.
14. Kendrick D, Fielding K, Bentley E, et al. Radiology of the lumbar spine in primary care patients with low back pain: randomised controlled trial. BMJ 2001;322:400-5.
15. Lavy C, James A, Wilson-MacDonald J, Fairbank J. Cauda equina syndrome. BMJ 2009;338:881-4.

Cauda Equina Syndrome

The spinal cord ends at the lower border of the first lumbar vertebra. The cauda equina, or "horse's tail", represents the continuation of the lumbosacral nerve roots in the subarachnoid space into the sacrum. Cauda equina syndrome is the result of mechanical compression of the neural elements below the end of the spinal cord (L1–L2). This causes pain and progressive neurological deficit, involving sphincters, gait and perineal sensation.

The conus medullaris syndrome is a similar syndrome which in contrast only causes sphincter disturbance. This occurs with compression of spinal elements just above the cauda equina at T12–L1.

The most common cause of cauda equina syndrome is central herniation of a lumbar intervertebral disc. Other possible causes include tumours, trauma, infections, spinal stenosis and spondylolisthesis.

Presentation: progressive neurological deficit

Most cases are of sudden onset and progress rapidly within hours or days. However cauda equina syndrome can evolve slowly and patients do not always complain of pain. Common presenting symptoms and signs include:

- Low back pain; usually the patient is in significant distress with severe pain
- Bilateral leg symptoms; including pain (classically bilateral lumbar radicular pain), lower motor neurone weakness (ranging from difficulty walking to complete paralysis) and sensory changes
- Saddle anaesthesia; loss of perineal sensation
- Urinary dysfunction; retention, difficulty starting or stopping a stream of urine, overflow incontinence and decreased bladder and urethral sensation
- Bowel disturbances; may include incontinence or constipation, although a patient may have no complaints about bowel function but be found to have reduced anal tone on per rectum (PR) examination
- Sexual dysfunction

Cauda equina syndrome is an emergency

The diagnosis is usually possible from the history and examination. Always err on the side of caution rather than risk leaving your patient with permanent disability. Refer any patient with suspected cauda equina immediately for a specialist consultation (neurosurgical or oncology if known cancer).

Urgent surgical spinal decompression is indicated for most patients to prevent permanent neurological damage. If surgery cannot be performed, radiotherapy may relieve cord compression caused by malignant disease.

Prognosis is dependent on the underlying cause, the extent of the initial neurological deficit and the time taken before effective treatment is provided. Late diagnosis and treatment increases the risk of a permanent neurological deficit.[15]

Management of **non-specific back pain** and **lumbar radicular pain**

www.bpac.org.nz keyword: nsbackpain

HAVING EXCLUDED serious pathology, the aim of management is to reduce distress and encourage return to activity, by addressing the patient's fears, educating about back pain and providing adequate analgesia.[1, 2]

Address fears

Patients' beliefs and attitudes warrant as much attention early on as the anatomical and pathological aspects of their condition. Fear about pain can be more disabling than pain itself and is a major determinant of disability and possible chronicity.[3]

It is helpful to encourage the patient to reflect on their emotions and concerns. Open questions following the standard "FIFE" format are useful:

- Feelings: What are your concerns?
- Ideas: What do you understand is the cause of your back pain?
- Function: How is it affecting you?
- Expectations: What do you think is needed to help?

The following factors (yellow flags) can be associated with poor prognosis for back pain:

- Belief that back pain is harmful and potentially severely disabling; "I hurt", "I can't move", I can't work" and "I'm scared"
- Avoiding behaviours for fear of damaging the back
- Past history of chronic pain, somatisation and preoccupation with health
- Negative attitudes and outlook and a tendency towards lowered mood and withdrawal from social activity
- Expectation that passive treatments will help more than active participation

Provide reassurance

Offer a biological model of the pain, for e.g.;

"It's like an ankle sprain, you have probably strained muscles or ligaments, perhaps involving a disc, that won't show on x-ray. It will take a few days or weeks to heal, but you can gradually get back to normal activities as soon as you are able."[4]

Encourage people with acute low back pain to stay in work if possible[5]

Although back pain may be precipitated by factors at work only a small proportion of cases are actually caused by work. Most people with back pain continue to work most of the time. Continuing to work, provided it does not require extended periods of immobility, speeds recovery and reduces recurrences.

Encourage people with acute low back pain to stay in work if possible. Consider suggesting work adjustments rather than signing the patient off work. If sick leave is unavoidable, make it short-term and review progress regularly. Patients initially unfit for work should be advised to return as soon as possible and not to wait until they are pain free

The key messages that need to be conveyed to the patient as part of the reassurance process are:

- There is no sign of any serious disease as red flags were excluded on history and examination.
- Most acute low back pain does get better:
  - Non-specific back pain may take some time to settle, even up to a couple of months. It is not unusual to experience "flare-ups" but this doesn't mean there is anything wrong. Over time most people have a complete recovery.
  - With lumbar radicular pain expect a dramatic reduction in severity of pain with simple analgesics and keeping active. 90% of patients with radicular pain, associated with a lumbar disc, will start to improve within six weeks and be free of leg pain at twelve weeks.[6]
- There is no need for x-rays initially as the majority of causes for acute low back pain are due to functional disturbance of the non-bony structures that do not show on x-ray. If the pain is not improving with conservative treatment over four to six weeks, radiological investigations may then be appropriate.
- If movement causes pain this does not indicate ongoing damage. Light activity will not harm the spine. Increased muscle tension and spasm can increase the pain and this can be relieved with simple stretching and mobilising the lumbar spine with light activity.

Provide advice about activity

Provide clear explanations about why exercise and activity is both safe and recommended. Encourage the patient to stay active despite pain rather than waiting for the pain to settle completely.[7] They should continue normal daily activities, including work if possible, and avoid bed rest as this delays recovery.

Practical tips:

- Teach some simple stretching techniques
- Advise walking as normally as possible and suggest

gradually increasing activity such as walking or swimming on a daily basis aiming for 30 minutes a day

- Refer early to physiotherapy[8]
- Reinforce recommendations with a green prescription

Prescribe adequate analgesia

Adequate analgesia from day one helps mobilisation. It does not cure the problem.

It is often appropriate to start with:

- Paracetamol 1 g four times daily
- Plus a NSAID, such as ibuprofen 400 mg four times daily (+/- gastro-protection e.g. omeprazole 20 mg)

NSAIDs have a small short-term effect on acute low back pain without radicular pain.[9]

If the above treatments do not provide adequate pain relief add:

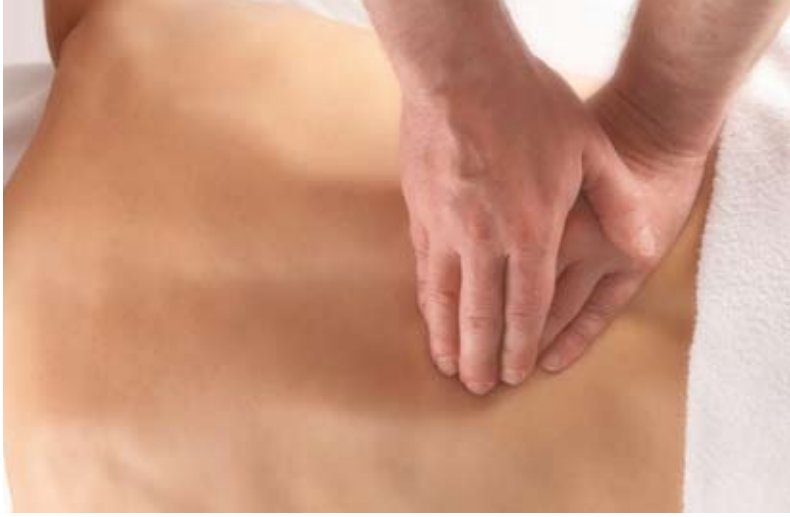
- A weak opioid such as codeine (30–60 mg 4 hourly) or tramadol (50 mg 6 hourly) plus laxatives

There is conflicting evidence that muscle relaxants (e.g. diazepam, orphenadrine) are effective in acute low back pain. Adverse effects of muscle relaxants include drowsiness, dizziness and dependence. These effects usually outweigh any benefit and therefore muscle relaxants are no longer routinely recommended.[10]

Tricyclic antidepressants have a place in the treatment of chronic pain but are not recommended for the treatment of acute low back pain.[11]

Alternative therapies

Local heat therapy is more effective than paracetamol or NSAIDs in the first 48 hours. Manipulation may provide some short-term improvement in pain, activity levels and patient satisfaction.[12] Massage may provide short term relief.



The role of manipulation

Spinal manipulation is safe in the majority of cases of back pain[13] including neurogenic pain from disc herniation.[14] However there are rare serious complications associated with nearby vessels and nerves.[15, 16] The risks are higher with cervical spine manipulation and when a serious underlying disease or structural abnormality has not been diagnosed.

Spinal manipulation should be avoided or used with caution in the following conditions; acute fracture, dislocation, ligamentous rupture, instability, tumour, infection, acute myelopathy, cauda equina syndrome, spondylolisthesis, recent surgery, acute soft tissue injury, osteoporosis, ankylosing spondylitis, rheumatoid arthritis, anticoagulant therapy and bleeding dyscrasias.

An improvement should be noticed, even if only transient, after one treatment. If the patient is no better after three treatments, they should stop.

Review regularly

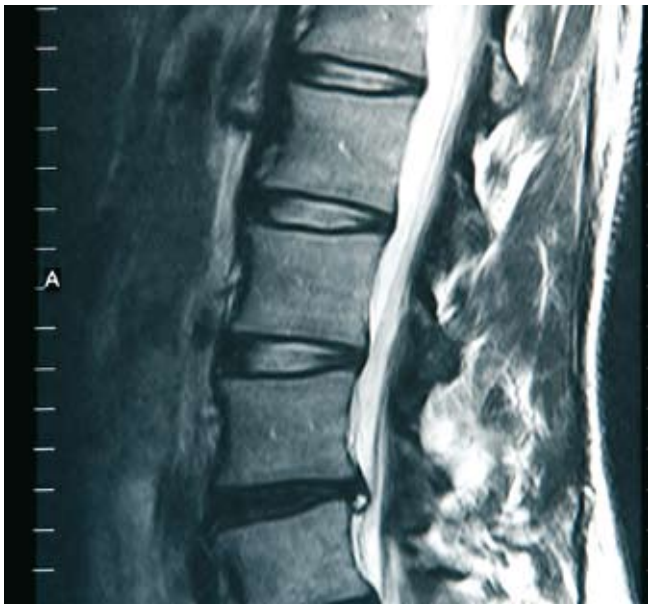
Each review is an opportunity to continue to develop a relationship with the patient, reinforce their active participation, monitor progress, and check for any emerging red flags. At each visit:

- Check for red flags and review any change in neurology; any deterioration should trigger urgent investigation or referral
- Reassess the patients ideas, the impact of the back pain, their concerns and expectations
- Review exercise and medication
- Reinforce previous explanations and advice

At four to six weeks

If the pain is not resolving or if the patient has not returned to normal activities, carefully reassess for red flags to exclude serious pathology and investigate as indicated. Re-assess yellow flags and address beliefs or behaviours that may be delaying recovery. A short course of manipulation may help (if not already tried).[17]

It is appropriate to refer for assessment (ACC GPSI programme or specialist) to help prevent long term problems and chronic back pain.[3] At this stage MRI is indicated, if neurogenic pain is not beginning to settle with simple analgesics and encouragement to resume daily activities, and if surgery is being contemplated.



References:

1. Cohen SP, Argoff CE, Carragee EJ. Clinical review: management of low back pain. BMJ 2009;338:100-6.
2. Thompson B. Low back pain management in primary care. N Z Fam Prac 2004;31(2):72-7.
3. NZGG. New Zealand acute low back pain guide, incorporating the guide to assessing psychological yellow flags in acute low back pain. 2004. Available from:www.nzgg.org.nz (Accessed April 2009).
4. Wilk V. Acute low back pain: assessment and management. Aust Fam Phys 2004;33(6):403-7.
5. Welsh Medicines Resource Centre. Management of acute low back pain. WeMeRec Bulletin 2008. Available from:www.wemerec.org (Accessed April 2009).
6. Saal JA, Saal JS. Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy. An outcome study. Spine 1989;14(4):431-7.
7. Hagen KB, Hilde G, Jamtveldt G, Winnem M. Bed rest for acute low back pain and sciatica. Cochrane Database Syst Rev 2004(4):CD001254
8. Pinnington MA, Miller J, Stanley I. An evaluation of prompt access to physiotherapy in the management of low back pain in primary care. Fam Prac 2004;21(4):372-80.
9. Roelofs P, Deyo RA, Koes BW, et al. Non-steroidal anti-inflamatory drugs for low back pain. Cochrane Database Syst Rev 2008(1):CD000396.
10. Australian Acute Musculoskeletal Pain Guidelines Group. Evidence-based management of acute musculoskeletal pain: a guide for clinicians. National Health and Medical Research Council 2004. Available from: www.nhmrc.gov.au (Accessed April 2009).
11. Urquhart D, Hoving JL, Assendelft WJ, et al. Antidepressants for non-specific low back pain. Cochrane Database Syst Rev 2008(1):CD001703.
12. Chou R, Huffman LH. Nonpharmacologic therapies for acute and chronic back pain: a review of the evidence for an American pain Society/American College of Physicians clinical practice guideline. Ann Intern Med 2007;147:492-504.
13. Barrett AJ, Breen AC. Adverse effects of spinal manipulation. J Roy Soc Med 2000;21(24):2860-71.
14. Oliphant D. Safety of Spinal Manipulation in the Treatment of Lumbar Disk Herniations: A Systematic Review and Risk Assessment. J Manip Physiol Therap 2004;27(3):197-210.
15. Oppenheim JS, Spitzer DE, Segal DH. Nonvascular complications following spinal manipulation. Spine J 2005;5(6):660-6.
16. Stevenson C, Ernst E. Risks associated with spinal manipulation. Am H Med 2002;112:566-70.
17. Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007;147:478-91.

A five minute back examination with neurological assessment

Adapted from Cameron 2009[1].
Also available online at **www.bpac.org.nz**

Ask the patient to remove enough clothing to allow full inspection of the back and legs.

With the patient standing:

- Inspect the spine for any developmental or traumatic deformities. Assess the lumbar lordosis; loss of curvature may indicate ankylosing spondylitis. Look for any muscle wasting (buttock, thigh, calf). Check for any discrepancy in leg length by comparing the levels of the iliac crests.
- Movement: Ask the patient to extend the spine, flex forward and then flex laterally by sliding their palm down their outer thigh. Most patients with non-specific back pain will be slightly stiff in extension, have pain on flexion, and asymmetric limitation and pain on lateral flexion.

With the patient lying supine

1. Rule out other joint involvement: check the hip joints for range of movement and pain. Perform stress test on sacro-iliac joints (e.g. FABER test), especially in young patients.
2. Test the nerve roots: Straight leg raise test. This stretches nerve roots L4, L5 and S1. Pick the leg up by the ankle. While keeping the knee fully extended, lift the leg up towards ninety degrees or beyond (Figure 2). If the patient has significant nerve root entrapment shooting leg pain will be reproduced before you get much beyond thirty degrees of elevation. Back pain produced by straight leg raising is common and does not always indicate nerve root involvement.

The FABER test

The Flexion, Abduction, and External Rotation (FABER) test is used to detect hip or sacro-iliac joint problems. The patient lies in a supine position, and the foot is placed on the opposite knee; in this position groin pain indicates a hip problem rather than a spinal problem. The doctor then presses on the flexed knee and on the opposite anterior superior iliac crest; pain in the sacroiliac area indicates a problem with sacroiliac joints (Figure 1).

Figure 1: FABER test

from Bernstein R and Cozen H 2007 [2]

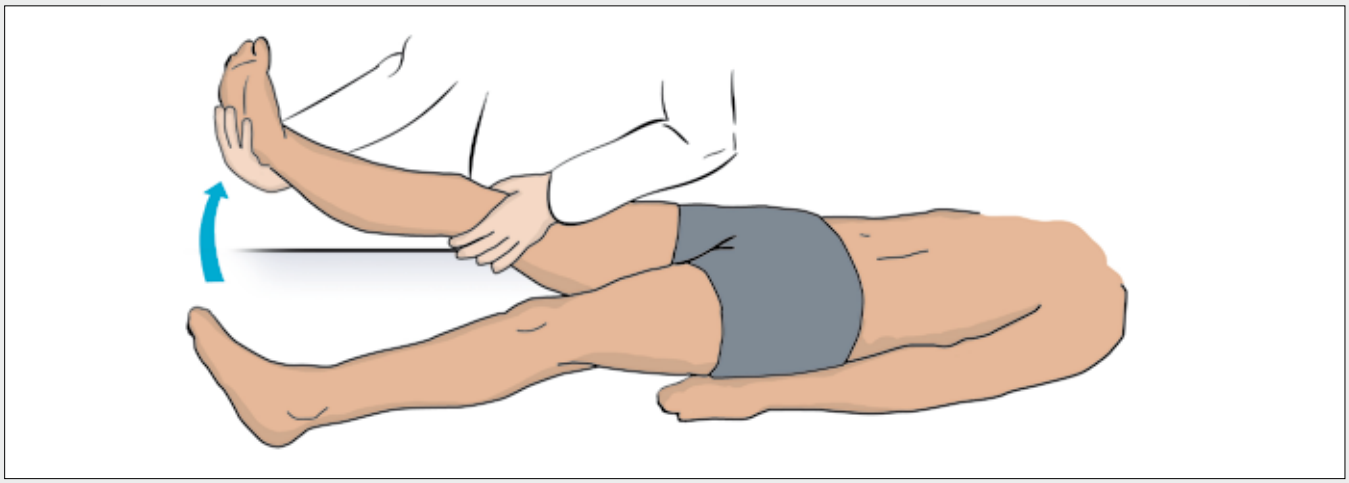
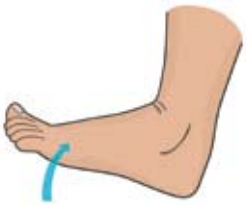
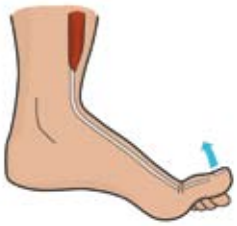
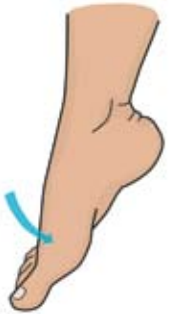


Figure 2: Straight leg raise test

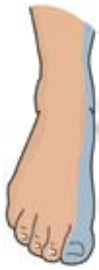
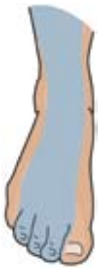
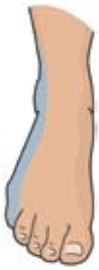
3. Assess muscle power

Muscle group	Nerve root
Resisted flexion of hip	L2 and L3
Resisted knee flexion	S2
 Resisted dorsiflexion of the ankle	L4
 Resisted extension of the big toe	L5
 Resisted eversion of the foot or resisted plantar flexion of the ankle	S1

4. Check the reflexes

Reflex	Nerve root
Knee jerk	L3 and L4
Ankle jerk	L5 and S1
Plantar reflex	Up-going toes in adults may indicate upper motor neurone abnormalities such as myelopathy or demyelinating disease, rather than common low back problem.

5. Check for skin sensory loss

Disk	L3–L4	L4–L5	L5–S1
Nerve root	L4	L5	S1
Sensory loss signature zone	Medial malleolus 	Dorsal third metatarsophalangeal joint 	Lateral heel 

With the patient lying prone

- Femoral nerve stretch test (nerve roots L2, L3 and L4): With the patient lying prone, flex the knee towards ninety degrees (Figure 3). Burning discomfort in the groin or anterior thigh will occur if there is femoral nerve involvement.
- Palpate the spine for tenderness and for muscle spasm

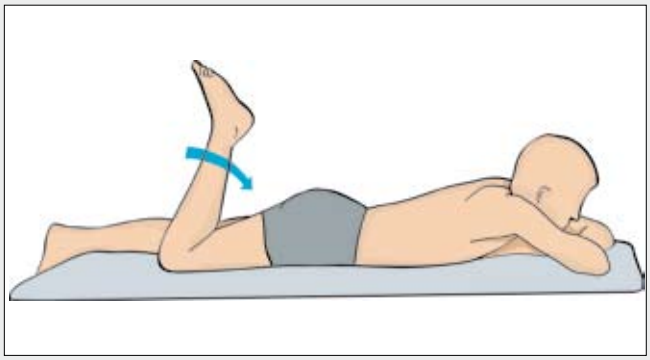


Figure 3: Femoral nerve stretch test

With the patient on their side

In patients who describe loss of sphincter control, or with serious or progressive neurological findings, test for impaired sensation in the saddle area (checking pin-prick sensation around the anus) and assess anal sphincter tone by digital examination while the patient tries to "squeeze" your examining finger.

References:

1. Cameron G. The assessment of lower back pain in primary care or family practice. Available from: www.jointenterprise.co.uk/backpain-1.htm (accessed April 2009).
2. Bernstein R, Cozen H. Evaluation of back pain in children and adolescents. AAFP 2007;76(11):1669-76.

Antibiotic choices for common infections

Key reviewers:
Associate Professor Mark Thomas, Infectious Disease Specialist, School of Medical Sciences, University of Auckland

Dr Susan Taylor, Clinical Microbiologist, Diagnostic Medlab and Middlemore Hospital, Auckland

A safe and effective strategy for antibiotic use involves only prescribing an antibiotic when it is needed and selecting an effective agent at the correct dose with the narrowest spectrum, fewest adverse effects and lowest cost.

Principles of antibiotic prescribing:

1. Only prescribe antibiotics for bacterial infections if:
   - Symptoms are significant or severe
   - There is a high risk of complications
   - The infection is not resolving
2. Use first-line antibiotics first
3. Reserve broad spectrum antibiotics for indicated conditions only

www.bpac.org.nz keyword: antibiotics

The following table is intended as a guide for selecting an appropriate antibiotic for infections commonly seen in general practice. Local resistance patterns and individual patient circumstances may alter the choice of antibiotic.

Respiratory

Acute bronchitis	
Management	Most acute bronchitis is of viral origin and therefore antibiotics are not indicated. Purulent sputum alone does not indicate the need for antibiotics. Antibiotics may be appropriate for those with co-morbidity or of advanced age.
Common pathogens	Respiratory viruses Less commonly: *Bordetella pertussis, Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*
Antibiotic therapy	Not usually indicated

Acute exacerbation of chronic bronchitis	
Management	Most exacerbations are likely to be viral and antibiotics are of limited benefit. Patients with severe exacerbations and those with more severe airflow obstruction at baseline are most likely to benefit from antibiotics.
Common pathogens	Respiratory viruses, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*
Antibiotic therapy	
First choice	Amoxycillin 500 mg 3 times daily for 5 days
Alternatives	Doxycycline 100 mg 2 times daily for 5 days

Pneumonia – adult	
Management	Consider chest x-ray to confirm diagnosis. The decision to treat with oral antibiotics as an outpatient depends on the age of the patient, co-morbidities and clinical signs indicating severity (HR > 100 bpm, RR > 24 bpm, temp. ≥ 38 °C, signs of focal consolidation on examination).
Common pathogens	Respiratory viruses, *S. pneumoniae, H. influenzae, M. pneumoniae*, *C. pneumoniae*, *Legionella pneumophila*, *Staphylococcus aureus*
Antibiotic therapy	
First choice	Amoxycillin 1 g 3 times daily **Plus either (to cover atypical infection):** Roxithromycin 300 mg daily or Doxycycline 200 mg stat then 100 mg daily Duration of treatment is approximately 7 days.
Alternatives	Monotherapy with erythromycin, doxycycline or co-trimoxazole are alternatives for those with a history of penicillin allergy.

N.B. Roxithromycin offers an alternative to erythromycin as they are both macrolide antibiotics.

Pneumonia – child	
Management	In a young child, suspect pneumonia if tachycardia, grunting, indrawing and high fever in absence of wheeze (auscultatory findings uncommon). The decision whether a patient receives inpatient or outpatient therapy depends on clinical severity. Patients who have systemic toxicity or any indication of respiratory failure should be treated in hospital.
Common pathogens	Respiratory viruses, *S. pneumoniae*, *H. influenzae*, *M. pneumoniae*
Antibiotic therapy	
First choice	Amoxycillin 25 mg/kg 3 times daily for 7 days
Alternatives	Erythromycin If no response in 48 hours, review diagnosis and consider referral to hospital.

Pertussis	
Management	Community outbreaks of pertussis occur approximately every four years (see page 42). Notifiable disease. Antibiotics do not effect the course of the disease if they are given more than seven days after the illness has started. However, they may be justified during the first four weeks of the illness to limit transmission to susceptible contacts.
Common pathogens	*B. pertussis*
Antibiotic therapy	
First choice	Erythromycin 10 mg/kg (up to 500 mg) 4 times daily for 14 days

Ear, nose and throat

Otitis externa – acute or "swimmers ear"	
Management	Gentle debridement of the ear canal may be necessary to enhance the effectiveness of topical treatment. Suction cleaning is also a safe and effective method of debridement. Most topical antibacterials are contraindicated in the presence of a perforated drum or grommets.
Common pathogens	*Pseudomonas aeruginosa*, *S. aureus*, polymicrobial infections
Antibiotic therapy	
First choice	Clioquinol + flumethasone (Locorten Vioform) 2 to 3 drops 2 times daily **or** Dexamethasone + framycetin + gramicidin (Sofradex) 2 to 3 drops, 3 to 4 times daily.
Alternatives	Acetic acid 2% (Vosol) or ciprofloxacin + hydrocortisone (Ciproxin HC)

Otitis media – acute	
Management	Immediate antibiotic therapy is usually unnecessary. Consider antibiotics for those in high risk groups such as children with systemic symptoms, children under 6 months or children under 2 years with severe or bilateral disease. Otherwise treat symptomatically (e.g. paracetamol) and arrange follow up or give a prescription to be dispensed if no improvement in next 24 hours.
Common pathogens	Respiratory viruses, *S. pneumoniae* , *H. influenzae*, *M. catarrhalis*
Antibiotic therapy	
First choice	Amoxycillin 40 mg/kg/day in 2 to 3 divided doses (max. 3 g daily) for 5 days (7 to 10 days if < 2 years, underlying medical condition, perforated drum, chronic or recurrent infections)
Alternatives	Erythromycin, cefaclor or co-trimoxazole

Pharyngitis	
Management	Most pharyngitis is of viral origin. Give antibiotics only if: ▪ Features of group A strep infection: temperature >38°C, no cough, tender cervical nodes, tonsillar swelling or exudates, especially if aged 3–14 years. If uncertain swab throat. ▪ Patient aged 3–45 years and at high risk of rheumatic fever: Māori and Pacific peoples, lower socioeconomic areas of North Island, past history of acute rheumatic fever. ▪ Existing rheumatic heart disease (treat at any age).
Common pathogens	Respiratory viruses, *Streptococcus pyogenes*
Antibiotic therapy	
First choice	Phenoxymethylpenicillin 500 mg (child 10 mg/kg) twice daily for 10 days **or** stat IM benzathine 0.6 MU if <27 kg or 1.2 MU if > 27 kg
Alternatives	Erythromycin ethylsuccinate

Acute sinusitis	
Management	Most patients with sinusitis will not have a bacterial infection. The following cluster of symptoms may suggest bacterial sinusitis: ▪ Purulent nasal discharge persisting more than 7 days ▪ Facial pain or maxillary tooth ache ▪ Unilateral sinus tenderness ▪ Fever Although studies suggest there may be limited benefit, an antibiotic can be considered if these symptoms are present.
Common pathogens	Respiratory viruses, *S. pneumoniae*, *H. influenzae*, *M. catarrhalis*, anaerobic bacteria (reflecting extension of dental abscess)

Acute sinusitis (continued)	
Antibiotic therapy	
First choice	Amoxycillin 500 mg (child 15 mg/kg) three times daily for 7 days
Alternatives	Doxycycline, cefaclor or co-trimoxazole If anaerobes suspected, use amoxycillin/clavulanic acid

Eyes

Conjunctivitis	
Management	Allergic, viral or bacterial. Bacterial more likely if eyelids very sticky or unilateral. Viral more likely if starts bilaterally. Most bacterial conjunctivitis (except *Chlamydia trachomatis* or *Neisseria* gonorrhoeae) is self-limiting and two thirds of cases improve in 2 to 5 days. Assess for keratitis (using fluorescein stain) in contact lens wearers before treating as conjunctivitis.
Common pathogens	Viruses, *S. pneumoniae*, *H. influenzae*, *S. aureus* In newborns, consider *C. trachomatis* or *N. gonorrhoeae*, in which case topical therapy is inadequate and referral to a paediatrician is recommended.
Antibiotic therapy	
First choice	Topical chloramphenicol until 48 hours after infection has cleared
Alternatives	Topical fusidic acid or topical framycetin

Skin

Bites and clenched fist* infections	
Management	Clean and debride wound thoroughly and treat with antibiotic. Assess patient's need for tetanus immunisation. Consider referral if bone or joint involvement.
Common pathogens	Polymicrobial infection , *Pasteurella multocida*, *Capnocytophaga conimorsus* (cat and dog bites), *Eikenella corrodens* (fist injury), *S. aureus*, streptococci and anaerobes
Antibiotic therapy	
First choice	Amoxycillin/clavulanic acid 500/125 mg three times daily for 5 to 10 days
Alternatives	Metronidazole plus either doxycycline or co-trimoxazole

* Injury to fist from contact with teeth

Boils	
Management	Most lesions may be treated with incision and drainage alone. Antibiotics may be considered if fever, surrounding cellulitis or co-morbidity e.g. diabetes or if the lesion is in a site associated with complications e.g. face. If recurrent boils (e.g. more than 10 boils over more than 3 months) consider staphylococcal decolonisation with a one week course of intranasal mupirocin or fusidic acid.
Common pathogens	*S. aureus*
Antibiotic therapy	
First choice	Flucloxacillin 500mg 4 times daily for 7 to 10 days
Alternatives	Erythromycin, cefaclor, co-trimoxazole

Cellulitis	
Management	Antibiotic treatment is indicated. Keep affected area elevated and assess response to treatment. May require referral if severe. For periorbital cellulitis, in all but very mild cases consider referral for IV antibiotics.
Common pathogens	*S. pyogenes*, *S. aureus*, Group C or Group G streptococci
Antibiotic therapy	
First choice	Flucloxacillin 500 mg 4 times daily for 7 to 10 days (the addition of penicillin is not required)
Alternatives	Erythromycin, cefaclor, co-trimoxazole

Diabetic foot infections	
Management	Length of treatment depends on clinical response or whether there is possible involvement of the bones of the feet. Referral may be required.
Common pathogens	Polymicrobial infection i.e. a mixture of anaerobes, Gram-positive and Gram-negative aerobes
Antibiotic therapy	
First choice	Amoxycillin/clavulanic acid 500/125 mg three times daily, usually 5 to 10 days
Alternatives	Cefaclor or co-trimoxazole plus metronidazole

Impetigo	
Management	Remove crusted area and apply topical antibiotic treatment. Keep affected areas covered and stay away from school for 24 hours after treatment initiated (see BPJ 19).
Common pathogens	*S. aureus*, *S. pyogenes*
Antibiotic therapy	
First choice	Fusidic acid cream for 7 days
Alternatives	Flucloxacillin (oral) for 7 days for extensive lesions or topical treatment failure

Mastitis	
Management	Treat with antibiotic and continue to breast feed from both breasts. This is an important component of treatment and poses no risk to the infant (see BPJ 18).
Common pathogens	*S. aureus*, anaerobes in non-lactating women or in men
Antibiotic therapy	
First choice	Flucloxacillin 500 mg 4 times daily for 7 days
Alternatives	Cefaclor, erythromycin

Gastrointestinal

Campylobacter	
Management	Most people will recover with symptomatic treatment only. Antibiotics have little impact on the duration and severity of symptoms but eradicate stool carriage. Antibiotic treatment is indicated if symptoms are severe or prolonged. Treatment may also be reasonable in food handlers, childcare workers and those caring for immunocompromised patients. For pregnant women nearing term, Campylobacter gastroenteritis should be treated with erythromycin to prevent exposure of the neonate to Campylobacter during vaginal delivery. Notifiable disease.
Common pathogens	*Campylobacter jejuni*, *Campylobacter coli*
Antibiotic therapy	
First choice	Erythromycin 250 mg – 500 mg (child 10 mg/kg) three times daily for 5 days
Alternatives	Norfloxacin 400 mg twice daily for 5 days is an alternative although resistance is likely if the infection was acquired overseas

Clostridium difficile toxin disease	
Management	Treat with metronidazole and discontinue other antibiotics when possible. Antidiarrhoeals (e.g. loperamide) should be avoided as the toxin may be retained and worsen colitis. Relapse occurs in approximately 20% of people.
Common pathogens	*Clostridium difficile*
Antibiotic therapy	
First choice	Metronidazole 400 mg orally three times daily for 7 to 10 days

Giardiasis	
Management	Avoid lactose-containing foods for one month after therapy. Notifiable disease
Common pathogens	*Giardia lamblia*

Antibiotic therapy	
First choice	Ornidazole 1.5 g orally once daily for 1 or 2 days **or** Metronidazole 2 g (child 30 mg/kg/day) orally once daily for 3 days
Alternatives	For treatment failure: ▪ Exclude re-infection from asymptomatic family contacts e.g. children ▪ Use metronidazole 400 mg (child 10 mg/kg) three times daily for 7 days

Salmonellosis	
Management	Routine treatment with antibiotics is usually unnecessary and may prolong excretion. Treat in severe disease or immunocompromised patients. Notifiable disease.
Common pathogens	*Salmonella enteritidis*, *Salmonella typhimurium*
Antibiotic therapy	
First choice	Norfloxacin 400 mg orally twice daily for 3 to 5 days
Alternatives	Co-trimoxazole (400 + 80 mg tablets) 2 tablets twice daily for 3 to 5 days

Urinary

Cystitis	
Management	Non-pregnant women with uncomplicated cystitis do not require investigation. Males, children and pregnant women require urine culture (see Laboratory Investigation of UTI, June 2006, for more information). Antibiotic therapy is indicated for all people who are symptomatic. Asymptomatic bacteriuria requires antibiotic treatment in pregnant women but not in elderly women or patients with long-term indwelling urinary catheters. Treat for longer in pregnant women (7 days) and in men (10 to 14 days). Pregnant women should have repeat urine culture 1 to 2 weeks after completing therapy to ensure cure.
Common pathogens	*E. coli*, *Staphylococcus saprophyticus*, *Proteus sp.*, *Klebsiella sp.*, *Enterococcus sp.*
Antibiotic therapy	
First choice	Trimethoprim 300 mg once daily for 3 days (usually avoided during the 1st trimester).
Alternatives	Nitrofurantoin 50 mg four times daily for 5 days (usually avoided at term), cefaclor 500 mg three times daily for 3 days or amoxycillin/clavulanic acid 500+125 mg twice daily for 3 days. Norfloxacin is an alternative but should be reserved for isolates resistant to initial empiric choices.

Acute pyelonephritis	
Management	Only treat as an outpatient if mild symptoms e.g. low fever and no nausea or vomiting. If systemically unwell or vomiting refer for IV treatment.
Common pathogens	*E. coli*, *Proteus sp.*, *Klebsiella sp.*, *Enterococcus sp.*
Antibiotic therapy	
First choice	Trimethoprim 300 mg once daily for 10 to 14 days
Alternatives	Co-trimoxazole 400+80 mg 2 tablets twice daily for 10 to 14 days or amoxycillin/clavulanic acid 500+125 mg three times daily for 10 to 14 days or cefaclor 500 mg three times daily for 10 to 14 days.

CNS

Bacterial meningitis	
Management	In most cases, give antibiotic before transport to hospital in suspected cases of meningococcal disease. If practical, collect blood cultures before antibiotic administration. Notifiable disease.
Common pathogens	*Neisseria meningitides*, *S. pneumoniae* Less common: *Listeria monocytogenes*, *H. influenzae*
Antibiotic therapy	
First choice	Benzylpenicillin 1.2 g (child – 50 mg/kg) IV or IM
Alternatives	Amoxycillin 1 to 2 g (child – 50 to 100 mg/kg) IV or IM Ceftriaxone 50 mg/kg up to 2 g IV or IM

References:

1. Everts R. Antibiotic guidelines for primary care, Nelson and Marlborough Districts 2007-2008.
2. Ellis-Pegler R, Thomas M. Approaches to the management of common infections in general practice. Diagnostic Medlab 2003.
3. Lang S, Morris A, Taylor S, Arroll B. Management of common infections in general practice: Part 1. NZ Fam Phys 2004;31(3):176-8.
4. Lang S, Morris A, Taylor S, Arroll B. Management of common infections in general practice: Part 2. NZ Fam Phys 2004;31(4):258-60.
5. Australian Medicines Handbook. Adelaide Australian Medicines Handbook Pty Ltd, 2006.
6. British National Formulary 56 ed. London: British Medical Association and the Royal Pharmaceutical Society of Great Britain, 2008.
7. Lang S, editor. Guide to Pathogens and Antibiotic Treatment. 7th ed, 2004. Available from: http://dml.co.nz/ (Accessed April 2009).

Fluoroquinolones increase risk of tendon disorders

THE USE OF ORAL FLUOROQUINOLONES is associated with increased risk of tendinitis and tendon ruptures. This association can sometimes be missed in clinical practice.

Research has shown that these tendon disorders usually occur during the first month of treatment,[1] but may occur as early as two hours after the first dose and as late as six months after treatment has stopped.[2] A review of the literature showed that the median duration of fluoroquinolone treatment before the onset of tendon injury was eight days.[2]

The fully subsidised fluoroquinolones available in New Zealand are ciprofloxacin and norfloxacin. Moxifloxacin and gatifloxacin are also available but unsubsidised.

Mechanism of damage

The mechanism of this unusual form of toxicity is not fully understood but the sudden onset of some tendinopathies, occasionally those that occur after a single dose of a fluoroquinolone, suggests a direct toxic effect on collagen fibres.[1] Some recent research has reported fluoroquinolones causing oxidative stress and mitochondrial damage to tendon cells.[3]

Elderly people and those on steroids are at higher risk

A large general practice based case-control study published in 2002 indicated that the adverse effect of Achilles tendon disorders (both tendinitis and rupture) associated with fluoroquinolone use was definite but also relatively rare.[1]

The adjusted relative risk of Achilles tendon disorders with current fluoroquinolone use was 1.9. The relative risk with current use was 3.2 among patients aged 60 and over and 0.9 among patients aged under 60 years. Concurrent use of corticosteroids and fluoroquinolones increased the risk to 6.2.The conclusion was that patients aged over 60 years of age, and those taking corticosteroids at the same time were at substantially increased risk.

In the USA, reports to the FDA of fluoroquinolone-associated tendon disorders have been accumulating since 1994. Common injuries reported are rupture of the shoulder tendons, Achilles tendon, hand tendons, as well as other tendons. A black box warning was added to all packs of fluoroquinolones in July 2008.[4]

In addition to the risk factors of increased age and concomitant corticosteroid use, chronic kidney disease (including those on haemodialysis)[2] and previous heart, kidney or lung transplant is also known to contribute to an individual being at increased risk.

Advice to prescribers

There are limited indications for using a fluroquinolone in a general practice setting. They should only be used for the treatment or prevention of an infection that is proven, or strongly suspected, to be caused by bacteria that would justify the use of a fluoroquinolone.

Prescribers should be aware of the increased risk of fluoroquinolone-associated tendinopathy especially in elderly people, those taking corticosteroids or those with chronic renal disease or post-organ transplantation. Care should also be exercised with patients with a previous history of tendon disorder.

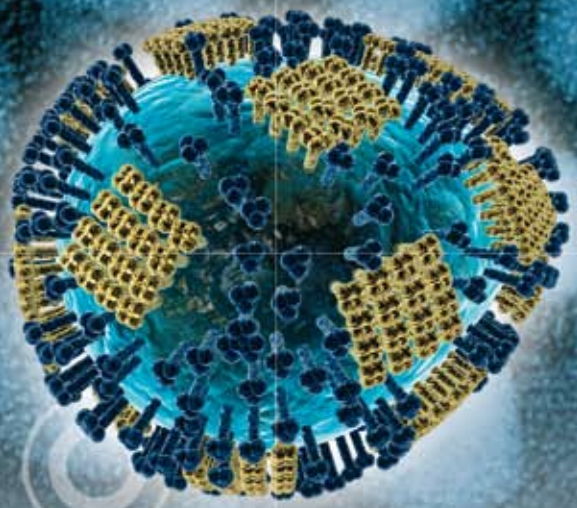
Prescribers should advise patients about the possibility of tendon pain, inflammation or rupture. If such pain occurs they should stop taking the fluoroquinolone and avoid exercise and use of the affected area, and promptly contact their doctor about changing to a non-fluoroquinolone drug.

It is useful to remember that tendon damage can occur during or after completion of a course of a fluoroquinolone.

References:

1. Van der Linden P, Sturkenboom M, Herings R, et al. Fluoroquinolones and risk of Achilles tendon disorders: case-control study. BMJ 2002;324:1306-7.
2. Khaliq Y, Zhanel G. Fluoroquinolone-associated tendinopathy: a critical review of the literature. Clin Infect Dis 2003;36(11):1404-10.
3. Lowes D, Wallace C, Murphy M, et al. The mitochondria targeted antioxidant MitoQ protects against fluoroquinolone-induced oxidative stress and mitochondrial damage in human Achilles tendon cells. Free Radical Res 2009;43(4):323-8
4. US Food and Drug Adminstration. Information for Healthcare Professionals. Fluoroquinolone antimicrobial drugs 2008. Available from: www.fda.gov/cder/drug/InfoSheets/HCP/fluoroquinolonesHCP.htm (Accessed May 2009).

Diagnosing and managing influenza



Key reviewers:
Associate Professor Mark Thomas, Infectious Disease Specialist, School of Medical Sciences, University of Auckland

Dr Susan Taylor, Clinical Microbiologist, Diagnostic Medlab and Middlemore Hospital, Auckland

Influenza is a highly infectious acute respiratory disease. In healthy people influenza is an acute, and usually self-limiting and uncomplicated disease, which can be managed symptomatically. However for those at risk of complications, it can be a significant cause of morbidity and mortality.

Immunisation is the primary way to prevent influenza and its complications.

Treatment with antivirals should be considered for patients with symptoms of influenza who are at risk of serious disease, e.g. elderly people and those with chronic illness.

Clinical diagnosis can be difficult because other respiratory illnesses can cause symptoms similar to influenza[1, 2]

Influenza is characterised by the sudden onset of symptoms including: fever (may be absent in elderly people), malaise, myalgia, headache, chills and cough. A wider range of symptoms may be seen in infants and children including lethargy, poor feeding and vomiting.

A diagnosis of influenza is more likely when influenza is circulating

During periods of increased influenza prevalence, the acute onset of fever and cough makes a diagnosis of influenza more likely. When prevalence is low, the presence of influenza-like symptoms is less accurate for diagnosing influenza.[3]

When a patient presents with symptoms and signs of influenza, four questions are useful to distinguish between influenza and influenza-like illness:[2]

1. Are influenza viruses known to be circulating in the area?
2. Did the patient experience a sudden onset of symptoms?
3. Is the patient's temperature significantly raised (> 38 °C)?
4. Does the patient have both systemic and respiratory symptoms, particularly cough?

If the answer is "yes" to all of these questions, influenza is the likely diagnosis.

Differential diagnoses include:[4]

- Other respiratory viral infections, e.g. respiratory syncytial virus, coronavirus, rhinovirus
- Meningitis
- Pneumonia
- Although rare consider malaria in people who have recently travelled to an area where malaria is endemic

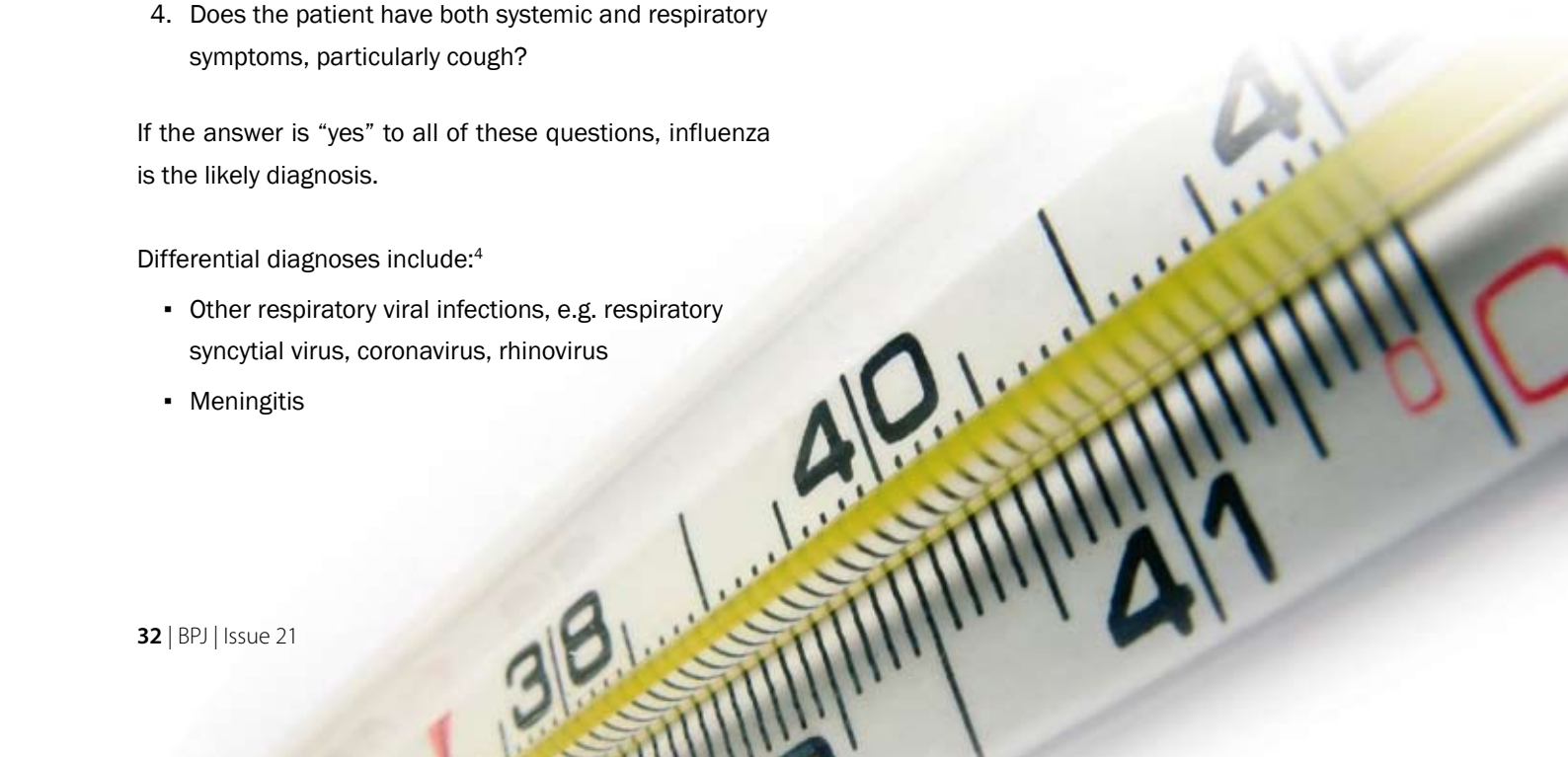
Laboratory diagnosis is rarely needed

Although a definitive diagnosis of influenza requires laboratory confirmation, it is not routinely needed in general practice as it unlikely to alter management. Laboratory tests are mainly used to survey influenza viruses to indicate when influenza is circulating, determine the current strains and monitor antiviral resistance.[3] In New Zealand, a group of sentinel general practices record the number of consultations for influenza-like illness, and collect respiratory samples for virus culture from patients with influenza-like illness.[1]

Immunisation is the primary intervention to prevent influenza and its complications

Vaccination is 70% to 90% effective in healthy adults when the vaccine strains match the current circulating strains well. The influenza vaccine is funded for all people aged over 65 years and those aged six months to 65 years with chronic medical conditions.

See BPJ 20 for more information about influenza vaccines.



Treatment of influenza

Healthy people with uncomplicated influenza do not usually require treatment with antivirals

Healthy people with uncomplicated influenza should be advised to rest, drink plenty of fluids and use analgesics such as paracetamol or ibuprofen for fever, headache and myalgia.[4]

Antiviral drugs (zanamivir or oseltamivir) may be appropriate for people who are at risk of complications

Elderly people and people with chronic co-morbidities who are frail, are at increased risk of influenza-related complications. It may be appropriate to treat these people with antivirals such as zanamivir and oseltamivir. Treatment is more effective the sooner it is given and must be initiated within 48 hours of the onset of symptoms.[6] Laboratory testing is unlikely to be useful in the decision to use antivirals, as results may take longer than 48 hours to be reported.

Antivirals can shorten the duration of influenza symptoms by one to three days if initiated within 48 hours of the onset of symptoms.[6] There is also some evidence that they can reduce the severity and incidence of complications of influenza, as well as shorten the length of hospital stay, and reduce mortality in patients with severe influenza.[6]

Recommended treatment doses

Oseltamivir (Tamiflu) is available as a tablet and a suspension. It is not subsidised for seasonal influenza and one course costs approximately $70. Note that as part of containment measures patients with suspected swine-origin influenza A are currently offered funded antiviral therapy.

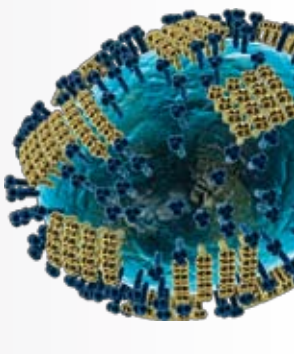
The recommended dose of oseltamivir for the treatment of influenza in adults and children aged 13 years and older is 75 mg twice daily for five days. A suspension is available for children aged one year and older and doses are based on weight. For patients with renal impairment (creatinine clearance less than 30 mL/min), the dose should be reduced to 75 mg once daily.[7]

The influenza virus

Influenza viruses are grouped into three types; influenza A, B and C. Influenza A and B cause most clinical disease.

Influenza A viruses are further grouped based on the two antigens on their surface: neuraminidase (N antigen) and haemagglutinin (H antigen). Influenza A and B viruses have a marked ability to change either by:[1, 3-5]

- **Antigenic drift** – minor changes in the H and N antigens as a result of point mutations. Antigenic drift occurs continuously and is responsible for the emergence of strains which differ slightly from those circulating in the previous winter. These new strains are responsible for each winter epidemic, and are the reason why vaccination received in the previous year, will provide little or no protection against the current circulating influenza viruses.
- **Antigenic shift** – major changes in the H and N antigens either arising by direct transmission of an avian virus to humans or after genetic reassortment in pigs, which can be infected with both avian and human viruses. Antigenic shift only occurs in influenza A viruses and has the potential to cause major epidemics and pandemics. Vaccines, which provide protection against influenza strains that circulated before the virus changed by antigenic shift, will provide little or no protection against the new strain. Similarly immunity generated by infection with previous strains will provide little or no protection against the new strain.



Oseltamivir and zanamivir

Oseltamivir and zanamivir are neuraminidase inhibitors

Neuraminidase inhibitors prevent the release of newly replicated virions from infected cells, therefore preventing the spread of infection.[11] Neuraminidase enables infection to spread by cleaving the sialic acid residues on receptors that bind virions to cells and to one another. Neuraminidase inhibitors bind to the active site, preventing the enzyme from cleaving the host-cell receptors.[12]

Resistance to neuraminidase inhibitors

During the 2008–2009 influenza season, high rates of oseltamivir resistant strain (H1N1) of influenza were detected in the United States, Europe, Australia and South Africa. Oseltamivir resistant strains have recently been detected in New Zealand.[13]

Resistance occurs when amino acid substitutions occur in the active site preventing oseltamivir from binding. While resistance to oseltamivir is concerning, this particular strain continues to be susceptible to zanamivir and amantadine.[12]

Local ESR surveillance data reports on which influenza strains are currently circulating and may be used to assist in the choice of an appropriate antiviral agent (available from: **http://www.surv.esr.cri.nz/virology/influenza_annual_report.php**).[13]

Zanamivir (Relenza) is available as an inhaled powder. It is not subsidised and one course costs approximately $65.

The recommended dose of zanamivir for the treatment of influenza in adults and children five years and older is 10 mg (two inhalations) twice daily for five days.[8]

Adverse effects and precautions with neuraminidase inhibitors

Adverse effects commonly associated with oseltamivir include nausea and vomiting (approximately 5 to 10%). This can be minimised by taking oseltamivir with food. Other adverse effects include abdominal pain and headache.[9]

Zanamivir has been reported to cause bronchospasm and a reduction in respiratory function, particularly in patients who have underlying respiratory disease. These people should be informed of the risk of bronchospasm and advised to have a fast-acting bronchodilator available, or if they are taking maintenance bronchodilator therapy, to use this before taking zanamivir.[4, 10]

Amantadine is not usually recommended for the treatment of influenza because of adverse effects and high rates of resistance

Amantadine, more often used for Parkinson's disease, is also licensed for the prophylaxis and treatment of influenza. However amantadine has significant CNS adverse effects including anxiety, insomnia, confusion and light-headedness. These adverse effects are particularly common in elderly people.[14]

There are also high rates of amantadine-resistance in influenza isolates and for this reason it is no longer recommended for the treatment of influenza. One exception is the treatment of oseltamivir-resistant influenza in those whom zanamivir is contraindicated.[6]

Antivirals for prophylaxis

Annual influenza immunisation is recommended to prevent influenza infection in people at high risk of complications. Antivirals are not routinely recommended

for prophylaxis against influenza, however they may be useful in some situations, e.g. in inadequately vaccinated high-risk communities such as an outbreak of influenza in a residential care facility.[9]

In this situation, antivirals must be started within 48 hours after exposure to a person with influenza (i.e. close contact with an infected person).

Doses used for prophylaxis:

- Oseltamivir, adults, 75 mg once daily for ten days
  or
- Zanamivir, 10 mg (2 inhalations) once daily for ten days

When exposure to influenza is ongoing, oseltamivir prophylaxis can be continued for up to six weeks or zanamivir for up to four weeks.[8]

References

1. Ministry of Health. Influenza. Immunisation Handbook 2006. Wellington: Minstry of Health, 2006.
2. Fleming D, Wood M. The clinical diagnosis of influenza. Curr Med Res Opin 2002;18(6):338-341.
3. Clinical Knowledge Summaries. Influenza, 2006. Available from www.cks.library.nhs.uk (accessed April 2009).
4. Dolin R. Epidemiology of influenza. UpToDate, 2008. Available from www.uptodate.com (accessed April 2009).
5. Nicholson KG, Wood JM, Zambon M. Influenza. Lancet 2003;362(9397):1733-45.
6. Zachary KC. Antiviral drugs for the treatment of influenza in adults. UpToDate 2009. Available from www.uptodate.com (accessed April 2009).
7. Roche. Tamiflu datasheet, 2008. Available from www.medsafe.govt.nz (accessed April 2009).
8. GlaxoSmithKline NZ Limited. Relenza datasheet, 2008. Available from www.medsafe.govt.nz (accessed April 2009).
9. Australian Medicines Handbook. Adelaide Australian Medicines Handbook Pty Ltd, 2006.
10. Medsafe. Influenza infection, Relenza (zanamivir) and adverse respiratory events, 2000. Available from www.medsafe.govt.nz (accessed April 2009).
11. Moscona A. Medical management of influenza infection. Annu Rev Med 2008;59:397-413.
12. Moscona A. Global transmission of oseltamivir-resistant influenza. N Engl J Med 2009;360(10):953-956.
13. ESR. Influenza in New Zealand. Public Health Surveillance 2008. Available from www.surv.esr.cri.nz/virology/influenza_annual_report.php (Accessed April 2009).
14. Zachary KC. Pharmacology of antiviral drugs for influenza, UpToDate 2008. Available from www.uptodate.com (accessed April 2009).
15. Royal College of General Practitioners. Department of Health. Preparing for pandemic influenza. Guidance for GP practices. 2008. Available from www.dh.gov.uk (accessed April 2009).
16. World Health Organisation. Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to WHO, 2009. Available from www.who.int (accessed April 2009).

Influenza and the threat of a pandemic

An epidemic is the occurrence of more cases of a disease than would be expected in a community or region in a given time period. A pandemic is an epidemic that has become widespread and is affecting a whole region, continent or the world. Current diseases of pandemic proportions include tuberculosis and HIV.

For an influenza pandemic to occur the virus must be:

1. A new subtype
2. Able to infect humans and cause serious illness
3. Able to spread easily and sustainably between humans

Influenza A (H5N1) –"bird flu"

Avian H5N1 influenza virus (bird flu), which has infected people in Africa, the Pacific, Europe and Asia meets two of these conditions. It is a new virus subtype and is able to infect humans and cause significant disease (from 2003 – 2009, of the 413 cases reported to the World Health Organisation, there have been 256 deaths)[16]

The H5N1 virus does not currently seem to have the ability to pass readily between humans. However it has shown it has the ability to mutate, and acquire genetic material from other strains, and there are fears that the H5N1 virus could potentially develop the ability to spread between people and cause a pandemic.[4]

Influenza A (H1N1) – "Swine flu"

"Swine flu" is the result of a novel reassortment of influenza A H1N1 from avian, swine and human strains. Human cases of this virus, with human to human transmission have been identified in Mexico and have spread to other countries. At the time of going to print, the current pandemic alert status in New Zealand is "Code Yellow" which is a standby phase when there has been a significant development in a virus overseas or single isolated cases in New Zealand.

Most confirmed cases of influenza A (H1N1) have been self-limiting, uncomplicated, respiratory infections with symptoms similar to ordinary seasonal influenza, e.g. fever, cough, headache, myalgia, although vomiting and diarrhoea have been more common.

It is expected that the influenza A (H1N1) virus will cause the same spectrum of illness severity as ordinary seasonal influenza, ranging from self-limited infection to severe illness including pneumonia. Those most likely to get severe illness and complications of influenza A (H1N1) virus are anticipated to be similar to those who would be most at risk during normal influenza outbreaks.

The possibility of influenza A (H1N1) should be considered in those who present with fever and respiratory symptoms who:

- Have developed symptoms within seven days of travel to areas of concern, e.g. Mexico or North America
- Are considered to be a close contact of a probable or confirmed case of influenza A (H1N1)

Any suspected cases of influenza A (H1N1) virus must be notified to the local Medical Officer of Health, who will follow-up and provide necessary treatment.

The influenza A (H1N1) virus is susceptible to oseltamivir and zanamivir but is resistant to amantadine.

For more information visit:
www.moh.govt.nz/influenza-a-h1n1
http://pandemicflu.gov/faq/swineflu

What general practice may need to do to prepare

During a pandemic it is likely that general practice will carry the major burden of disease management in the community.[4]

Things to consider for general practice:

- Implementing national schemes – e.g. having comprehensive lists of at-risk groups who may be contacted in the event that a vaccine becomes available
- Large increase in demand – e.g. coping with increased demand for services, increased home visits, increased numbers of staff off sick, prioritising work and separation of flu and non-flu patients
- How to care for non-flu patients – e.g. patients with chronic conditions requiring routine medication
- Managing spread of infection – e.g. hand hygiene, control of spread from patients who are coughing or sneezing, adequate supplies of protective equipment (surgical face masks, gloves, aprons, eye protection), enhanced cleaning procedures

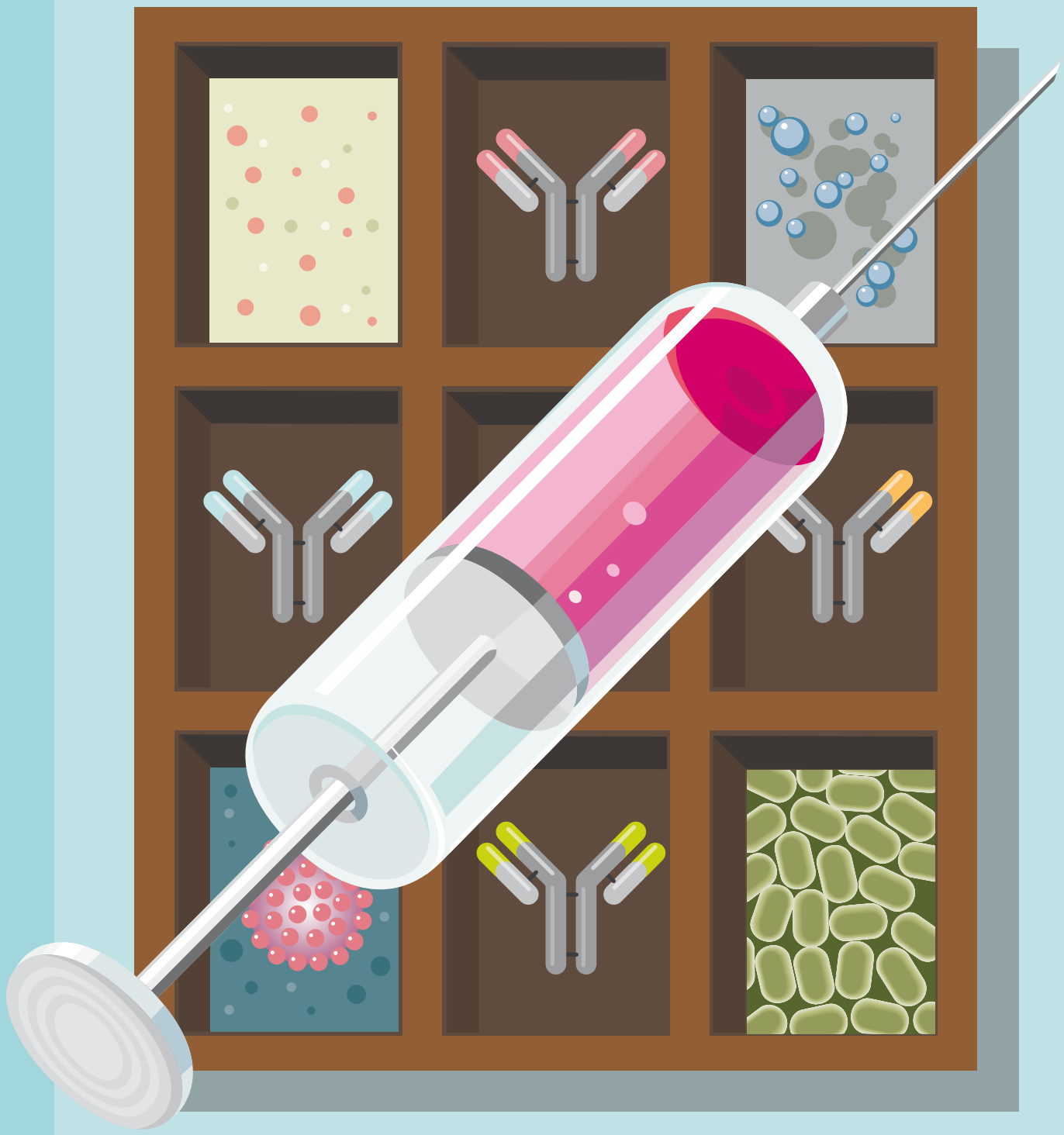
More information about influenza and influenza pandemic planning available from: **www.guidetools.com/influenza/index.html**

The difference between 'ordinary' influenza and pandemic influenza[15]

Feature	"Ordinary" influenza	Pandemic influenza
Influenza virus	Seasonal activity and epidemics usually occur due to minor changes in influenza strains i.e. "antigenic drift"	Usually caused by a completely new influenza virus strain that results from "antigenic shift"
When do they occur?	Every year during the winter months in temperate climates	Pandemics have occurred sporadically throughout history and can take place in any season
How many people may be affected?	Influenza may affect 10–20% of the population and cause approximately 40 deaths in New Zealand annually	A quarter of the population may be affected Associated with much higher rates of illness and death e.g. the 1918 "Spanish Flu" caused around 40 million deaths worldwide
Who is affected?	While anyone can be infected with influenza, elderly people account for most (>90%) of the deaths attributed to influenza and resulting pneumonia	People of all age groups may be affected by pandemic influenza e.g. during the "Spanish Flu" the 20–40 year old age group had a disproportionately high mortality rate
Recovery from influenza illness	Most people with ordinary influenza recover within one to two weeks without requiring medical treatment	Pandemic influenza is usually a more severe illness and therefore associated with a higher risk of death
Vaccine availability	An influenza vaccine is developed each year based on the virus strains expected to be circulating. These can be fairly reliably predicted	Due to the influenza strain being completely new, a vaccine against pandemic influenza will not be available at the start of a pandemic
Treatment and prevention of influenza	Annual vaccination to prevent influenza Antivirals may be used for those at risk of severe influenza and complications	Due to the large numbers affected supply of antivirals may be limited Efficacy for pandemic influenza is not known

Immunisation update

Key reviewer: **Dr Nikki Turner**, Director, Immunisation Advisory Centre, University of Auckland



www.bpac.org.nz keyword: immunisation

THERE WERE THREE significant changes to the immunisation schedule in 2008, with the addition of the new pneumococcal and HPV vaccines and the removal of the special MeNZB programme.

New pneumococcal vaccine for infants

In June 2008, the PCV7 (Prevenar®) vaccine was added to the New Zealand immunisation schedule. This vaccine provides protection against the seven most common strains of *Streptococcus pneumoniae* seen most commonly in infants and implicated in severe pneumococcal disease such as meningitis, septicaemia and pneumonia.

Pneumococcus is also the most common bacterial cause of otitis media in children and a frequent cause of sinusitis and pneumonia in all age groups.

Polysaccharide pneumococcal vaccines such as 23PPV (Pneumovax®23) have been available for many years; however they are not effective in children aged under two years. The introduction of a conjugate pneumococcal vaccine, PCV7 (Prevenar®) will allow for the protection of infants, reducing incidence of disease in the broader community through herd immunity.

It is expected the introduction of the PCV7 vaccine will result in similar benefits in New Zealand to those seen in the United States. Following the introduction of the vaccine in the US in 2000, there was a decline of 85% in invasive pneumococcal disease incidence in young children, and a decline in invasive pneumococcal disease (IPD) in unimmunised adults from the herd immunity effects, created by vaccinating the infants.

Children in New Zealand are offered the PCV7 immunisation at ages six weeks, three months, five months and 15 months.

High-risk pneumococcal programme

Children considered at risk of pneumococcal disease may be eligible for the High-risk Pneumococcal Programme. This is a programme aimed at children aged under five years with a chronic condition. Children who meet the criteria are eligible for the PCV7 (pneumococcal conjugate, Prevenar®) vaccine and the 23PPV (pneumococcal polysaccharide, Pneumovax®23) vaccine at the ages recommended in the immunisation schedule.

Eligibility criteria for the High-risk Pneumococcal Programme:

Children under five years with the following conditions:	
▪ On immunosuppressive or radiation therapy ▪ Primary immune deficiencies ▪ HIV ▪ Renal failure or nephrotic syndrome ▪ Organ transplants ▪ Cochlear implants or intracranial shunts ▪ With chronic CSF leaks ▪ Cardiac disease with cyanosis or failure ▪ Insulin dependent diabetes ▪ Down syndrome	▪ On corticosteroid therapy for more than two weeks, at daily dose of prednisone of 2 mg/kg or greater, or a total daily dosage of 20 mg or more ▪ Children pre or post splenectomy or with functional asplenia ▪ Pre-term infants, born at under 28 weeks' gestation ▪ Chronic pulmonary disease (including asthma treated with high-dose corticosteroid therapy)

MeNZB vaccine programme

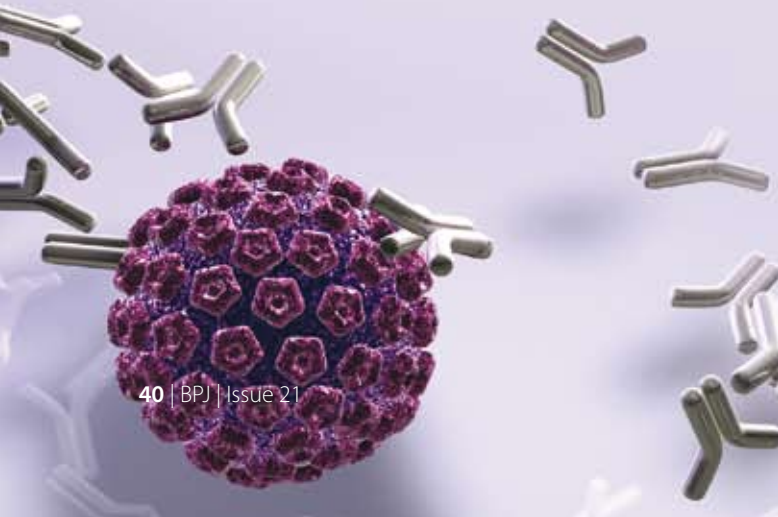
The MeNZB vaccine was introduced to control an epidemic of a specific strain of Group B meningococcus circulating in New Zealand. There has been a significant sustained decrease in confirmed cases since the completion of the programme in 2006. With the epidemic waning, MeNZB is no longer on the National Immunisation Schedule.

The MeNZB vaccine is still available and funded for individuals of any age, with a high risk of invasive meningococcal infection, and specific conditions including:

- Actual or functional asplenia
- Sickle cell anaemia
- Some complement deficiencies
- Individuals with HIV infection, who may be safely immunised with meningococcal polysaccharide vaccines.
- Microbiology and laboratory workers

HPV vaccine programme

See BPJ 18 (December 2008) for information on the new HPV vaccine.



Contraindications and precautions to vaccination

Contraindications

There are only a few contraindications to vaccination, these are listed in Table 1.

Precautions

There are a number of precautions to vaccination.

Giving a live vaccine less than four weeks after another live vaccine

There is a theoretical risk that the administration of multiple live virus vaccines within four weeks of one another, if not given on the same day, will result in a suboptimal immune response.

Pregnancy

Generally, vaccines are not tested in pregnant woman therefore there is little safety data available for this group. However in other countries the use of the influenza vaccines (and others) in pregnant women has been shown to be safe.

Allergy to Vaccine components

Provided there is no history of anaphylaxis, allergies to vaccine components, such as asthma following exposure to feathers or a rash following consumption of eggs, should be treated as a precaution only. A longer period of observation following immunisation may be prudent.

Guillain Barré Syndrome

In people with a history of Guillain Barré Syndrome (GBS) within six weeks of previous influenza vaccination, but who are not at high-risk for severe influenza complications, it is prudent to avoid further influenza vaccination.

In people with a history of GBS, but also at high-risk for severe complications from influenza, the established benefits of influenza vaccination justify yearly vaccination.

Thrombocytopenia or history of thrombocytopenic purpura and MMR

In most instances, the benefits of vaccination are greater than the potential risks and will justify giving MMR, particularly in view of the even greater risk of thrombocytopenia following measles or rubella disease.

Haemophilia and related bleeding disorders

People with haemophilia and related bleeding disorders should be immunised. In some cases of severe haemophilia the vaccine can be given subcutaneously rather than intramuscularly. Prophylaxis should be given on the same day as the vaccine.

False contraindications

The following conditions or circumstances are not contraindications to vaccination:

- Minor infections without significant fever or systemic upset
- Asthma, hayfever, eczema, "snuffles"
- Severe allergy to foods or medications unrelated to the vaccine
- Treatment with antibiotics or locally acting steroids
- Pregnancy in the child's mother
- A child who is breastfeeding
- Neonatal jaundice
- Low weight in an otherwise healthy child
- The child being over the usual age for immunisation
- Family history of vaccine reactions, seizures or Sudden Infant Death Syndrome
- Prematurity in an otherwise well infant who is not in hospital
- Established neurological conditions such as cerebral palsy or Down syndrome
- Contact with an infectious disease
- Clinical history of pertussis, measles, mumps or rubella (clinical history without laboratory confirmation can not be taken as proof of immunity)

Table 1: Vaccine Contraindications

Vaccine	Contraindications
All Vaccines	▪ Anaphylactic type reaction to a previous dose of that vaccine, or to any vaccine component (not trace element)
Pertussis-containing vaccines	▪ Previous encephalopathy within seven days after a previous pertussis-containing vaccine ▪ Evolving (undiagnosed) neurological problem
Measles, Mumps, Rubella, MMR, Varicella, Yellow Fever, Oral Polio	▪ Immunosuppressed individuals ▪ If blood, plasma or immunoglobulin were given in the last 11 months ▪ Pregnancy
Influenza, Yellow Fever	▪ Anaphylactic reaction to chickens, including eggs, egg protein, feathers etc

Epidemic update

Pertussis (Whooping cough)

New Zealand currently appears to be in the early phases of a pertussis epidemic. Recent surveillance data shows a marked increased in pertussis cases, from 28 cases in February 2008 to 140 cases in February 2009. The highest numbers of cases are being reported from Canterbury, Nelson Marlborough and Waikato DHBs.

New Zealand has a pertussis epidemic every four to five years, with the most recent epidemics in 1999–2001 and 2004. In 2004 alone, 3489 cases were reported. Since 2000 four infants have died from pertussis. Three out of the four were too young to have been immunised.

Minimal maternal protection to pertussis is passed to the foetus and breast-feeding offers very little protection. Infants who are too young to be fully immunised are vulnerable to disease. Their only protection is from other methods such as herd immunity, vaccinating close contacts and avoiding contact with those carrying the bacterium.

The best way to contain an epidemic is immunisation and effective management of confirmed cases

It is important to ensure children get their immunisations on time and "catch up" immunisations are offered to those who are overdue. At four years and 11 years children have booster pertussis vaccinations which provide protection through adolescence.

Adults can also be given a pertussis booster vaccine and in particular, close contacts of infants such as parents, grandparents and health professionals, should consider receiving a pertussis booster vaccination.

Management of confirmed cases includes exclusion of the infected person from school or work, until they have received at least five days of a 14-day course of erythromycin, or exclusion for three weeks from the date of onset of typical paroxysms of cough.

When the household includes any child aged less than 12 months, who has received fewer than three doses of pertussis vaccine, then other members of the household should also be given a course of antibiotics (14-day course of erythromycin).

Pertussis is a notifiable disease and it is essential to report suspected and confirmed cases to the local Medical Officer of Health. Collection of a nasopharyngeal swab is indicated in suspected cases.

Measles outbreak

Since the start of 2009 there has been an increased number of confirmed cases of measles. Between January and March 2009, ESR has recorded a total of 28 confirmed cases - 23 of which were reported in the Otago DHB region. Local data from Public Health South indicates that the number of cases of measles in people aged 4 to 22 years in Otago, since January may be as high as 31. "To put this into perspective, in the whole of the United States there are on average 64 cases of measles a year"–Richard Bunton, Chief Medical Officer, Otago DHB.

It has been estimated that to prevent recurrent outbreaks of measles, 95% of the population must be immune. This level of immunity has been difficult to achieve because the measles vaccine efficacy is 90–95% and not all children receive the first scheduled dose. To improve the overall level of community immunity, a course of two vaccines for all children is recommended at age 15 months and four years

References

Ministry of Health. 2008 National Immunisation Schedule. Health Provider Booklet. Wellington: Ministry of Health, 2008.

Immunisation Advisory Centre. Health professionals online resource centre: Available from: www.immune.org.nz (Accessed April 2009).

www.bpac.org.nz keyword: fundedmedicine

Accessing funded medicines in New Zealand

www.bpac.org.nz keyword: cvdguidelines

What's new in the 2009 New Zealand Cardiovascular Guidelines Handbook?

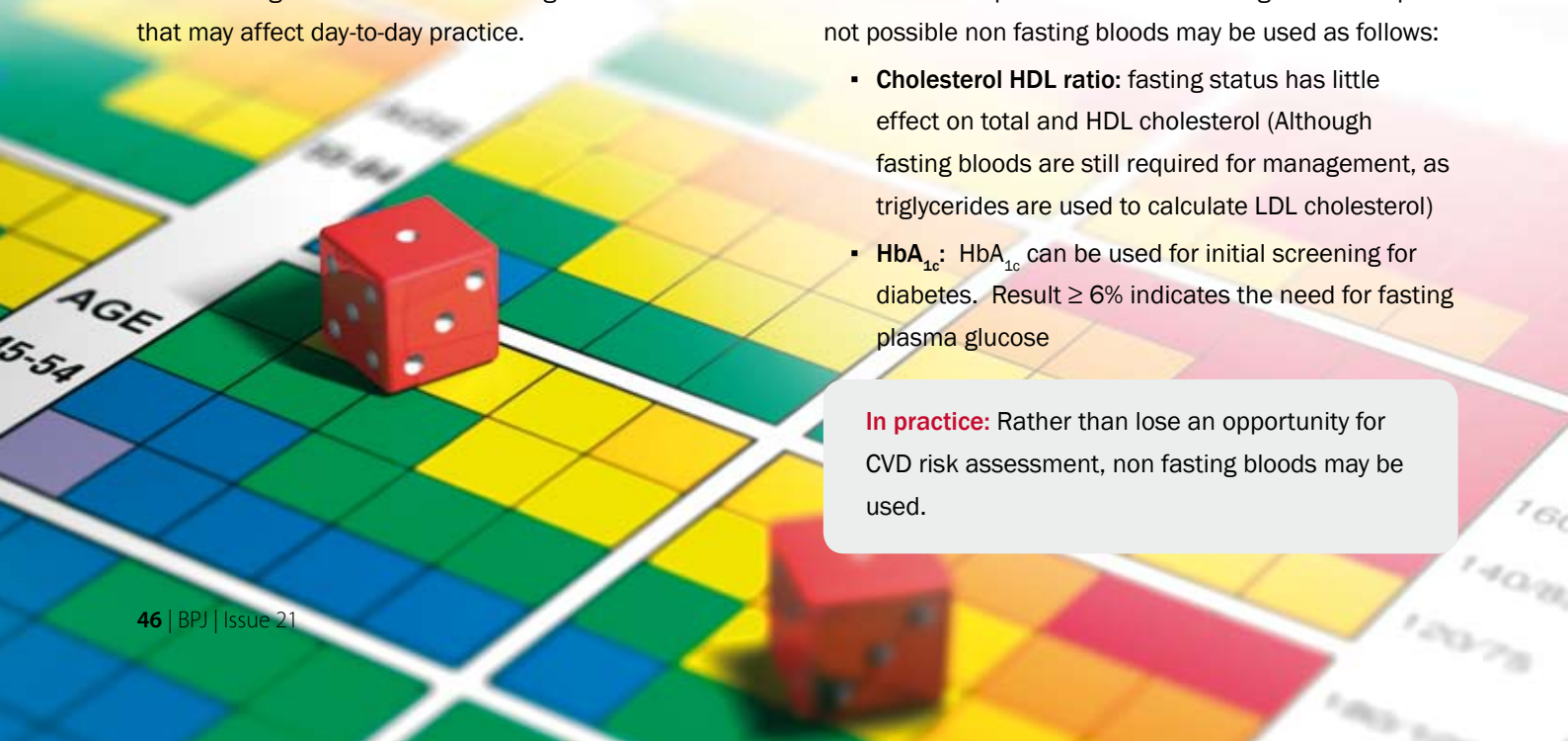
Key reviewer: **Dr Michael Crooke**, Chemical Pathologist, Wellington Hospital and Aotea Pathology

The New Zealand Guidelines Group has recently released their updated Cardiovascular Guidelines Handbook.

Topics covered in the handbook include:

- Cardiovascular risk assessment and diabetes screening
- Cardiovascular risk factor management
- Smoking cessation
- Atrial fibrillation
- Coronary heart disease
- Stroke and transient ischaemic attack
- Rheumatic fever (new)
- Prevention of infective endocarditis (new)
- Heart failure

The following article details the changes to the handbook that may affect day-to-day practice.



Cardiovascular Risk charts

There are two main differences in the cardiovascular risk charts:

- Ages bands on the risk charts now state an age range (i.e. 55–64 years), instead of choosing the age closest to the patient (i.e. 60 years)
- Only systolic blood pressure is required for the calculation of risk

In practice: Less ambiguity for both age and blood pressure making the charts easier to use

Non-fasting blood tests may be used in some circumstances

Initial assessment using fasting blood tests remains recommended practice. When a fasting blood sample is not possible non fasting bloods may be used as follows:

- **Cholesterol HDL ratio:** fasting status has little effect on total and HDL cholesterol (Although fasting bloods are still required for management, as triglycerides are used to calculate LDL cholesterol)
- **HbA_{1c}:** HbA_{1c} can be used for initial screening for diabetes. Result ≥ 6% indicates the need for fasting plasma glucose

In practice: Rather than lose an opportunity for CVD risk assessment, non fasting bloods may be used.

Renal disease recognised as contributing to cardiovascular risk

eGFR has become well accepted as a means of assessing renal function, therefore the handbook recommends that both ACR (albumin : creatinine ratio) and eGFR have roles in assessing renal function, and in guiding further management of those with diabetes or renal disease.

People with an eGFR <60ml/min/1.73m^2 should begin having CVD risk assessments at age 35 years for men and age 45 years for women.

In practice: Start CVD risk assessment for people with an eGFR <60ml/min/1.73m^2 at age 35 years for men and age 45 years for women

Lipids targets lower

Optimal targets for lipids for people with CVD, diabetes or a calculated CVD risk greater than 15% are lower than in the previous handbook.

The target for:

- LDL cholesterol is now less than 2.0mmol/L (down from 2.5 mmol/L)
- Total cholesterol/HDL ratio is now less than 4.0 (down from <4.5)
- Total cholesterol remains at less than 4.0 mmol/L

In practice: Be aware of new optimal targets for lipid lowering, more aggressive treatment may be required

New blood pressure target people with chronic kidney disease

The handbook now recommends more aggressive management of blood pressure for people with chronic kidney disease, setting a target of less than 125/75 mmHg.

In practice: Be aware of new optimal targets for blood pressure in people with chronic kidney disease, more aggressive treatment may be required.

Change in the recommended frequency of CVD risk assessment

The new handbook recommends frequent CVD risk assessments for people with a CVD risk of between 10–15%. These people should have a CVD risk assessment every two years.

In practice: Update your recalls for people with a CVD risk of 10–15%

Metabolic syndrome no longer recognised as a separate risk factor

The definition of metabolic syndrome as an entity remains contentious, and there is no clear evidence of its importance as a risk factor, aside from the other recognised risk factors for CVD.

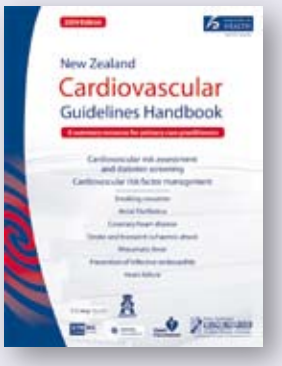
See BPJ 18 (December 2008) "Metabolic Syndrome: Useful or not?"

Universal BMI target

Separate BMI's for Māori and Pacific peoples have been omitted; the handbook now includes one BMI table. A BMI of less than 25kg/m^2 is considered desirable. This level may be lower for people of Asian descent.

Advice on diabetes management has been removed.

Advice on diabetes management has been removed pending a full revision of the Type 2 Diabetes Management Guideline due in 2010.



New Zealand Cardiovascular Guidelines Handbook 2009 Edition. Available from: www.nzgg.org.nz (downloadable online version plus order form for hard copy).

Self Management Plans for Asthma
– obsolete or needing a fresh start?

www.bpac.org.nz keyword: asthma

Contributed by **Professor D Robin Taylor**, Medical Adviser, Asthma and Respiratory Foundation of New Zealand

THE GOOD NEWS ABOUT ASTHMA is that hospital admission rates have halved since their peak in the mid 1980s. The bad news is that they have remained almost static for the last ten years, and are still occurring at twice the rate of the 1970s (Figure 1). Hospital admissions are a good surrogate for other important morbidity associated with asthma e.g. days off work and school. The problem of asthma morbidity still requires our attention.

One approach to this problem has been the promotion of Self Management Plans (occasionally referred to as Action Plans if the focus is exclusively on how to treat acutely deteriorating asthma). These are designed to encourage early intervention so that even if an acute exacerbation of asthma cannot be prevented, perhaps its severity can be significantly reduced. The literature is mixed as to the success of Self Management Plans ranging from enthusiasm to ambivalence.[1-3] Perhaps for this reason, complacency has set in and they have become less commonly used. In one Canadian report only about 10% of patients with asthma had an Action Plan.[4]

Targeting vulnerable patients

Plans are not necessary for everyone. However, at the very least, Self Management Plans should be given to individuals who are susceptible to troublesome or life-threatening asthma. This is judged on their individual

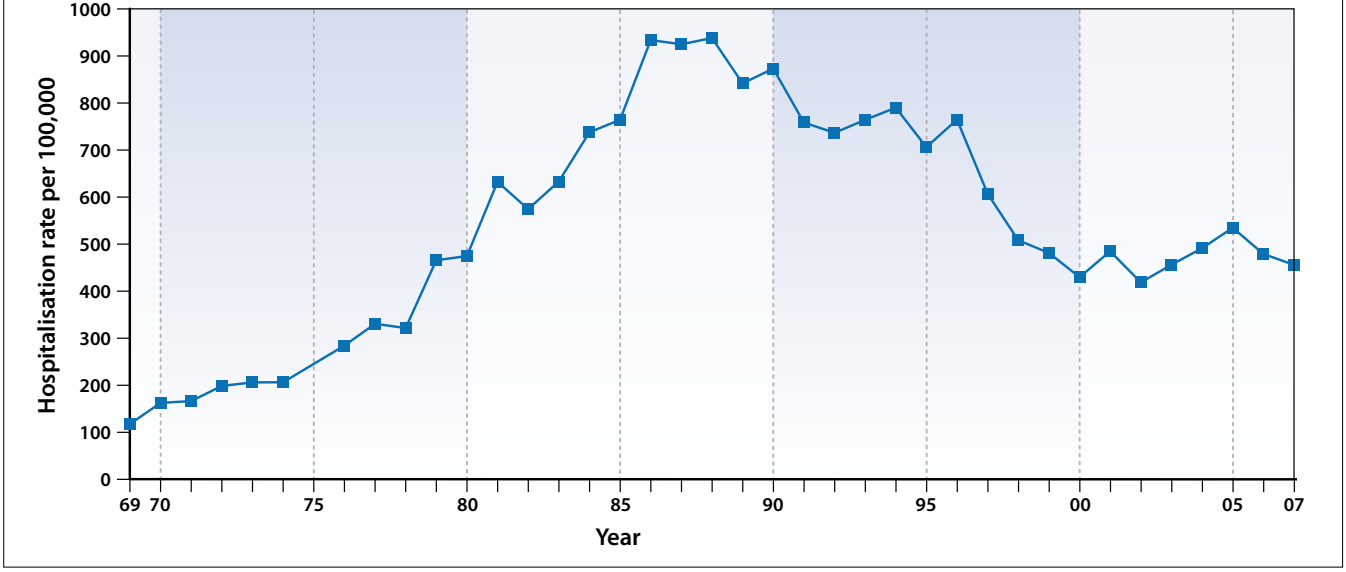


Figure 1: New Zealand hospital admission rates for asthma

history. What has happened in the past has the potential to happen again in the future. If a patient's history includes life-threatening episodes, hospitalisation, or more commonly, the need for a course of oral steroid, then time and effort should be devoted to providing a Plan for these individuals so that the past is not repeated. Patients with "brittle asthma" should also be targeted.

Educational and other objectives

The aim is to empower patients to take action which will reduce the severity of asthma exacerbations and hence the risk. Delay should be avoided. Limited access and the financial cost of medical advice after hours or at weekends should be factored in. Any disincentive to early intervention should be addressed. There is clear evidence that a Plan should be written rather than just oral. If possible it should be provided to a carer or immediate support person as well as the patient.

Heeding the warning signs

It helps to work through and reflect on the narrative of a previous asthma exacerbation and focus on key warning signals. Plans should usually be based on changes in symptoms rather than peak flows. The onset, or an increase in night waking or an increasing need for "reliever" inhaler medication, are classic signs of deteriorating asthma.

Using peak flows should be encouraged to objectively verify what the patient is experiencing. In "poor perceivers" they may have an even more critical role. If possible use peak flow data from a previous exacerbation. For example, if a patient previously required admission to hospital and at that time the peak flow was 250 L/min., then this is clearly undesirable irrespective of whether it is 70% or 40% of the predicted peak flow. The intervention with oral steroid should have begun when the peak flow reached say 350 L/min. There is scope for variation in the threshold for intervention depending on experience.

Intervention

Instructions should be given regarding the use of "reliever" bronchodilator as well as the use of oral prednisone. The liberal use of beta-agonist in this setting is not contra-indicated, but adverse effects may occur when consumption remains high for more than a few days. The use of Symbicort (formoterol/budesonide) as "reliever" (so-called SMART regimen) should be limited to 12 additional doses. Thereafter if bronchospasm persists, salbutamol via a spacer should be used.

Different practitioners may advise different regimens for oral prednisone tapering. There is no evidence one way or the other. What the patient has used in the past and is familiar with will usually suffice. Prednisone 40 mg/day for five days followed by 20 mg/day for five days is our usual "recipe". It is not set in stone. A shorter period of treatment may suffice. It may also be appropriate to give the patient a supply of prednisone tablets.

The Asthma and Respiratory Foundation of New Zealand has developed a revised version of its Self-Management Plan for asthma (available from **www.asthmanz.co.nz**). Patients with asthma, as well as their family doctors and practice nurses, are encouraged to revisit using a Self Management Plan. Perhaps we can again make inroads into reducing asthma morbidity in New Zealand.

Further reading

1. Gibson PG, Powell H. Written action plans for asthma: an evidence-based review of the key components. Thorax 2004;59(2):94-9.
2. Toelle BG, Ram FS. Written individualised management plans for asthma in children and adults. Cochrane Database Syst Rev 2004(2):CD002171.
3. Zemek RL, Bhogal SK, Ducharme FM. Systematic review of randomized controlled trials examining written action plans in children: what is the plan? Arch Pediatr Adolesc Med 2008;162(2):157-63.
4. Beauchesne MF, Levert V, El Tawil M, Labrecque M, Blais L. Action plans in asthma. Can Respir J 2006;13(6):306-10.

Adverse reaction reporting tool

Contributed by Medsafe Clinical Risk Management Team

Key concepts

- An electronic adverse reaction reporting tool has been launched in New Zealand
- Reporting suspected adverse reactions enables the detection of medicine safety signals
- The reporting tool pre-populates patient details making reporting adverse reactions easier and allowing more data to be included
- The reporting tool will help with the identification of medicine safety issues and enable more timely advice to be provided to prescribers

Launch of an electronic adverse reaction reporting tool

Adverse reaction reporting is regarded as one of the most important sources of data for assessing the safety of a medicine. Adverse reaction reports enable the detection of medicine safety signals and medicine quality defects.

On April 1 2009, the Minister of Health launched a new electronic adverse reaction reporting tool in New Zealand.

The tool is designed to facilitate the reporting of adverse reactions to the Centre for Adverse Reactions Monitoring (CARM), and it uses an online reporting form pre-populated with patient details from the GP practice software.

The reporting form can be easily accessed by clicking on an icon within the Patient Management Software (a link to the instructions for creating an icon for the reporting form on the MedTech32 pallet can be found in the *bestpractice* Decision Support news items on the menu page). Once opened the tool automatically pre-populates the patient's medical history, medicine history and gives the reporter the option of including laboratory test results.

As vaccines make up approximately 50% of the adverse reaction reports received every year, the tool has been designed with a specific vaccine tab. If the suspected medicine is a vaccine, the tool pre-populates the batch number, the date of administration and how the vaccine was given.

Once a description of the reaction and other pertinent information is entered, one click of the mouse sends a confidential encrypted report electronically to CARM.

ADR reporting in New Zealand

CARM receives on average 4000 spontaneous adverse reaction reports a year. General Practice accounts for approximately 60% of these adverse reaction reports.

When CARM receives a report, it is processed, coded then assessed by expert clinicians. Every report receives a personal reply from CARM. including advice on the likely cause of the reaction, information specific to that reaction and how frequently the reaction is reported.

The World Health Organisation rates New Zealand as having the highest number of adverse reaction reports submitted per capita compared to other countries in their programme. In addition, reports from New Zealand are also regarded as being of the highest quality. This is because New Zealand has one of the best reporting systems in the world. It is also apparent that New Zealand's healthcare professionals, who are interested in the safety of medicines, are motivated to report and understand that adverse reaction reporting is part of their professional responsibility.

Although our adverse reaction reporting is rated highly, research indicates that at best only one in ten adverse reactions are being reported in New Zealand i.e. the rate of under-reporting is in excess of 90%. Moreover, recent research conducted in New Zealand examined the data stored in the Patient Management Systems of 30 General Practices. Of the 725 entries in the medical warnings files, that recorded an adverse reaction or allergy to at least one medicine, only 21 were reported to CARM.

As many GPs will know there are a number of barriers to reporting adverse reactions. These barriers include the absence of a prompt to initiate reporting, realising that an adverse reaction has occurred, considering that the reaction is already well known and finally, the time required to manually fill in reaction forms.

What are the benefits of using this tool?

First and foremost, the adverse reaction tool has been developed to help decrease the time involved in reporting. Pre-populating the reporting form with patient data means manual entry of information is minimal. Electronic reporting means less paperwork for busy GPs and removes the need to post or fax reports to CARM.

The ability to extract data from Patient Management software makes it easier for reporters to include results from laboratory tests and other investigations. It is hoped that this will improve the ability of CARM's experts to review the data and to determine whether the medicine is causing the reaction.

Improving the analysis of adverse reaction reports is expected to provide direct benefits for all healthcare professionals. As well as improving the identification of medicine safety issues, it will enable more timely advice to be provided. In the future CARM will provide feedback to reporters electronically. This information can then be entered directly into the patient's records.

CARM is also able to add patient specific alerts through the medical warning module of the NZHIS system. Alerts are attached to the patient's unique NHI number so,

for example, when a patient is admitted to hospital the presence of the alert reduces the risk of that patient receiving medication they have already reacted to.

Reporters can be assured that the confidentiality of patient and reporter details is maintained in the electronic reporting tool. As with the paper-based form, the information provided in the report is only viewed and used by CARM.

This new reporting tool is one of the first in the world that allows direct electronic reporting of adverse reactions from GP practices. Regular use of the system will strengthen the close relationship that exists between prescribers and the medicines safety community, and cement New Zealand's position as a world leader in monitoring and managing medicines safety issues.

bestpractice
DECISION SUPPORT FOR HEALTH PROFESSIONALS

Adverse Drug Reaction Reporting

Medsafe funded Module

This module has been developed on behalf of Medsafe to improve and facilitate the electronic reporting of adverse drug reactions in general practice. This online reporting form automatically populates details from the general practice Practice Management Software, including current medications and vaccinations.

The completed form is sent electronically to the Centre for Adverse Reactions Monitoring (CARM) while a copy is retained within the patient record.

***Bestpractice* now provides this nationally funded module at no cost to practices**

Enquire now to access the latest innovation to improve patient management

Contact

Jamie Murley
bestpractice Decision Support
Level 8, 10 George Street
PO Box 6032
Dunedin
Ph: 03 479 2816 Fax: 03 479 2569
Email: **jamiem@bpac.org.nz**

www.bestpractice.net.nz

Evidence That Counts

Four Approaches to Dyspepsia

Journal Watch, Vol. 29, No.7, April 1, 2009

All yielded similar 1-year outcomes.

Initial approaches to dyspepsia vary considerably among clinicians. In a U.K. trial involving 762 patients (28% age ≥50) with dyspepsia, investigators compared outcomes of four treatment strategies. Patients with symptoms that suggested malignancy and those with previously diagnosed esophagitis or peptic ulcer were excluded.

Patients were randomised to one of four treatment groups:

- Early endoscopy: Patients underwent endoscopy with urease testing for *Helicobacter pylori* (HP) infection. HP-positive patients with ulcers or erosions received treatment to eradicate HP; all others received proton-pump inhibitors (PPIs) for one month.
- Test and refer: Patients underwent serologic HP testing. HP-positive patients underwent endoscopy and were treated similarly to those in the early-endoscopy group; HP-negative patients received PPIs for one month.
- Test and treat: Patients underwent serologic HP testing. HP-positive patients received eradication therapy; HP-negative patients received PPIs for one month.
- Empirical treatment: Patients received PPIs for one month.

About one third of patients whose initial management did not involve endoscopy eventually underwent the procedure because of persistent symptoms. At one year, about half of patients in each group were asymptomatic, and patient satisfaction and overall use of dyspepsia medications were similar in the four groups. The early endoscopy group had the fewest sub sequent office visits for dyspepsia. Test-and-treat was the most cost-effective strategy. Overall, only four cancers were diagnosed.

Comment

The finding that one-year outcomes were similar with four approaches to dyspepsia should apply to populations in which prevalence of *Helicobacter pylori* infection is similar to the 37% prevalence in this study. The authors conclude, reasonably, that early endoscopy remains appropriate in older populations and that test-and-treat or empirical therapy is appropriate in younger populations.

— Allan S. Brett, MD

Reference

Duggan AE et al. Clinical trial: A randomised trial of early endoscopy, *Helicobacter pylori* testing and empirical therapy for the management of dyspepsia in primary care. Aliment Pharmacol Ther 2009 Jan; 29:55

Prescribe systemic corticosteroids in acute asthma

NeLM 06/04/2009
www.nelm.nhs.uk

In this article, the authors look at the available evidence from meta-analyses of systemic corticosteroids in acute exacerbations of asthma, and how this intervention has been shown to reduce rates of admission, relapse, and symptom duration. Despite this, studies have shown that systemic corticosteroids are under-prescribed, and they discuss the possible barriers to change.

The following key points are highlighted:

- Prescribe systemic corticosteroids for all but the

mildest exacerbations of acute asthma

- Systemic corticosteroids reduce admission rates, relapse rates, symptom duration, and requirement for "reliever" medications
- An appropriate daily dose is 1mg/kg a day of prednisolone (or equivalent) for up to seven days in adults and for three to five days in children
- Insufficient evidence exists that inhaled corticosteroids are as effective as oral steroids after acute asthma attacks.
- Inhaled corticosteroids have not yet been shown to be as effective as oral steroids for acute asthma attacks

—Nicola Pocock

Reference

BMJ 2009;338:b1234 (published early online 3rd April 2009)

Diagnosis and Treatment of Adult Asthma

New Zealand Guidelines Group Sept 2002

Pharmaceutical principles in acute asthma: systemic corticosteroids

- Systemic corticosteroids should be given early in acute severe asthma.
- A short course of corticosteroids according to response (eg, 40 mg prednisone for 4 –10 days) following an acute exacerbation of asthma reduces the number of relapses requiring additional medical care (OR 0.35, NNT=13) and decreases ß2-agonist use without any apparent increase in adverse effects.
- There is no evidence of benefit in using a dose of more than 100 mg of prednisone or prednisolone.
- Inhaled corticosteroids reduce admission rates in people with acute asthma who are not receiving concomitant systemic corticosteroids. However, there is insufficient evidence to determine whether ICS provide additional benefit when used in combination with standard systemic oral corticosteroid therapy. There is some evidence that high dose ICS alone may be as effective as oral corticosteroid therapy when used in mild asthmatics but further research is required to clarify this. Oral prednisone is recommended for all acute severe episodes.

Full text available from: **www.nzgg.org.nz**

US guideline on low-dose aspirin for primary cardiovascular prevention

NeLM 17/03/2009

www.nelm.nhs.uk

The U.S. Preventive Services Task Force (USPSTF) has updated its previous (2002) recommendations on use of aspirin for prevention of cardiovascular disease. The authors have reviewed the evidence published since the previous recommendation to assess the benefits and harms of taking aspirin for the primary prevention of myocardial infarctions (MI), strokes, and death. As there is an indication of possible sex-related difference, the review and recommendations are structured according to sex.

The literature search located new evidence from one good-quality randomised controlled trial (RCT), one good-quality meta-analysis, and two fair-quality sub-group analyses of RCT (not including the CHARISMA analysis published in the same journal issue). After analysis of the new data in association with the previous guideline, the authors conclude that aspirin use reduces the number of CVD

Evidence That Counts

events in patients without known CVD. Men have fewer MI, and women fewer ischaemic strokes. Aspirin does not seem to affect CVD mortality or all-cause mortality in either men or women. The risk for major bleeding events, primarily gastrointestinal bleeding events, is increased in both men and women. Men, but probably not women, have an increased risk for haemorrhagic strokes with aspirin use.

Based on the literature review, the authors recommend that clinicians:

- Encourage men aged 45 to 79 years to use aspirin when the potential benefit of a reduction in myocardial infarctions outweighs the potential harm of an increase in gastrointestinal haemorrhage.
- Encourage women aged 55 to 79 years to use aspirin when the potential benefit of a reduction in ischemic strokes outweighs the potential harm of an increase in gastrointestinal haemorrhage.
- Do not encourage aspirin use for cardiovascular disease prevention in women younger than 55 years and in men younger than 45 years.

They consider that evidence is insufficient to assess the balance of benefits and harms of aspirin for cardiovascular disease prevention in men and women 80 years or older.

An accompanying editorial discusses the guideline, along with the CHARISMA analysis published in the same journal issue. The author concludes that the guideline is clear and user-friendly: its routine use will increase use of aspirin and prevent many cardiovascular events. Nevertheless, shared decision-making with the patient will be important, with discussion of the risks and benefits.

Reference

Ann Intern Med 2009; 150: 396-404; 405-10; 414-6

N.B. New Zealand Cardiovascular Guidelines Handbook. 2009 Edition.

Cardiovascular risk factor management: Long-term antiplatelet therapy

- Aspirin reduces the risk of a cardiovascular event by about 25% over 5 years
- The decision to use aspirin should be based on a balance of the risks and benefits for each person taking into account their absolute risk of an event (see Table opposite)

Pneumococcal Polysaccharide Vaccine: Efficacy Remains Controversial

Journal Watch, Vol. 29, No.4, February 15, 2009

Although pneumococcal polysaccharide vaccine is a standard preventive intervention — and serves as a "quality indicator" in some settings — controversy over its effectiveness simmers in the background. This latest contribution is a meta-analysis funded by the World Health Organisation, which recently published a position paper on this topic.

Researchers analysed 22 randomised trials with 100,000 subjects. The current 23-valent vaccine was used in eight trials, and previous lower-valent vaccines were used in the others. In the overall analysis, pneumococcal vaccine significantly lowered incidences of "presumptive pneumonia" and "all pneumonia" (by 36% and 27%, respectively). However, the vaccine was ineffective in a subgroup of higher-quality trials (e.g., double-blind trials with adequate concealment of allocation). Moreover, the vaccine was ineffective in older people and adults with chronic illness, regardless of study quality. The effect

Indications for long-term aspirin use

5-year CVD risk	Recommendation
Risk >20% clinically	After angina or MI commence low dose aspirin (75–150 mg), a beta-blocker, a statin and an ACE inhibitor After ischaemic stroke or TIA commence low dose aspirin and a statin. Start or increase doses of BP lowering drugs (two usually required)
Risk calculated >15%	Commence low dose aspirin (75–150 mg/day) unless contraindicated Low dose aspirin is as effective as higher daily doses and may be associated with less bleeding
Risk assumed to be >15%: isolated high-risk factors • TC ≥8 mmol/L • TC:HDL ratio≥8 • BP ≥ 170/100 mm Hg	
No clinical CVD and calculated 5-year CVD risk <15%	The risk of a significant bleed or major haemorrhage **outweighs** the benefits of aspirin for the prevention of CVD. Other indications may exist

Full text available: **www.nzgg.org.nz**

of vaccine on pneumococcal bacteremia was unclear because too few cases were available for analysis.

Comment

The authors express reservations about current recommendations to provide pneumococcal polysaccharide vaccination for all older adults (age, ≥65) and for younger adults with chronic diseases. However, editorialists dispute certain elements of this meta-analysis and believe that vaccination at least protects against invasive (i.e., bacteremic) pneumococcal disease. The authors and the editorialists both express hope that herd immunity from use of the newer pneumococcal conjugate vaccine in children will lower the incidence of pneumococcal disease in adults, as has been suggested recently (N Engl J Med 2009; 360:244).

— Allan S. Brett, MD

Reference

Huss A et al. Efficacy of pneumococcal vaccination in adults: A meta-analysis. CMAJ 2009 Jan 6; 180:48. Andrews R and Moberley SA. The controversy over the efficacy of pneumococcal vaccine. CMAJ 2009 Jan 6; 180:18.

Increase in HDL cholesterol does not reduce cardiovascular disease morbidity and mortality

NeLM 16/02/2009
www.nelm.nhs.uk

According to data published online in the BMJ, "simply increasing the amount of circulating HDL cholesterol does not reduce the risk of coronary heart disease events, coronary heart disease deaths, or total deaths."

These findings come from a systematic review and meta-regression analysis of 108 RCTs involving 299,310 patients at risk of cardiovascular events. The studies tested lipid modifying interventions to reduce cardiovascular risk, reported HDL cholesterol and mortality or myocardial infarctions separately for treatment groups, and treated and followed participants for at least six months. The following findings were reported:

- All analyses that adjusted for changes in LDL cholesterol showed no association between treatment induced change in HDL cholesterol and risk ratios for coronary heart disease deaths, coronary heart disease events (coronary heart disease death and non-fatal myocardial infarction), or total deaths.
- With all trials included, change in HDL cholesterol explained almost no variability (< 1%) in any of the outcomes.
- The change in the quotient of LDL and HDL cholesterol did not explain more of the variability in any of the outcomes than did the change in LDL cholesterol alone.
- For a 0.26 mmol/l reduction in LDL cholesterol, the relative risk reduction when adjusted for change in high density lipoprotein cholesterol and drug class was:

  7.2% (95% CI, 3.1% to 11%; p = 0.001) for coronary heart disease deaths

  7.1% (4.5% to 9.8%; p < 0.001) for coronary heart disease events

  4.4% (1.6% to 7.2%; p = 0.002) for total deaths

The researchers note that though some of the treatments increased levels of HDL cholesterol, these increases were not independently associated with a decrease in cardiovascular risk. This was in contrast to decreases in LDL cholesterol that occurred with many of the treatments evaluated, and which were strongly associated with reduced risk of cardiovascular disease. They conclude that their findings support reduction in LDL cholesterol as the primary goal for lipid modifying interventions to modify cardiovascular risk.

They acknowledge that these findings are limited by the use of aggregated study data rather than individual patient data in the meta-regression analysis, and the analytical power was constrained by the modest change and variability in mean HDL cholesterol concentrations in available studies (mean increase of 0.04 mmol/l).

An accompanying editorial notes that the clinical message of this analysis is relatively simple and consistent with clinical practice guidelines, i.e. LDL cholesterol should be the primary target of lipid lowering treatments. However, it adds that the study does not prove that increasing HDL in selected patients with low HDL cholesterol has no value, because the meta-regression analysed mean HDL cholesterol changes in each trial only. What is less clear is whether low HDL cholesterol concentrations should be treated with drugs in patients already receiving targeted treatment for LDL cholesterol, as efficacy and safety data on combined treatments are limited. The editorial concludes "the demonstration that a marker is independently associated with risk does not mean that treatments that modify levels of the marker will also modify clinical risk."

—Yuet Wan

Reference

BMJ 2009; 338: b92 (study), a3065 (editorial)

Management of impetigo

Dear bpac,

I have just read about the management of impetigo in BPJ 19 (February 2009). I have recently been asked about treating asymptomatic elderly carriers of MRSA that are resident in a rest home. This was on the basis of the eradication of the carrier status in a patient that was treated with antibiotics for another infection.

Is there any evidence for treating asymptomatic carriers in such a setting?

Dr Paul Kennedy, GP, Te Awamutu

Choosing whether to treat or not to treat a MRSA carrier depends on three factors; how successful is treatment likely to be, is the patient (or others) at risk of MRSA infection, and what is the local policy?

An individual MRSA carrier may be treated, or decolonised, with a combination of antiseptic washes and shampoos, topical antibiotic to the nostrils (mupicirin) and usually at least two oral antibiotics. This treatment reduces the amount of the original Staphylococcus aureus on the body. Following treatment recolonisation occurs either with the original strain (the MRSA) or a new strain. Clearing MRSA completely is difficult and colonisation can be very long term.

Individuals colonised with MRSA are asymptomatic. Therefore treatment is only recommended if there is a high risk of MRSA infection either for the patient or those around them. For instance treatment may be recommended by the hospital infection control team prior to elective surgery, to reduce the risk of peri-operative complications for the individual, and to reduce cross contamination between in-patients.

The risk of serious infection with MRSA is less in the community and decolonisation is not usually recommended. Instead standard infection control procedures to reduce cross colonisation are recommended for all residents, e.g. good hand hygiene and occlusive dressing of open wounds

Isolation of MRSA positive patients is not recommended. They should socialise as normal. However they should not share a bedroom if they (or their roommate) have a chronic open wound or invasive devise such as a catheter.

Should asymptomatic elderly carriers of MRSA in residential care be treated? Unless expecting surgery, probably not.

References

General Health Protection, Department of Health. Infection control guidance for care homes. Crown copyright, London 2006. Available from www.dh.gov.uk/publications (accessed April 2009)

Coia JE, Duckworth GJ, Edwards DI, et al. Guidelines for the control and prevention of meticillin-resistant Staphylococcus aureus (MRSA) in healthcare facilities. J Hosp Infect 2006;63S:1-44.

Royal College of Nursing. Methicillin-resistant Staphylococcus aureus (MRSA): guidance for nursing staff. London 2005.

CVD and Antioxidants

Dear bpac,

In your article "The Science Behind Lifestyle Risk Factors for Cardiovascular Disease" (BPJ 18), you state that "a higher intake of certain anti-oxidants has been shown to lower the incidence of heart disease". Interestingly you do not give any references to support this.

The recently published New Zealand Cardiovascular Guidelines Handbook (Page 31) contradicts this with "RCT evidence shows that vitamin supplementation with these anti-oxidant vitamins (beta carotene, vitamin C and vitamin E) does not reduce cardiovascular risk".

This topic made for lively debate in my peer review group.

How should we be advising our patients?

Dr Marion Taylor, GP, Wanganui

It is clear that there is benefit associated with greater consumption of fruits and vegetables. However, whether this benefit is due to antioxidant content remains to be determined, although there are a number of pointers in that direction.

"Up to Date" reviewed the literature on this topic again at the beginning of this year and their conclusion was as follows:

"Antioxidants have been evaluated for both primary and secondary prevention of coronary heart disease (CHD). Studies of the mechanisms of atherosclerosis suggest that antioxidants might be protective. Observational studies appeared to show benefits with higher intake of some antioxidants. Additionally, cardiovascular protection has been associated with diets high in antioxidants (from fruit and vegetables and with higher circulating levels of alpha tocopherol).

Despite this, most randomised controlled trials have not found antioxidant supplementation to be effective for the prevention of CHD. It is more difficult to assess the efficacy of dietary antioxidants in randomised trials. The association between dietary antioxidants and cardioprotection, despite the lack of benefits seen in trials of supplements, may reflect issues of confounding and bias in observational studies, or may occur because the full complement of antioxidants in foods are different from what is found in supplements or are present in more optimal ratios."

The guidelines are correct to say supplementation has not been proven to reduce CVD risk, however this is different to the statement in the bpac article that "a higher intake of certain anti-oxidants has been shown to lower the incidence of heart disease" albeit it a somewhat subtle difference.

The best advice to patients is to ensure an adequate intake of fruit and vegetables as the "shortcuts" have yet to be proven.

Reference

Tangney C, Rosenson R. Nutritional antioxidants in coronary heart disease. UpToDate 2009. Available from www.uptodate.com (Accessed May 2009).

Erratum – STI testing report

Dear Editor

In the recent STI testing report (April 2009), I found one paragraph quite confusing which made me go to the referenced article to clarify what you meant. In the report you state the following;

> "Currently, approximately only 9% of all Chlamydia tests performed in New Zealand return a positive result. A study in London was able to demonstrate that by using a risk assessment strategy based on testing those under 25 who had two or more sexual partners in the past year, they were able to increase the yield of positive results to 87%."

This seemed to be saying that by using this strategy you could get 87% of your tests positive - a remarkable number indeed but alas not so. The 87% refers to detecting 87% of the positives in the screened population. To do this they had to screen 49% of their sample of women. The actual yield rate would have been about 20/445 or about 5%.

Am I right or have I misread this?

Dr Michael Brewer, GP, Motueka

The short answer is that you are right. This paragraph has confused the number of positive results with the sensitivity of a particular screening strategy. The best way of clarifying this may be to take a closer look at the study itself (the long answer).

The study by Grun et al had three objectives:

1. *To estimate the prevalence of Chlamydia trachomatis in asymptomatic women attending general practice*
2. *To assess the potential of the ligase chain reaction as a screening tool*
3. *To evaluate selective screening criteria*

The third objective was the focus of the bpac^{nz} report because asymptomatic infection is unlikely to be detected without a screening programme. But given the relatively low prevalence of infection, it is more appropriate to consider targeted screening than universal testing.

When considering targeted screening strategies Grun et al noted that younger age and multiple partners were associated with infection. They tested possible combinations of age and number of partners to identify a strategy that detected the greatest number of infections for the least number of people tested.

The study group of 879 women aged 18–35 years were all tested and 23 Chlamydia infections were detected.

- *If only women aged 25 years or less had been screened, 17 of 23 infections (74%) would have been detected by testing approximately 35% of the study population*
- *If only women aged 29 years or less had been screened, 20 of 23 infections (87%) would have been detected by testing approximately 67% of the study population*
- *If only women aged 25 years or less **and** all women who had had two or more partners in the past year had been screened, 20 of 23 infections (87%) would have been detected by testing approximately 49% of the study population*

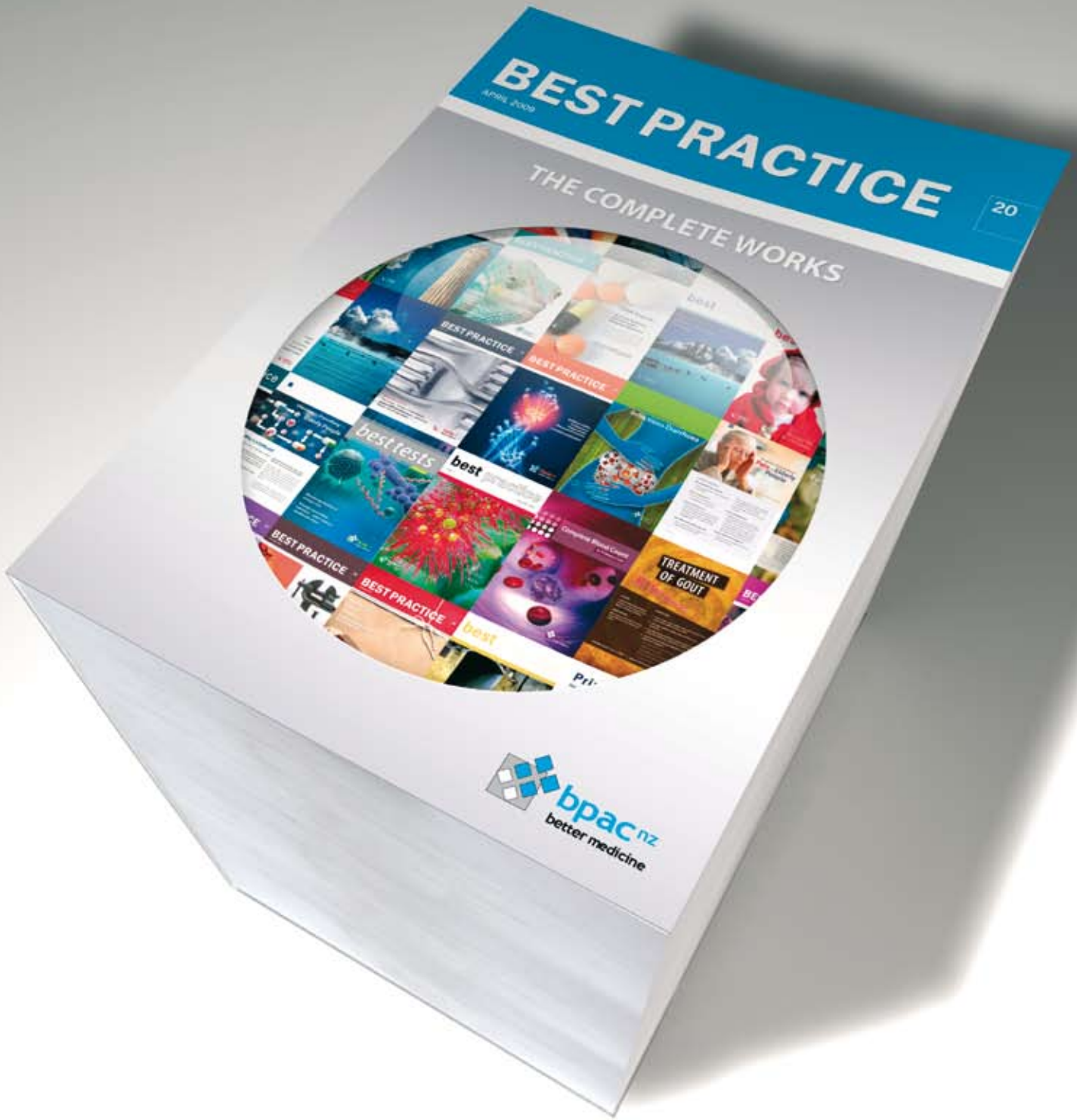
*While no targeted screening strategy detected all the cases of infection, testing women aged 25 years or less **and** all women who had had two or more partners in the past year detected the greatest number of infections for the least number of people tested.*

Reference

Grun L, Tassano-Smith J, Carder C, et al. Comparison of two methods of screening for genital chlamydial infection in women attending in general practice: cross sectional survey. BMJ 1997;315:226-230.

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

for **BEST PRACTICE** online



visit us at **www.bpac.org.nz**

Call us on **03 477 5418**
Email us at **editor@bpac.org.nz**
Freefax us on **0800 27 22 69**
www.bpac.org.nz