BEST PRACTICE

14

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Managing insomnia • Enuresis in children • Treating head lice

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Contributed by **Professor Innes Asher**, Head of Department, Paediatrics, School of Medicine, University of Auckland

Reducing inequalities in asthma care for children

As a follow up to BPJ 13, Professor Asher continues the theme of addressing disparities in Māori health.

All New Zealand children with asthma need:

- Access to appropriate medication
- An asthma management plan
- Appropriate consultation time
- Recording of household smoking status in clinical records
- Identification of who usually supervises their asthma inhaler use

For some children, there is a lot of ground to make up, and we each have a role. Māori children may need extra care to ensure they have the best chance of a good outcome. We need to examine our own cultural attitudes. I have learnt more from reading about our history,[1] modern interpretation of the Treaty,[2] staying on marae and starting to learn Te Reo. Each of these has helped to open my eyes, and my heart, and I hope that I am becoming a better doctor because of it.

In our consultations, what can practitioners do? Firstly, be well connected with our patients and recognise that we can do things better. We must ensure that Māori are well-informed about asthma and its management, let them know what is possible, and discuss what is wanted by Māori parents and whānau. We also need to be able to identify and work with different family dynamics and parenting structures. If we find out about barriers to care faced by whānau, we can give consideration to any role we may have in overcoming these barriers. Often we may need to "go the extra mile" to ensure that children can achieve the best possible health care outcomes. Referral to Māori health services may augment our care. Clinical audits that compare the care we deliver to children in each ethnic group against guideline recommendations may be helpful.

New Zealand is a great place to be a child – but only if you don't live in severe or significant hardship, as 26% of our children do.[3] Our society has become the most unequal in the OECD, and we have very high rates of preventable diseases.[4] Admissions to hospital for preventable diseases such as bronchiolitis, pneumonia, bronchiectasis, gastroenteritis and serious skin infections are higher than they used to be.[5] State policies since 1990 that have led to a reduction in incomes of households with children contribute to this increase in inequality,[6, 7] with many families having to choose between basic necessities. The issue of low income needs to be addressed, taking into account factors such as the retrofitting of uninsulated homes which would help respiratory health.[8] It is welcome that the costs of primary health care have reduced for many children, but after-hours primary care costs remain a significant barrier and can lead to delays in treatment.

In this whole picture, where is asthma? From the 1980s to 1990s asthma became more common in New Zealand, and symptoms of asthma in New Zealand children are still among the highest of any country in the world, though the prevalence has fallen in the last decade.[9, 10] This indicates that energies are better directed towards optimising treatment. The evidence-based Paediatric Society of New Zealand Guideline,[11] based on the SIGN guidelines, defines the approach we should take. While standards

of asthma care in children have improved over the last two decades, with greater use of preventers, and the availability of spacers, many of our children still receive suboptimal care.

In particular, **Māori children with asthma have more severe symptoms compared with Pacific and European New Zealanders**.[12] They are admitted to hospital almost twice as commonly as non-Māori, and have more days off school due to asthma.[13] A community-based study of asthma-related primary care for children found ethnic differences in the provision of asthma education, parental asthma knowledge and medication that suggested there were differences in the quality of care received by Māori and Pacific children compared to "Other" ethnic group children.[14] PharmHouse data ending May 2007 suggests that the greatest unmet need for inhaled corticosteroid treatment is among Pacific and Māori children, continuing earlier trends.[15] At the same time, short acting β-agonists are more commonly used in Māori and Pacific children from birth to nine years. In view of our constitutional commitment to the Treaty of Waitangi, and our legal commitment to the UN Declarations on the Rights of Children and the Rights of Indigenous Peoples, why do we have this inequality?

There are many reasons for this disparity. The experience of some Māori with the health care system discourages rather than encourages future health seeking behaviours. Māori are more likely to report having experienced discrimination in the healthcare setting.[16] Māori are over-represented among lower socioeconomic families, where the cost of accessing health care may involve choosing between other necessities such as nutritious food.[6] There may be cultural differences in communication (verbal and non-verbal) which can result in poorer care if not recognised and addressed. We also need to accept that the New Zealand health system, and those working within it, have contributed to this disparity. The important thing is that, as doctors working in this system, we can be part of the solution.

Ma tōu rourou, ma tōku rourou, Ka ora ai nga tamariki

With your small basket and my small basket we can keep the children well

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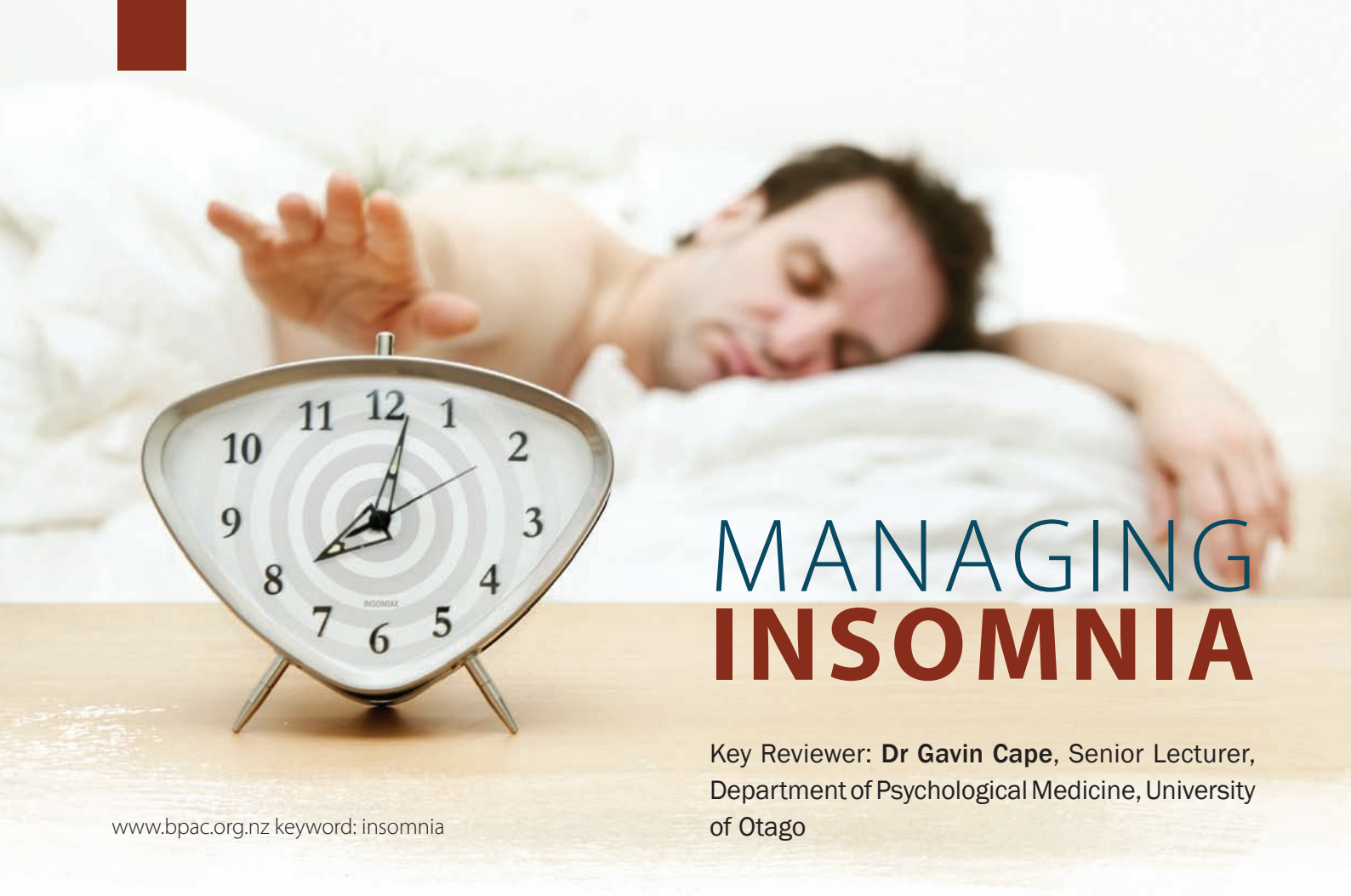

MANAGING INSOMNIA

Key Reviewer: **Dr Gavin Cape**, Senior Lecturer, Department of Psychological Medicine, University of Otago

www.bpac.org.nz keyword: insomnia

Key Concepts

- Insomnia is usually secondary to other factors such as underlying health issues or a poor sleep environment
- Best initial treatment is non-drug interventions including sleep hygiene tips (ASLEEP)
- If pharmacological therapy is required, use a short-acting benzodiazepine or zopiclone at the lowest effective dose for a short duration
- Avoid hypnotics for older people who are at increased risk of confusion and falls
- Antidepressants and antihistamines are not routinely recommended

Insomnia is common and can significantly affect wellbeing

Insomnia is defined as difficulty in falling or staying asleep, leading to impairment of daytime functioning.

Insomnia affects one in three adults intermittently and one in ten adults chronically.[1] People with insomnia frequently experience excessive daytime sleepiness, irritability and a lack of energy. Chronic insomnia may lead to psychiatric problems (e.g. depression, anxiety), problem use of drugs or alcohol, reduced quality of life and cognitive impairment in elderly people.[2]

Clinical assessment of insomnia includes a detailed history and examination

Many conditions can present with symptoms of insomnia

Only about 15 to 20 percent of patients with insomnia have no other associated diagnosis. It is usually secondary

to other factors such as underlying health issues, poor sleep environment, shift work or use of medications or other substances that interfere with sleep.[1] Box 1 lists some common causes of insomnia.

If initial evaluation of insomnia identifies an acute stressor such as grief or disruption of the sleep environment by noise, no further evaluation may be needed. A more comprehensive evaluation may be required in patients who fail to respond to initial treatment or if a co-morbid condition is present or suspected.[1,3] This may include a sleep diary, laboratory testing or referral to a sleep clinic, depending on the suspected underlying cause.

Mistaken beliefs about sleep are common

It is a common belief that people require eight hours of sleep each night. However in reality physiological changes with age and decreased activity often result in a reduced requirement for sleep with no interference with daytime functioning. For example, a 15 year old requires an average of eight hours sleep whereas many people over 70 need less than six hours sleep each night.[2]

Education about normal sleep requirements may be all that is needed to reassure a person they do not have insomnia.

Treating insomnia

The primary goal is to remove or treat any underlying problems, prevent progression from transient to chronic insomnia and to improve the patient's quality of life.

Manage insomnia with non-drug options if possible

Education about sleep hygiene and stimulus control are part of the management of everyone with insomnia regardless of whether they require further treatment with drugs or other behavioural therapies.

Box 1: Causes of Insomnia[2]

Psychological

Loss, crisis, worry, anxiety, depression, dementia, other mental health issues such as hypomania or psychotic disorders.

Physical

Movement disorders – Restless legs syndrome or periodic leg movements.

Respiratory disorders – Obstructive sleep apnoea, dyspnoea and coughing.

Painful conditions – Arthritis or headaches.

Urinary frequency – UTIs or prostatic problems.

Endocrine disorders – Hyperthyroidism (sweats), diabetes mellitus (nocturia), diabetes insipidus (nocturia).

Drugs

Ceasing medication – rebound insomnia e.g. hypnotics, antidepressants.

Alcohol – may help initiate sleep but reduces quality and causes early wakening.

Caffeine – especially in the evening e.g. coffee, tea, energy drinks, cola.

Medications – Appetite suppressants, chronic benzodiazepine use, some antidepressants (mostly SSRIs), thyroid hormones, sympathomimetics (agitation), diuretics (nocturia), corticosteroids (agitation) and beta-blockers (bad dreams).

Illicit drugs – e.g. amphetamines, "ecstasy", BZP, drug withdrawal states.

ASLEEP is a useful acronym for remembering sleep hygiene tips

Alcohol, caffeine and nicotine should be avoided

Sleep and sex should be the only uses of the bed

Leave laptops, TV and paperwork out of the bedroom

Exercise regularly but not within two to three hours of bedtime

Early rising – avoid sleeping-in or daytime naps

Plan for bedtime – establish a bedtime routine such as having a warm drink or a bath.[1,4]

There are many different opinions about the effect of reading books in bed. A trial of not reading in bed might be useful.

Other behavioural interventions attempt to alter mistaken beliefs and attitudes about sleep, reduce autonomic arousal, and change maladaptive sleep habits that may contribute to maintaining insomnia. Some examples are sleep restriction, relaxation techniques and cognitive behavioural therapy (Box 2). Sleep restriction and relaxation techniques can normally be initiated in primary care however cognitive therapy usually requires referral to a psychologist.[2,4]

In clinical practice, these methods can be initiated according to the most important perpetuating factors for insomnia. For example, sleep restriction may be more suitable for those patients who have adapted to insomnia by spending excessive amounts of time in bed. Stimulus control for those who have engaged in sleep incompatible activities and relaxation techniques may be suitable for people with tension and anxiety.[4]

Pharmacological treatments

When other approaches prove inadequate, prescription drug therapy may be required. Although drug therapy is effective in the short term there is limited evidence of its effect long term and significant concern exists about dependence, tolerance and difficulty withdrawing people after long term continuous use.

Concomitant use of hypnotics with behaviour therapy may reduce the efficacy of the behaviour therapy.[1,4]

It may be appropriate to prescribe a short course of hypnotics for someone with a brief history of insomnia that is expected to resolve quickly (e.g. jet-lag, short term stress). Caution is required for someone who has a brief

Box 2: Types of non-pharmacological interventions[3,5]

Stimulus-control therapy

- Avoid bright lights (including television), noise and temperature extremes, large meals, caffeine, tobacco and alcohol at night.
- Minimise evening fluid intake, leave the bedroom if unable to fall asleep within 20 minutes, limit use of the bedroom to sleep and intimacy.

Sleep restriction

- Reduce time in bed to estimated total sleep time determined by a sleep diary (minimum five hours).
- Increase time in bed by 15 minutes every week when ratio of time asleep to time in bed is at least 90 percent.

Relaxation therapy

- Tensing and relaxing different muscle groups, meditation, hypnosis, biofeedback or imagery.

Cognitive therapy

- Education to alter false beliefs and attitudes about sleep.

history of insomnia that is likely to persist (e.g. stress that is likely to be long term). Hypnotics are best avoided for someone with a history of chronic insomnia because the risks of long term use are high. Behavioural therapies are more durable and safer long term.[6]

Short acting benzodiazepines and zopiclone are the drugs of choice when pharmacological therapies are required. Antihistamines and antidepressants are less suitable for insomnia.

When drug treatment is required short-acting benzodiazepines or zopiclone are recommended

Short-acting benzodiazepines

Benzodiazepines potentiate the inhibitory effects of gamma-aminobutyric acid (GABA) throughout the central nervous system, decreasing time taken to fall asleep and increasing sleep duration. Short acting benzodiazepines, such as temazepam, are more suitable for the treatment of insomnia because they act for a shorter time, have no active metabolites and little or no hangover effect.

Longer acting benzodiazepines, such as diazepam and nitrazepam, are not usually recommended because they have a more prolonged action and may cause residual effects the following day.[7]

Adverse effects associated with benzodiazepine use include drowsiness and light headedness the next day, psychomotor impairment and amnesia.

Although benzodiazepines are effective, their potential for tolerance and dependence limit their use to short-term insomnia. It has been estimated that 10 to 30% of chronic benzodiazepine users are dependent on them and 50% of all users suffer withdrawal symptoms. Dependency is more likely with long term use, higher doses, higher potency benzodiazepines, or in people with psychiatric illness or a history of drug or alcohol abuse.

It is recommended that the use of benzodiazepines for insomnia is restricted to the treatment of severe short term insomnia and treatment should be at the lowest effective dose for the shortest possible time (less than four weeks and preferably five to ten days).[8]

Zopiclone

Zopiclone, a non-benzodiazepine hypnotic, is a selective GABA agonist that was developed with the aim of overcoming some of the disadvantages of benzodiazepines, such as next day sedation, dependence and withdrawal. However there is limited evidence of a clinically useful difference between zopiclone and the shorter acting benzodiazepine hypnotics in terms of effectiveness, adverse effects or

Prescribing points for hypnotics [1,3,9]

- Identify and address any conditions or circumstances contributing to insomnia.
- Provide advice about non-drug therapies, for example good sleeping habits.
- Prescribe the lowest effective dose of a short acting hypnotic.
- Prescribe hypnotics intermittently and for short durations (less than four weeks but preferably five to ten days).
- Avoid hypnotics or use with caution for patients with a history of substance abuse, myasthenia gravis, respiratory impairment or acute cerebrovascular accident.
- Review for side effects – in particular, daytime sleepiness.
- Before prescribing for older patients, give advice about the increased risk of use and enquire about difficulties with balance which may indicate an increased susceptibility to falls.

potential for dependence or problem use. Zopiclone has been shown to cause hangover effects and impair psychomotor performance in a similar way to temazepam.[9] Dependence has also been reported in a small number of people.[7] An adverse effect commonly reported with zopiclone is a bitter or metallic taste in the mouth.

> Zopiclone should be treated with the same caution as benzodiazepines – use for severe short term insomnia at the lowest effective dose for the shortest possible time (less than four weeks and preferably five to ten days).

Hypnotic use in older people

Caution is required when hypnotics are used to treat insomnia in older people because they increase the risk of falls, fractures and car accidents, and also impair cognition, slowing reaction times and decreasing energy.

An analysis of hypnotics in older people found that improvements in sleep were statistically significant but the magnitude of the clinical effect was small. The increased risk of adverse events was both statistically and clinically significant in older people already at risk of falls and cognitive impairment. In older people, the benefits of these drugs may not justify the increased risk, particularly in those patients with additional risk factors for cognitive or psychomotor adverse events.[10]

> Hypnotics are best avoided in elderly people who are at increased risk of falls or confusion (ideally avoid in all elderly people). Increasing age, previous history of falls or confusion and concomitant medicines should be considered when assessing risk in a particular patient.[2]

Withdrawing people from long term hypnotics

Many people take hypnotics on a continuous basis, however this should be avoided because of tolerance to effects, dependence and an increased risk of adverse events.

Where appropriate, patients should be encouraged to gradually withdraw. Slowly tapering the dose over a number of months may help to reduce the withdrawal effects such as agitation, anxiety and insomnia.[2]

Some successful strategies that have been used to initiate withdrawal and reduce benzodiazepine use include: [9]

- Letters sent by GPs to long-term users explaining possible problems and inviting patients to gradually reduce their use under supervision. After six months, benzodiazepine use was reduced by one third.
- Review of patients' prescriptions by GPs at regular consultations. Over eight months one in six patients stopped using benzodiazepines.
- Review of older people's medication regimens by pharmacists. This reduced adverse events and reduced the use of sedatives and hypnotics by up to 20%.

Antidepressants are not recommended for insomnia in the absence of depression

Antidepressants are no more effective than short acting benzodiazepines and zopiclone for treating insomnia and their side effect profile, which includes cardiac dysrhythmia and orthostatic hypotension, is more severe.[1]

Antidepressants, like hypnotics, increase the risk of falls in elderly people.[2] They appear to have less potential for abuse than hypnotics which is an advantage in people who have a history of drug or alcohol abuse.[1]

SSRIs can exacerbate insomnia so when used for depression they are taken in the morning.

Antihistamines are not recommended for insomnia

Antihistamines have limited evidence of effectiveness for insomnia. Morning hangover effects may be greater than those of short acting benzodiazepines and zopiclone and they may induce significant anticholinergic effects.[1]

Alternative remedies are not routinely recommended for insomnia

The efficacy and safety of agents such as valerian, kava or St John's wort for insomnia is not clear and has not been well studied.

Melatonin may be useful for short-term adaption to jet lag or other circadian rhythm sleep disorders. Effectiveness for chronic insomnia is less clear and optimal dose and long term adverse effects are unknown.[1,11]

Summary

Insomnia is often secondary to other causes. It is essential to address these causes wherever possible before initiating pharmacological therapy. Initial treatment of insomnia involves behavioural therapies to improve sleeping habits and environment, improve relaxation and address false beliefs about sleep.

If drug therapy is needed, short-acting benzodiazepines or zopiclone are preferable. Short courses at the lowest effective dose are recommended. Hypnotics are best avoided in older people at risk of falls or confusion.

Antihistamines have limited evidence of effectiveness for insomnia and may cause significant adverse effects. Antidepressants are not recommended for insomnia in the absence of depression.

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Clozapine: A reminder about safe and effective use

CLOZAPINE IS THE DRUG OF CHOICE for treatment resistant schizophrenia. It is one of the few drugs in New Zealand that can not be prescribed by GPs but they still need to be aware of potential problems associated with its use.

Clozapine use is closely monitored including weekly blood tests (white blood cell count and absolute neutrophil count) for the first 18 weeks of treatment, then four weekly thereafter throughout treatment and for four weeks after discontinuation.

Guidance has previously been provided about significant adverse effects and interactions with clozapine. In this article, we re-emphasise the vital role that primary care has in recognising and managing these issues.

Best practice tip: A Christchurch GP has set up a pop-up alert on the medical record of his one patient receiving clozapine. It states: "On clozapine, watch for constipation, neutropenia and myocarditis". Erythromycin, co-trimoxazole, trimethoprim and nitrofurantoin are entered under medical warnings with a note saying "Avoid – interaction with clozapine". **See Page 38 for a ten minute tutorial on setting up a pop-up alert for patients on clozapine.**

Significant adverse effects

Adverse effects associated with clozapine include constipation, blood dyscrasias, myocarditis and metabolic syndrome.

Constipation

Constipation is a common adverse effect of clozapine and is related to its anticholinergic effects. It can be potentially serious; four deaths resulting from complications of severe constipation have been reported in New Zealand.[1]

What can GPs do?

- Recognise and treat constipation in patients receiving clozapine to prevent the development of more serious complications such as obstruction and paralytic ileus.
- Avoid concomitant anticholinergic drugs (e.g. amitriptyline) as constipation is more likely to occur.
- Encourage patients to adopt measures which may prevent constipation such as a high fibre diet (at least three serves of vegetables, two serves of fruit and some cereal, bread, rice or pasta every day or more than 30g of fibre per day), adequate fluid intake (1.5 – 2 litres of fluid per day) and physical activity.

Blood dyscrasias

Clozapine can cause potentially fatal neutropenia and agranulocytosis (number needed to harm = 59).[2]

What can GPs do?

- **Patients who present with evidence of infection such as flu-like symptoms, sore throat or fever must have a full blood count done immediately to rule out neutropenia or agranulocytosis. It must be indicated on the laboratory form that the patient is on clozapine and it must be ensured that results are provided on the same day. Depending on the result, urgent haematology referral or emergency hospital admission may be required.** Recently a patient died from agranulocytosis seconday to clozapine. The laboratory was not aware that the patient was on clozapine and the sample was also clotted. This lead to a significant delay in diagnosing the agranulocytosis.
- Avoid concomitant use of antibiotics that increase the risk of neutropenia. This includes those that are known to have a substantial potential to depress bone marrow function for example, sulphonamides, trimethoprim, co-trimoxazole and nitrofurantoin, or those that increase the plasma concentration of clozapine such as erythromycin.

Myocarditis

Fatal myocarditis and cardiomyopathy have been reported rarely with clozapine use.

What can GPs do?[3]

- Check patients, who have persistent tachycardia at rest, for other symptoms of myocarditis or cardiomyopathy. These include palpitations, arrhythmias, symptoms mimicking myocardial infarction, chest pain or other symptoms of heart failure.
- If myocarditis or cardiomyopathy is suspected, refer to the prescribing psychiatrist who may stop the patient's clozapine treatment. The patient should also be evaluated by a cardiologist.

Metabolic syndrome

Weight gain, hyperglycaemia and dyslipidaemia are all associated with clozapine.

What can GPs do?

- Give advice about diet and exercise. It may also be appropriate to use pharmacological management.
- Monitor relevant parameters such as lipid profile, fasting glucose, blood pressure and BMI.

Important drug interactions

Smoking

People who smoke metabolise clozapine faster than those who do not smoke. When a person stops smoking, the resulting increase in plasma levels can cause or worsen adverse effects. Smoking cessation should always be planned with the clinical team so that this effect can be monitored and managed.

Other drug interactions

Drugs that may increase the plasma concentration of clozapine include SSRIs, cimetidine, caffeine, lamotrigine, risperidone and combined oral contraceptives (interactions may only be supported by isolated case reports). Drugs that may decrease clozapine levels include rifampicin and anticonvulsants such as carbamazepine and phenytoin.

See BPJ 4, April 2007, "Clozapine: Safe and effective use".

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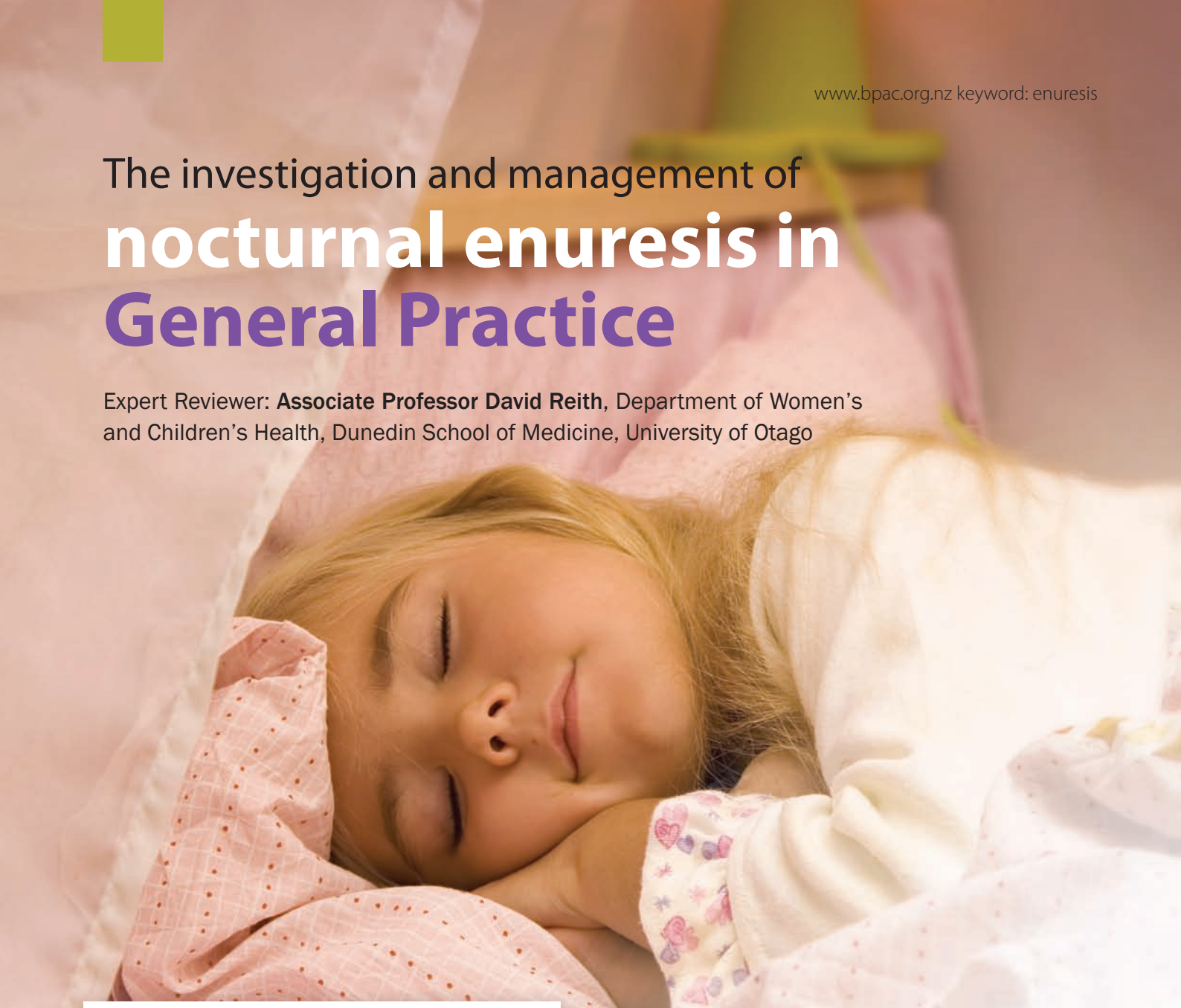
The investigation and management of nocturnal enuresis in General Practice

Expert Reviewer: **Associate Professor David Reith**, Department of Women's and Children's Health, Dunedin School of Medicine, University of Otago



Key Concepts

- Nocturnal enuresis is common and children usually grow out of it
- Simple advice regarding fluids and use of rewards may be appropriate in the first instance
- If the child and their family are motivated to try treatment, then the use of bed alarms with support offer the best chance of long-term success
- Desmopressin can be prescribed with caution for occasional short term use

Considerations:

- At what age is bedwetting abnormal?
- What is it normally due to?
- Are any investigations needed?
- What advice can I give to parents?
- When should I refer?
- What non-drug treatments are recommended?
- What medications are recommended?

Defining bedwetting

Primary nocturnal enuresis is bedwetting in a child who has never been consistently dry at nights for a period of six months.

Secondary nocturnal enuresis is bedwetting in a child who has previously had a period of at least six months of dryness.

Bedwetting can place considerable stress on the individuals affected and their families. Although this article is aimed mainly at children, similar principles apply to adolescents and adults who are still bedwetting.

At what age is bedwetting abnormal?

The International Children's Continence Society defines nocturnal enuresis as:[1]

- A child five to six years old with two or more bedwetting episodes per month
- A child over six years old with one or more bedwetting episodes per month

However, most management strategies are aimed at children aged seven years or older, as this is when bedwetting is usually considered to be a problem by both the child and their family.

Bedwetting is common but reduces with age. It affects approximately:

- 15% of 5 year olds
- 5% of 10 year olds
- 2% of 15 year olds
- 1% of adults

Spontaneous remission occurs in about 15% of affected children each year and is more likely to occur if there is a family history of nocturnal enuresis.[2]

A recent study found that children with the most frequent bedwetting were more likely to persist with the problem.[3]

It is thought that fewer than half of parents with a child with nocturnal enuresis, consult their doctor about the problem.[4]

Causes of bedwetting

The exact cause of nocturnal enuresis is unknown. It appears to be a neurodevelopmental problem which is probably multifactorial. Discussion with patients and parents may centre around the following:

- Sleep polyuria
- Reduced night-time bladder capacity
- Lack of arousal from sleep
- Psychosocial factors
- Genetics

Sleep polyuria

Nocturnal polyuria can result from a deranged circadian rhythm of antidiuretic hormone (ADH) secretion which occurs in approximately 70% of children with bedwetting.[5] ADH, also known as vasopressin, is a peptide secreted from the posterior pituitary and plays a key role in the control of urine production. Usually ADH secretion increases during the night to concentrate the urine and this in turn helps to produce low volumes of urine.

Reduced night-time bladder capacity

A recent Chinese study included ultrasound examination of 500 children with nocturnal enuresis and showed a reduced functional bladder capacity in approximately 40% of children with nocturnal enuresis.[6]

Lack of arousal from sleep

Sleep and arousal is one of the least understood factors in the pathophysiology of enuresis. Many parents will comment that their child with bedwetting is a "deep sleeper". A 1999 study using EEG analysis suggested that both deeper sleep and impaired arousal is more common in children with enuresis,[7] however other studies have conflicting results.

Psychosocial factors

Psychological problems are rarely the cause of primary nocturnal enuresis but teasing, bullying or punishment can be the result of it. Secondary nocturnal enuresis is more likely to be due to a psychosocial stressor such as parental separation, a new baby in the family, sickness or problems at school.

Genetics

Genetic factors are strongly implicated in the etiology of primary nocturnal enuresis, so it is worthwhile taking a family history of bedwetting.

Approximately 70% of children with bedwetting have a sibling or parent who was late in becoming dry. Children with one parent who had enuresis have a 44% risk of nocturnal enuresis and those with two affected parents have a 77% risk.[4]

Most inherited nocturnal enuresis exhibits an autosomal dominant mode of transmission with high penetrance (90%). However, a third of all cases are sporadic, and the difference between sporadic and familial forms is not known.[8]

Differential diagnosis –what else might it be?

When a child presents with bedwetting, enquire about the presence of daytime symptoms, which could indicate that the bedwetting is secondary to other causes.

- UTI and other acute illness might cause short periods of bedwetting in someone who has previously been dry.
- Diabetes mellitus, diabetes insipidus or renal failure may cause bedwetting but there are usually other symptoms e.g. daytime polyuria, excessive thirst.
- Chronic constipation may result in bladder instability, a careful history of bowel pattern is required.
- Bladder instability can cause daytime and night-time incontinence.
- Caffeinated drinks may irritate the bladder.

Investigation of bedwetting

A careful history is important

- Distinguish between children with nocturnal enuresis (the majority) and children who also have episodes of enuresis during the daytime.
- Distinguish between primary and secondary nocturnal enuresis.
- Ask about the pattern of voiding, the number of dry nights in the past week or month, fluid intake at bedtime, intake of caffeine at bedtime (e.g. tea, coffee, cola, chocolate).
- Discuss practical issues such as can the child reach the toilet, do they need a light on to see their way to the toilet, any night time fears.
- Ask about any possible stressors at home, school or with friends.

- Discuss what has been tried already, including punishments and rewards.
- Elicit previous medical history, such as previous UTIs.

The examination of the abdomen, perineum, spine and nervous system is normal in a child with nocturnal enuresis. Any abnormalities found would lead to additional investigation.

Ultrasound examination of the kidneys and urinary tract to exclude anatomical abnormalities is only recommended in children who are wet during the day, after UTI or when nocturnal enuresis is unresponsive to treatment.[9]

Investigation with urine dipstick and culture can be helpful.[4] However, checking specific gravity is usually not.[10]

Treatment options for bedwetting

Waiting

Most children will outgrow bedwetting. For this reason most treatments are delayed until the child is at least seven years old. However treatment might begin earlier if the situation is perceived to be damaging the child's self esteem or relationships with family and friends.

Behavioural strategies

Parents could be advised to:

- Ensure that the child empties their bladder well at bedtime.
- Improve the child's access to the toilet (e.g. have them sleep on the bottom bunk, have a torch within reach).
- Use waterproof covers on mattress and duvet (especially for boys) and then absorbent layers over the mattress cover.
- Shower/bathe the child in the morning prior to attending school to remove odour.
- Do not restrict fluids. The child should have about eight drinks per day, spaced out throughout the day, the last one about an hour before bed. Avoid caffeine in night-time food and drink (e.g. tea, coffee, cola, chocolate).[4]
- Treat constipation if present.
- Reward systems.[11] Advise use of positive reinforcement to encourage a desired behaviour. The aim is to positively reinforce dry nights (or any steps towards that) and to reduce the negative emphasis on wet beds.
- Scheduled wakening is preferable to "lifting" a child. Scheduled wakening involves waking the child periodically (one to three times) at night and walking them to the toilet to pass urine. Eventually the time between awakenings is stretched until the child can go a full night without wetting the

bed. Lifting is thought to be counterproductive in some children as the child is denied the opportunity to learn the sensation of a full bladder and is encouraged to urinate without wakening.[4]

- Older individuals may use an alarm clock to wake themselves before their usual time of enuresis.[12]

When should GPs refer?

If after initial advice, more active treatment is sought, then referral to a paediatrician, enuresis clinic (if available in your area) or a continence advisor might be the next step to working out a programme most suited to the child. The programme would usually centre on the use of bed alarms.

Supported bed alarm programmes

Enuresis alarms emit a loud tone when moisture is sensed, so that the child is awoken as soon as they begin to wet the bed. **They are considered a good long-term and safe treatment.**

Bed alarms have a 65 to 80% success rate when used with support (such as an enuresis nurse) and if the child is motivated to become dry.[4] They help "condition" the child to wake at the sensation of a full bladder. Efficacy is better than behavioural treatments alone and relapse rate is lower than with pharmacological treatments.[4]

Alarms are usually needed for three to five months. When dryness has been achieved for 14 nights, children should be encouraged to drink extra fluid (up to 500mL of water in the hour prior to bedtime), and continue with this until there have been another seven to 14 consecutive dry nights. This form of challenge is used in conjunction with the bed alarm and is known as "overlearning". This reduces the rate of relapse from 50% to 25%.[4]

Children who relapse should be promptly offered the supported alarm programme again.

Desmopressin

Desmopressin is a synthetic analogue of ADH and is the only available antidiuretic drug. It works by reducing the volume of urine produced during the night but only on the nights it is used, **so does not cure the problem in the long term**.

In most situations, before considering this medication, it would be appropriate to have tried a bed alarm programme.

Safety concerns about desmopressin

In April 2007 the UK Medicines and Healthcare Products Regulatory Agency (MHRA) issued a drug safety alert stating that hyponatraemia, water intoxication and convulsions were associated with the use of desmopressin nasal spray. Following this, the nocturnal enuresis indication has been withdrawn from desmopressin nasal spray in the UK.

In December 2007 US drug regulators, the FDA, stated that they no longer approved desmopressin nasal spray for use in nocturnal enuresis after two deaths and a review of data that showed that 41% of hyponatraemic-related seizures occurred in people younger than 17 years old, using desmopressin most commonly for primary nocturnal enuresis.

The BNF 2008 states: "The Committee on Safety of Medicines has advised that patients should stop taking desmopressin during an episode of vomiting or diarrhoea (until fluid balance normal). The risk of hyponatraemic convulsions can also be minimised by keeping to the recommended starting dose and by avoiding concomitant use of drugs which increase secretion of vasopressin e.g tricyclic antidepressants".[13]

Occasional short term use of desmopressin

Desmopressin intranasal spray is currently available fully funded on specialist recommendation. The tablets are not currently funded. GPs might be asked to consider prescribing desmopressin for short-term use such as for

school camps or sleepovers. Desmopressin can also be offered as an adjunct to alarm therapy if required to assist family coping.[4]

A Cochrane review of desmopressin concluded that it was effective in reducing bedwetting compared with placebo. When desmopressin is used, most of the children have fewer wet nights (one night less on average per week) and more become dry (19% compared with 2% using placebo treatment in five trials involving 288 children).[14]

The usual dose of desmopressin is 20 to 40 micrograms intranasally or 200 to 400 micrograms orally, at bedtime. Fluid intake should be restricted one hour before to at least eight hours after the dose, and patients or parents should be told to report symptoms of water retention and hyponatraemia e.g. headache, nausea, vomiting, weight gain or convulsions.

There is insufficient data to reliably assess whether a higher dose is any more effective than a lower dose, so to minimise side effects and costs, the lowest effective dose should be used.[4]

Other drug options?

Oxybutynin can be useful in daytime enuresis and may also improve nocturnal enuresis. It can be considered in patients with bladder instability or in children who do not respond to desmopressin.

Tricyclic antidepressants are contraindicated for use in children for nocturnal enuresis. Tricyclic antidepressants, most commonly imipramine, have historically been used for the treatment of nocturnal enuresis and have evidence of effectiveness but with safety concerns.[15] A particular concern is overdose, which can be fatal.

Indomethacin, diclofenac and diazepam are not recommended as initial therapy for children with nocturnal enuresis.

Useful resources

KEEA – Kiwi Enuresis Encopresis Association

www.keea.org.nz

KEEA was registered as a charity in New Zealand in 2001 and helps with information and advice on bedwetting and soiling. They have a useful database which shows who to contact in your area for a bed alarm, what costs may be involved, the waiting list length and whether a GP referral is necessary.

Kidshealth

www.kidshealth.org.nz

This website covers a range of information on child and youth health – use 'bedwetting' as a search term.

NZCA (The New-Zealand Continence Association)

www.continence.org.nz

The NZCA has a children's continence section on it's website.

Patient information leaflets are also available – Incontinence in children, and Adults and bedwetting.
Email: jan@continence.org.nz
or call free 0800 650 659

Parent to Parent

www.parent2parent.org.nz

This is a support service for parents of children with a range of conditions and can put parents in touch with other parents experiencing similar situations.

Paediatric Society of New Zealand

www.paediatrics.org.nz

The society has published a best practice evidence-based guideline.

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Treating head lice

Key Reviewer: **Victoria Bryant**, Charge Nurse/ Manager, Otago Public Health Nursing Service

What are head lice?

Head lice (kutu, cooties, nits) are small parasitic insects uniquely adapted to living on human scalp and neck hair. They do not discriminate between young or old, male or female, clean, dirty, long or short hair.[1] Head lice are mainly transferred by direct head to head contact - walking from one head to another when heads are touching. They cannot fly, jump, hop or swim.

Less than half of head lice infestations cause itching and some cause no symptoms at all.[2] Scratching can result in scalp infections.

The greatest harm associated with head lice results from the well intentioned but misguided use of caustic or toxic substances to eliminate them e.g. kerosene, fly spray, veterinary products.[3]

Key Concepts

- Detection combing is the most effective way of finding head lice and is also a good way of treating head lice without using chemicals
- Treatment with insecticide should only be considered if a live louse is discovered in the hair
- A combination of insecticide based treatment and detection combing may be the most successful way to get rid of headlice
- Repeat treatment with insecticide is required after seven to ten days
- There is no published evidence on the effectiveness of herbal and alternative treatments

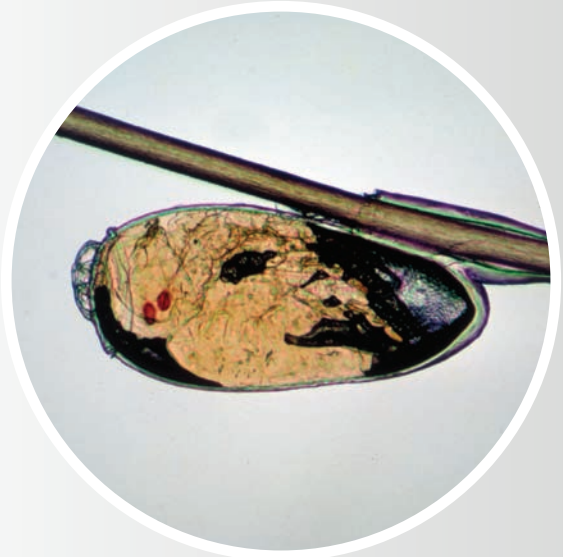
Diagnosing head lice

Accurate diagnosis can only be made if live lice are found. This can be difficult as they move quickly through the hair away from disturbances. Eggs alone are not a sign of active infestation. It is difficult to determine if eggs are alive or dead, although eggs found greater than 1cm from the scalp are either dead or empty cases.

Detection combing (see side bar over page) is the most effective way of finding head lice. Although time consuming to do properly (30 to 90 minutes), it is also a good way of treating head lice without using chemicals.

Detection Combing (bug busting, wet combing)[1, 2]

- Completely cover dry hair from root to tip with a layer of conditioner (this stuns lice for 20 minutes).
- Use an ordinary comb to detangle the hair and ensure the conditioner covers all parts of the hair.
- Comb thoroughly with a fine tooth lice comb.
- Wipe the conditioner off the comb onto a paper tissue after each stroke, checking for lice and eggs.
- Repeat combing for every part of the head.
- Examine the comb for lice and eggs.
- If you find head lice, continue combing the whole head until all of the conditioner is gone and no more head lice appear in the comb.
- Repeat the conditioner and combing at least every two or three days until no head lice are found for ten days. This may take three to four weeks.



Treatment and management of head lice

Live lice are found:
Detection combing by itself can be an effective treatment if performed properly every two to three days. This avoids the need for insecticide treatment.

Insecticide treatment is also an option when live head lice are discovered.[2,4] There are no products currently available that kill all of the eggs so a repeat treatment after seven to ten days is necessary to kill the lice that may have hatched from eggs that survived the initial treatment. Check for lice after treatment by combing.

Eggs found, not previously treated:
If the infestation has not previously been treated and eggs are found within 1cm of the scalp and no live lice are seen, detection combing or insecticide treatment can be considered.

If only eggs greater than 1cm from the scalp are found, there is no indication for insecticide treatment, but continued detection combing is advisable to check for live lice.

Eggs found, previously treated:
If the infestation has previously been treated and only eggs are found, detection combing is advisable to check for live lice.

Treatments

Malathion and pyrethroids (permethrin and phenothrin) are effective against head lice.

In general, products need to be applied twice, seven days apart, to kill lice emerging from any eggs that survive the first application.

It is important that product instructions are carefully followed. If it appears that head lice are resistant to the

insecticide it may be because the product has been used incorrectly. Check the effectiveness of the insecticide by detection combing – the lice will be dead within 20 minutes if the product is effective. If live lice are found after the product has been used correctly then a product from a different active group should be used. For example, if a course of permethrin or phenothrin has failed then malathion should be tried.

Detection combing by itself can be an effective treatment if performed properly every two to three days. This avoids the need for insecticide treatment.

Most of the currently available products can cause skin irritation and stinging. They should not be used in infants less than six months of age. Malathion is considered safe but all products should be used with caution in pregnancy and lactation.

Organophosphates

- Malathion (Derbac M* , Malathion Lotion, A-Lices Shampoo*)
- Maldison (Prioderm)

Pyrethroids

- Permethrin (available as a combination product with Malathion - Para Plus spray).
- Phenothrin (Full Marks mousse, Parasidose Extra Strength Lice Shampoo)

* Fully funded

Occlusive dressings (e.g. gladwrap, bathing caps and plastic bags) should not be used as there is a significant risk of the insecticide being absorbed into the scalp (e.g. malathion which can cause organophosphate poisoning). There is also a risk of suffocation.

There is limited evidence of clinical efficacy of herbal treatments and electric combs.

Natural products should be used with caution as they may cause toxicity with excess use. Electric combs cannot be used in children aged less than three years, or with epilepsy, heart disease, a pacemaker or other neurostimulator or if their scalp is broken. In addition, electric combs should not be used on wet hair.[5]

Head lice products which claim to repel head lice may cause more harm to children than head lice and give a false sense of security.[1]

Preventing the spread

Parents should:

- Inform the school, kindergarten, preschool, kohanga reo, friends and family about any outbreak.
- Check the whole family's hair once a week (daily when there is an outbreak in the community).
- Tying back long hair may help minimise contact with other children's hair and possible reinfestation.
- Avoid sharing of hats, hair brushes or combs.

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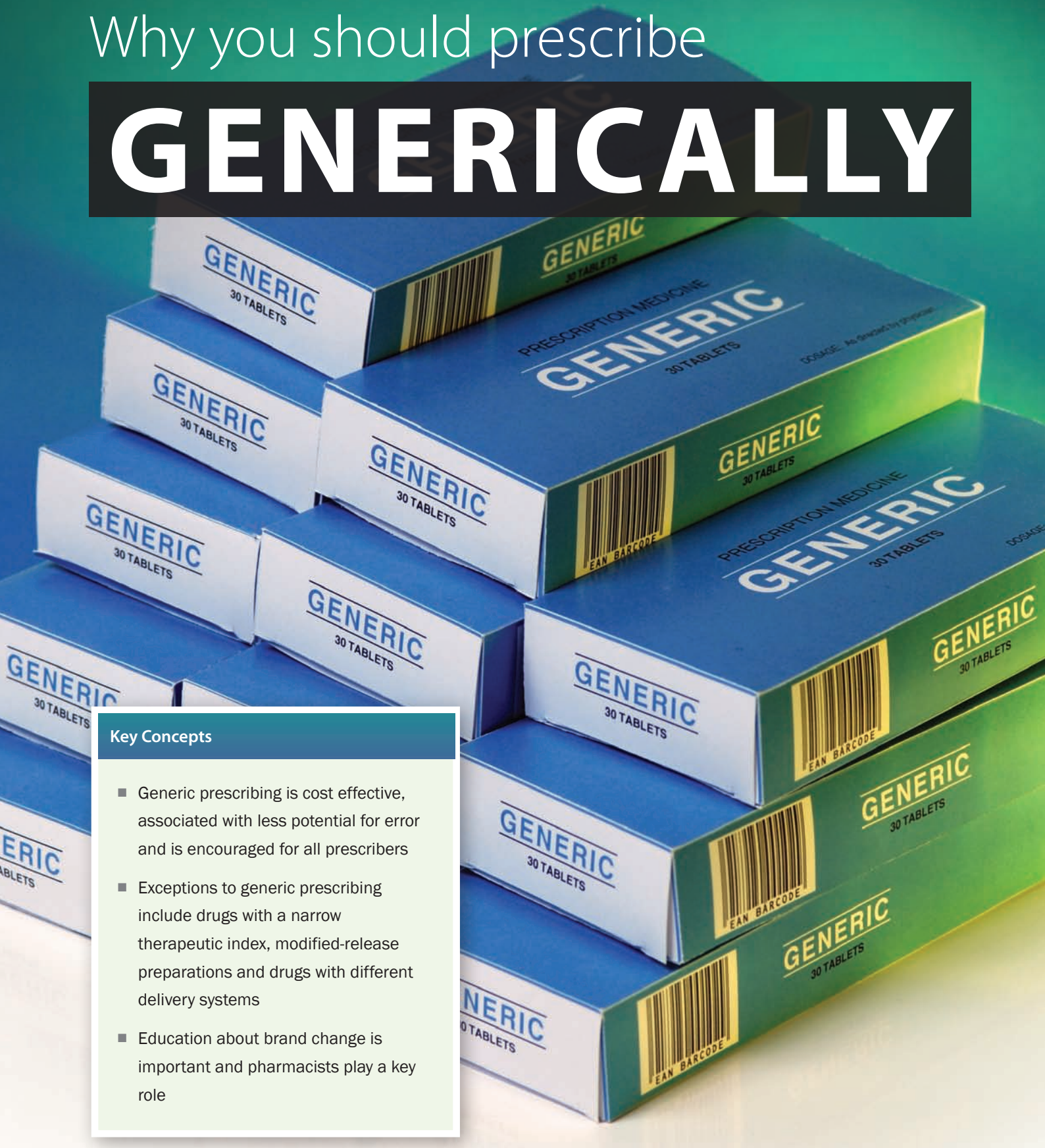
Why you should prescribe GENERICALLY



Key Concepts

- Generic prescribing is cost effective, associated with less potential for error and is encouraged for all prescribers
- Exceptions to generic prescribing include drugs with a narrow therapeutic index, modified-release preparations and drugs with different delivery systems
- Education about brand change is important and pharmacists play a key role

Prescribing a drug generically is an indicator of good prescribing practice

Generic prescribing is promoted in many countries as a way of reducing pharmaceutical costs. In the UK the adoption of generic prescribing is used as a measure to assess performance in primary care[1] and some practices have achieved a generic prescribing rate of 80%.

Prescribers in New Zealand are encouraged to write prescriptions for a medicine, using the generic name of its active ingredient rather than its brand name. While there are exceptions to this rule (see below) and some issues to take into consideration, generic prescribing can be regarded as rational and cost-effective use of medicines.

Six reasons to prescribe medicines using generic rather than brand name

1. You don't have to remember different brand names
2. You don't have to remember which brand is currently subsided
3. There is less potential for confusion and error, especially when brand names are similar
4. Less expensive medication brands can be used more often, allowing other medicines to be funded
5. Pharmacists can dispense the medication in stock without having to consult the prescriber
6. The generic name provides a guide to the drug's pharmacology and chemical class

✓	✗
Omeprazole 20 mg	Losec 20 mg, Omezol 20mg, Dr Reddy's Omeprazole 20 mg

Not all drugs should be prescribed generically

Examples of medicines which should **not** be prescribed generically include:

All anticonvulsants, all antiarrhythmics, theophylline, warfarin, cyclosporin, thyroxine and lithium.*

Some medicines are generally not considered interchangeable when:

The product has a narrow therapeutic range. In medicines where efficacy and/or toxicity are critically dependent on plasma concentration, the allowable differences in bioavailability between the reference and generic product in bioequivalence testing may result in changes in clinical effect between brands, although this is unlikely.

The product is modified release. The composition and pharmacokinetics of modified release medicines are more difficult to standardise compared to standard release formulations. For this reason, substitution of a modified release product may not be advisable once treatment has been initiated.

The delivery systems or dose forms of the products are not pharmaceutically equivalent. The use of transdermal patches (e.g. oestradiol patches), suppositories and systemically acting creams or ointments may be supported by data demonstrating bioequivalence with oral or other dose forms. However because of variability due to pharmaceutical form, these products are not considered interchangeable.

* Lithium tablets and capsules are also not equivalent

Medsafe determines bioequivalence for generic drugs

In New Zealand, Medsafe is responsible for determining that a generic copy of an innovator drug is bioequivalent, before it is released onto the market. Bioequivalence is determined using international regulations and guidelines. It is defined as the absence of a significant difference in the rate and extent of absorption into systemic circulation of two pharmaceutically equivalent medicines, when administered in the same dose under similar conditions.[2]

See BPJ Special Edition March 2007 "What is bioequivalence" for further information on the calculation of bioequivalence and variables in bioequivalence studies.

Patient perception may influence therapeutic effect

Generic prescribing may lead to a patient receiving a different brand of drug than previously used which in turn may give rise to concerns about new side effects and variation in therapeutic effect. Different brands of an equivalent medicine must include identical amounts of the active ingredient in the same dose formulation and route of administration but some excipients (inactive ingredients) are allowed to differ.[2] Some people may have individual sensitivities to excipients, for example lactose intolerance or avoidance of excipients of animal origin.

Worsening in symptom control or intensity of effect may be perceived as therapeutic inequivalence but actually may be due to disease progression[3] or psychological factors. These factors may include patient preference and perceived inferiority of a generic brand and may be more significant when the drug in question is used to treat a serious medical condition or psychological illness.[4]

See BPJ Special Edition, March 2007 "Changing to a generic drug" and BPJ 6, June 2007 "Upfront: Brand change" for further information on patient perceptions.

Education and counselling about brand change is important

The different appearance of medicines when brands change can be troubling to some patients and can lead to confusion and difficulty in complying with medicine regimens. Pharmacists have an important role in helping patients through this change. Many pharmacies place stickers on new brands of medicine to assure patients that they are still receiving the same drug in the same amount, despite the different appearance. Verbal or written information may be required to counsel patients through changes in medicines associated with mental illness and other serious conditions.

Pharmacists can also ensure that patients get the same brand of medication for repeat prescriptions.

See BPJ Special Edition, March 2007 "Counselling patients through a brand change".

Prescribers should make the best decision for their patient

Despite education, counselling and reassurance, generic prescribing for some patients may compromise their ability or willingness to comply with their medicine regimen. In these situations it may be reasonable for the doctor to prescribe a branded medication.

Brand names should be used when prescribing drugs with a narrow therapeutic index, modified release formulations and drugs with different delivery systems. For all other situations, **generic prescribing is best practice**.

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Some generic drug names about to change

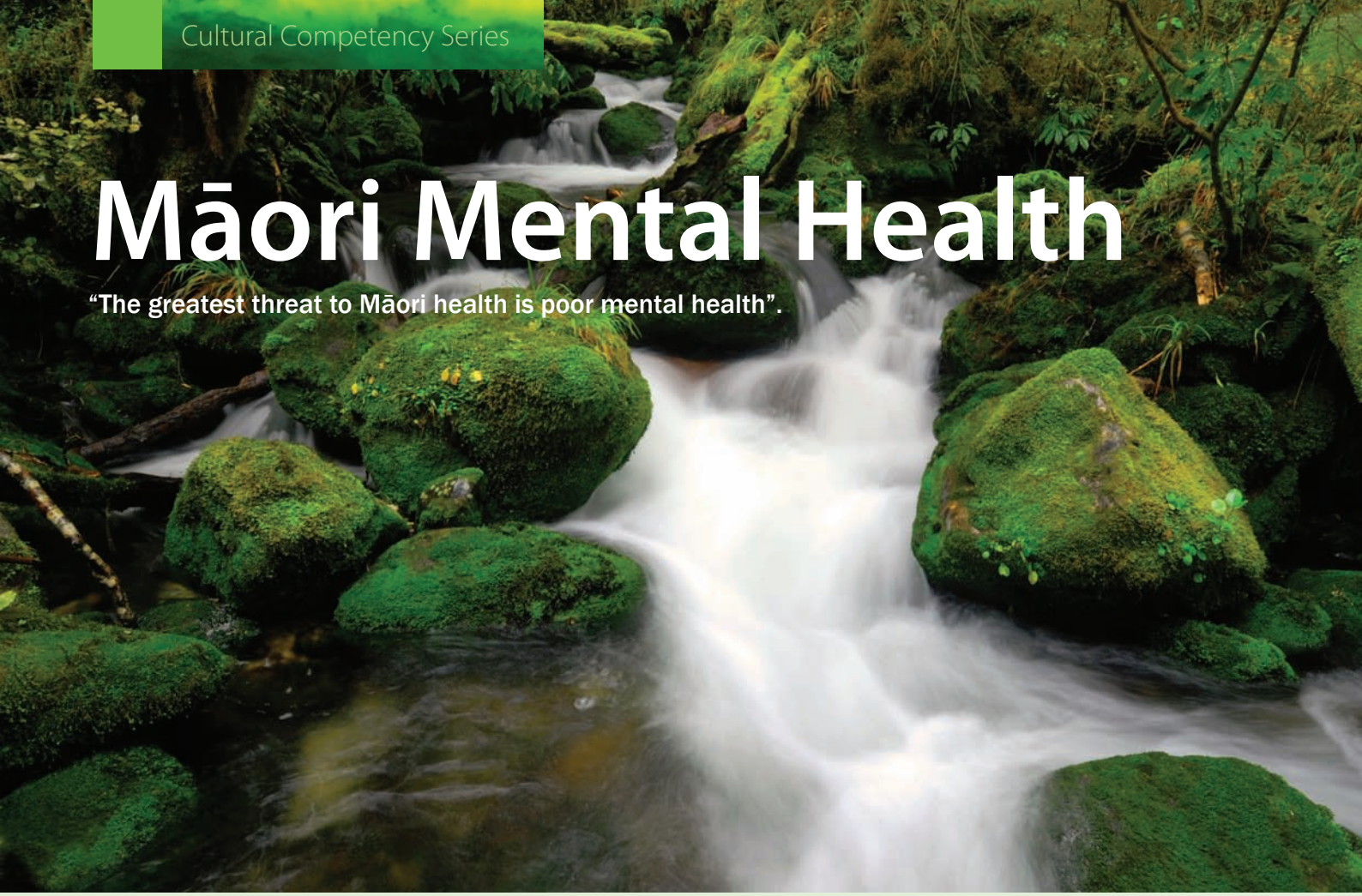
Submitted by Safe and Quality Use of Medicines Group

The World Health Organisation agreed many years ago that all drugs should have a recommended international non-proprietary name (rINN). Problems arise when this rINN differs from the British Approved Name (BAN) that most of us are familiar with. For many years manufacturers continued to use the BAN or even the US adopted name (USAN), however in 2004, UK manufacturers had to change to rINNs to obey a European directive.

This means that the names of some common drugs could be about to change on the shelves of pharmacies and on patients' bottles – in some cases they have changed already. Patients may worry about suddenly finding their frusemide has turned into furosemide and whether levothyroxine is the same as the thyroxine they had last time.

Some name changes may cause dispensing errors and confusion. Mercaptamine is very similar to mercaptopurine in name but not pharmacologically. Levothyroxine (previously thyroxine) sounds like liothyronine. Methotrimeprazine has started arriving on shelves labelled as levomepromazine. The oestrogens will no longer be spelt with an "o" and acyclovir and cyclosporin become aciclovir and ciclosporin respectively.

Self monitoring of blood glucose for people with non-insulin treated type 2 diabetes: An update



Māori Mental Health

"The greatest threat to Māori health is poor mental health".

Improving Māori mental health is a government health priority. This commitment can be achieved through tangible and culturally appropriate mental health services.[1]

Cultural Identity

Good mental health depends on many factors and among indigenous populations, cultural identity is considered critical. Being Māori is recognised as the basis for recovery for tāngata whaiora (Māori mental health service users) and lies firmly within the context of ones identity as Māori. The rediscovery of whakapapa – the connections that make us who we are and where we come from – is the foundation of recovery.[2]

One of the challenges for early intervention is the ability to define what actually constitutes a problem – what should health professionals be worried about and what is normal for the patient and whānau. Understanding cultural differences in the presentation of symptoms is important.[3]

Considerations may include:

- Is this just them?
- Is this how they normally are?
- Is something else wrong?

Cultural assessment

Cultural assessment is the process through which the relevance of culture to mental health is determined.[4] It is widely accepted as a key element of mainstream mental health service delivery and responsiveness to Māori. It supports providers to develop and maintain culturally effective and relevant services to tāngata whaiora and whānau.[1]

Kaupapa Māori Mental Health Services

Given the high prevalence of mental health issues in Māori and the fact that general practice is the leading source of service contact, GPs need to consider establishing links with Māori consumers, Māori providers and Māori mental health teams. Consideration needs to be given as to how shared care with Māori providers might work.[3]

Kaupapa Māori community health workers are willing to share their knowledge and skills with mainstream services, in order to develop intersectoral relationships and a team approach to improve outcomes for whānau.

While there is no set criteria, Kaupapa Māori mental health services offer a range of treatment and support services that typically include:[5]

- Whanaungatanga (kinship, family relationships)
- Whakapapa (genealogy)
- Cultural assessment
- Empowerment of tāngata whaiora and their whānau
- Te Reo Māori (Māori language)
- Tikanga Māori (customs and culture)
- Kaumātua guidance
- Access to traditional healing
- Access to mainstream health services
- Quality performance measures relevant to Māori

Kaupapa Māori mental health services can be accessed through DHB mental health services. There are also a number of contracted Kaupapa Māori mental health non-government organisations operating in some communities.

Cultural assessment should only be carried out by those trained to do so

Guidelines published by the Mental Health Commission in 2001 emphasise that cultural assessment is complementary to clinical assessment and should only be carried out by those trained to do so. Expertise in Tikanga (customs), Te Reo (language) and Matauranga (knowledge) Māori are fundamental prerequisites.[1]

The assessment is used to determine the mental state of tāngata whaiora. It can also be used to determine the significance of cultural factors and to plan treatment and rehabilitation processes to address these. Cultural assessment is only useful if it leads to a comprehensive recovery plan that includes appropriate cultural support throughout the whole clinical care pathway.[1]

Cultural assessment may take different forms and is an integral part of all points of care.

Cultural disparities in mental health care

Successful mental health management in primary care relies on the GP's ability to recognise and appropriately respond to mental health problems. However some research suggests that there may be disparities in provision of care. For example, a study of one South Auckland general practice published in 2002, found that while Māori were no more likely to be depressed, they were significantly less likely to be treated with antidepressants than non-Māori. However the study was not able to identify the reason for this difference, whether it was a disparity in care or an issue of patients not wanting to take medication.[6]

Some New Zealand health professionals hold unfounded beliefs that Māori are genetically more prone to psychosis and other serious mental illnesses.[7, 8]

Mate Māori and other specific Māori Concepts relating to Mental Health[9,10]

Some mental and behavioural states cannot be accommodated in Western classifications and Māori explanations for poor health can be quite different from Western beliefs.

Mate Māori, for example, is related to spiritual causes, and requires the intervention of a tohunga or priest. The term refers essentially to a cause of ill health or uncharacteristic behaviour which stems from an infringement of tapu (a tribal law) or the infliction of an indirect punishment by an outsider (a mākutu). It may take several forms, physical and mental, and various illnesses, not necessarily atypical in presentation, may be ascribed to it.

Mate Māori applies to physical as well as mental illnesses and has increasingly become a focus to explain emotional, behavioural and psychiatric disorders. Māori may be reluctant to discuss mate Māori fearing ridicule or pressure to choose between psychiatric and Māori approaches. However, one approach need not exclude the other as cooperation between traditional Māori healers and health professionals is now becoming acceptable to both groups.

Mate Māori does not mean there cannot be a mental disorder. Rather, it may be used to explain the cause of the illness rather than the symptoms. Mate Māori remains a serious concept within modern Māori society, and may be more convincing to Māori than complex clinical explanations.

While it is useful for health professionals to have some idea that mate Māori and other specific conditions exist and to have heard the terms, it is very important that they do not assume that they understand or have any expertise in them. This is the area of expertise of tohunga and kaumātua assisted by Māori cultural workers. **It is vital to seek expert cultural assistance if these concepts arise when working with Māori.**[10]

Some New Zealand health professionals hold unfounded beliefs that Māori are genetically more prone to psychosis and other serious mental illnesses.

The New Zealand Mental Health Survey 2003/4, Te Rau Hinengaro[11]

- Just over half of Māori have experienced a mental disorder during their lifetime, and just under a third within the past 12 months.
- The most common lifetime disorders for Māori were anxiety (31.3%), substance disorder (26.5%) and mood disorders (24.3%).
- Mental disorders for Māori were common in those aged 16 to 44 years, those living in low income households and those living in areas of high deprivation. There were no differences in rates by region or rurality.
- Contact with health services for mental health needs was low for Māori relative to need. Only half of those with a serious disorder in the previous 12 months had any contact with mental health services (compared with two-thirds of non- Māori).
- General practice was the leading source of service contact.

Other behaviours may also present as if they were mental disorders, for example:

- Whakamā - a mental and behavioural response that arises when there is a sense of disadvantage or loss of standing and can manifest as marked slowness of movement and lack of responsiveness to questioning, as well as avoidance of any engagement with the questioner. A pained, worried look can add to a picture that is suggestive of depression or even a catatonic state. But the history is different and the onset is usually rapid - unlike other conditions where a more gradual development occurs.
- Sometimes, because Māori will often report seeing deceased relatives or hearing them speak, a diagnosis of schizophrenia or some other psychosis may be made. However, visions or hearing voices in the absence of other mental health symptoms are not a firm basis for diagnosing a serious mental disorder in Māori.

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CASE STUDY FROM BEST HEALTH OUTCOMES FOR MĀORI: PRACTICE IMPLICATIONS[12]

"Smoking can be bad for your health"

Recognition of complementary world views

A 62 year old Māori man who works in a bank, visited his Pākehā GP because he didn't feel well and was short of breath. As the consultation progressed, the doctor felt that it was not going too well, so he shared these thoughts with the patient and asked if there was something else bothering him. The patient sighed and said yes. He said, "I know what's wrong, doc. I know why I'm crook. I took tobacco to the urupā (cemetery) and then had a smoke." The GP told the Māori patient that he didn't know what the significance of that was and asked if he could explain. The patient revealed that the urupā is tapu (sacred), while cigarettes are noa (common), so he had committed a serious breach. The doctor asked the patient if he knew what he had to do about that. The Māori patient heaved another sigh and explained that he had to see a priest.

Without deriding the patient's belief system ("No, you've got heart failure caused by hypertension and atherosclerosis."), the doctor acknowledged that while the patient sought assistance for the violation of tapu within the Māori culture, he could prescribe medicines to help with the breathlessness.

The patient's firmly held belief as to why he is unwell ("disease attribution") is rooted in his cultural world view: he's unwell because he's breached tapu by taking tobacco into the urupā and then smoking it. It is generally non-productive to argue disease attribution with a patient, as it is usually perceived as a sign of disrespect to their belief system.

By contrast, if you can show respect for their beliefs while simultaneously offering complementary assistance from the world of Western, orthodox medicine, your suggestions are much more likely to be adopted. In this case, the GP was comfortable with his patient maintaining his disease attribution and following the correct protocol for dealing with that breach of tapu, but he simultaneously offered supportive treatment for the breathlessness associated with heart failure. The patient was comfortable with the idea of seeking help from both Māori and Western cultures, and accepted the GP's prescribed treatment.

Antioxidants and ageing:

harmless placebo or dangerous to your health?

www.bpac.org.nz keyword: antioxidants

IN A RECENT BEST PRACTICE ARTICLE ("Alternative remedies and lifestyle measures for longevity" BPJ11, February 2008) we discussed the role of antioxidants in the ageing process. Antioxidants are known to counteract the effect of free-radicals which contribute to ageing. Eating fruit and vegetables, which contain antioxidants, can reduce the risk of some age related diseases. However there is very little evidence that supplements containing antioxidants can provide any benefit additional to dietary consumption and there is no evidence that they have any effect on human ageing.

A recently published systematic review of the effect of antioxidant supplements on mortality assessed 67 randomised controlled trials, with a total of 232 550 participants. The authors concluded that there was no evidence that antioxidants prevented ageing and in fact, some may increase mortality.

Overall, 13.1% of study participants randomised to receive antioxidant supplements died, compared to 10.5% of those receiving placebo or no intervention. Analysis of individual supplements found that vitamin A (Relative Risk RR 1.16, 95% C.I. 1.10 to 1.24), beta-carotene (RR 1.07, 95% C.I. 1.02 to 1.11) and vitamin E (RR 1.04, 95% C.I. 1.01 to 1.07) were all significantly associated with an increase in mortality, when given alone or combined with other antioxidants.

There were no differences in the effect of the antioxidants between study participants who were healthy or those who had various diseases. Treatment duration had no significant effect on the results but dose was significant. What this means is that someone who takes vitamin A supplements, especially at a dose close to or exceeding the recommended daily intake, may increase their risk of mortality by 16%.

The other antioxidants included in the study were vitamin C and selenium. There was no evidence that vitamin C increased longevity and a lack of evidence for selenium, although neither supplement increased mortality. More research is needed to establish the benefit or harm of these antioxidants.

So what do you tell your patients?

- Antioxidant supplements are unlikely to increase longevity.
- It is not necessary for a healthy individual to take antioxidant supplements.
- Excessive use of vitamins A and E and beta carotene may have a negative effect on lifespan.
- Fruit and vegetables containing antioxidants are not harmful.
- It is unknown if antioxidants have a role in the treatment of specific diseases or in specific patient groups, more research is needed.

Reference:

Bjelakovic G, Nikolova D, Gluud L et al. Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases. Cochrane Database Syst Rev 2008;2:CD007176.

Antioxidants

Vitamin A encompasses the retinoid group including retinol, retinal and retinoic acid. Vitamin A preparations may also be in the form of retinyl acetate or palmitate.

Beta carotene is a precursor (inactive) form of vitamin A and is the substance in carrots that makes them orange.

Vitamin E is also known as α-tocopherol and is most commonly sourced from wheat germ and soybean oils.

Vitamin C is also known as L-ascorbate and occurs naturally in many fruits and vegetables.

Selenium is a chemical element which occurs in different forms in the environment including as a trace element in soil. Other forms may be selenide salts or selenic acid.

Ten Minute Tutorial

Adding an alert for patients on clozapine

To set up an alert to use for patients on clozapine

1. From the menu select: Setup > Patient Register > Alert
2. Put a code, perhaps “cloz”, in the appropriate box and put “On Clozapine” in the description box.
3. Click OK, your alert is now set up for use.

To use the clozapine alert for a particular patient

1. Open the patient’s clinical records.
2. From the menu select: Module > Alerts
3. Click on the box in the window that opens to assign a new alert to the patient.
4. In the code box enter “cloz” or whatever code you used.
5. In the text box underneath write relevant details such as:

   “On clozapine --- watch for constipation, neutropaenia, myocarditis”

6. Tick the box labelled “Auto Prompt Alert”.
7. Click “OK”, your alert should now open whenever the patient’s clinical records are accessed.

How many patients in your practice have been taking a PPI for more than 6 months without review?

Free Starter Kit Samples of ranitidine and patient resources are available to help you manage these patients.

A step down regimen off a PPI, usually in 4-8 week steps, is appropriate for most patients.[1]

> 'It is recommended that within three months of initiation of treatment with a PPI, patients regimen be reviewed with the aim of reducing the dose and/or initiation of an alternative treatment option where clinically appropriate'.[2]

To help you manage your patients step down off a PPI, starter packs of ranitidine and patient information can be ordered at **www.gutreaction.co.nz**.

You can also e-mail **gutreaction@pharmac.govt.nz**

To order resources go to **www.gutreaction.co.nz** or fax your order direct to **0800 488 4636**.

1. Dyspepsia and Heartburn Management guidelines 2004, NZGG. 2. NZ Gastroenterology Society.

recognise review reduce relief

gutreaction

Evidence That Counts

The role of drugs in road safety

Australian Prescriber 2008;31:33-5
Full text: www.australianprescriber.com/magazine/31/2/33/5/

Drug use is increasingly associated with road accidents. While alcohol and illicit substances dominate, some prescription drugs can contribute to injury and death. Most drugs do not significantly increase the risks of accidents if they are taken as prescribed, however a number of commonly used drugs can impair the ability to drive safely. Awareness that some drugs affect driving will help to reduce their potential impact on road safety.

Driving skills

Foremost among the skills required for safe driving are vigilance, and the ability to interpret traffic situations and to divide attention between tasks.[1] The driver's behaviour and attitude also contribute to the risk of having an accident.

A large range of substances are known to impair the cognitive or psychomotor skills required for safe driving. Any drug acting on the central nervous system has the potential to adversely affect driving skills. Central nervous system depressants reduce vigilance, increase reaction times and increase errors associated with decision making and speed control in a very similar manner to alcohol. Drugs that affect behaviour may exaggerate adverse behavioural traits and risk-taking behaviour.

Prescribed drugs

With the exception of benzodiazepines the evidence for the role of prescribed drugs in road trauma is uncertain. In general, most drugs tend not to be significant risk factors on the road when the drugs are used as prescribed.

Some drugs can cause impairment due to their central nervous system depressant properties, particularly early in treatment before the patient becomes accustomed to the drug, or when the drug is misused.[2] Table 1 shows some prescription drugs and their relative risk of causing impairment. The most common examples seen in road trauma are the anticonvulsants and the antidepressants, but their presence does not necessarily mean that they had a contribution to the crash.

In many cases two or more impairing drugs including alcohol are detected. Combinations of drugs increase the opportunity for impairment and the risk of a serious crash.

Benzodiazepines

Benzodiazepines are well known to increase the risk of a crash.[3,4] They are found in about 4% of fatalities[5] and 16% of injured drivers taken to hospital.[6] In many of these cases benzodiazepines were either abused or used in combination with other impairing substances. When abuse occurs, the drugs may not have been prescribed to the person concerned. The illicit trade in these drugs is significant and they are often obtained by "doctor shopping". Medical practitioners do need to be aware of this possibility when prescribing benzodiazepines and related hypnotics such as zopiclone. If a hypnotic is needed a shorter-acting drug is preferred. Tolerance to the sedative effects of the longer-acting benzodiazepines used in the treatment of anxiety gradually reduces their adverse impact on driving skills.

Antipsychotics

This diverse class of drugs can improve performance if substantial psychotic-related cognitive deficits are present. However, most antipsychotics are sedating and have the potential to adversely affect driving skills through

Table 1: Medicines that may impair driving skills

Drug	Risk of causing impairment
Anticonvulsants (such as carbamazepine, gabapentin, phenobarbitone, phenytoin, valproate, vigabatrin)	Moderate to high
Antihistamines – sedating (such as azatadine, chlorpheniramine, cyproheptadine, diphenhydramine, promethazine, doxylamine, trimeprazine)	Moderate to high
Antihistamines– less sedating (such as cetirizine, desloratadine, fexofenadine, loratadine)	Low to moderate
Antipsychotics (such as amisulpride, chlorpromazine, haloperidol, pericyazine, clozapine, olanzapine)	Moderate to high
Benzodiazepines and related compounds (such as temazepam, nitrazepam, oxazepam, alprazolam, clonazepam, diazepam, zopiclone)	Moderate to high
Drugs for diabetes	Low to moderate
Muscle relaxants (such as baclofen, dantrolene, orphenadrine)	Moderate
Opioid analgesics (such as codeine, buprenorphine, methadone, morphine, oxycodone, pethidine, tramadol)	Moderate to high
Serotonin, mixed reuptake inhibitors and reversible monoamine oxidase inhibitor antidepressants (such as fluoxetine, sertraline, paroxetine, citalopram, venlafaxine, moclobemide)	Low
Tricyclic and tetracyclic antidepressants (such as amitriptyline, clomipramine, dothiepin, doxepin, imipramine, trimipramine, mianserin, mirtazapine)	Moderate to high
Sympathomimetics (such as pseudoephedrine, phenylephedrine)	Low to moderate

These risks relate to possible situations when the drug or a member of a drug class is used incorrectly or abused. Risk of significant impairment usually only occurs early in treatment.

blockade of central dopaminergic and other receptors. Older drugs such as chlorpromazine are very sedating due to their additional actions on the cholinergic and histamine receptors. Some newer drugs are also sedating, such as clozapine, olanzapine and quetiapine, while others such as aripiprazole, risperidone and ziprasidone are less sedating. Sedation may be a particular problem early in treatment and at higher doses.

Drugs for diabetes

Hypoglycaemia can be a significant problem. The drugs themselves have no major effect on skills, but how well they control blood glucose will affect driving performance.

Advice to patients

The product information of some drugs contains a precaution about driving. This caution may also be given on

the label the pharmacist attaches to the prescription. For many drugs, once patients are stabilised, their potential low risk of causing significant impairment is offset by their therapeutic benefit. Nevertheless, it is necessary to appropriately warn patients about the dangers of driving a motor vehicle early in treatment and when the patient is not mentally alert possibly due to persistent drug effects. Moreover, patients driving at night or working shifts where normal sleep patterns are altered are also at an increased risk of fatigue-related crashes. Many drugs can exacerbate the effects of sleep deprivation and increase the risk of a crash. Taking drugs with alcohol increases impairment of driving skills.

– Olaf H Drummer, Adjunct Professor and Head, Forensic and Scientific Services, Victorian Institute of Forensic Medicine, Department of Forensic Medicine, Monash University, Melbourne.

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Should we be concerned about jaw osteonecrosis with oral bisphosphonates?

Journal Watch, Vol. 28, No. 9, May 1, 2008
Full text www.journalwatch.org

Bisphosphonate-related osteonecrosis of the jaw (BRONJ) is defined as exposed, necrotic bone in the maxillofacial region that persists for more than eight weeks in current or past recipients of bisphosphonate therapy.[1,2] The condition can occur spontaneously or after invasive dental procedures. These lesions often expand – sometimes involving large areas of the alveolar bone – and no treatment has been proven to be effective. Reports of BRONJ first surfaced about five years ago, primarily in cancer patients who had received IV bisphosphonates for hypercalcemia and bone metastases. Experts generally accept the association between jaw osteonecrosis and IV bisphosphonates, which are often given repetitively in high doses.[2,3]

The data are less compelling for oral bisphosphonates. Based on case reports and surveys, jaw osteonecrosis has been described in relatively few patients who have received oral bisphosphonates to treat osteoporosis.

The American Association of Oral and Maxillofacial Surgeons (AAOMS) has published a position paper on BRONJ.[1] The authors recognise limitations in the published data, but they believe that jaw osteonecrosis occasionally occurs in patients who have received oral bisphosphonates. They recommend no alteration to or delay in necessary dental surgery for patients who have taken oral bisphosphonates for fewer than three years and have none of the characteristics that are thought to be risk factors (which can be found in the report[1]). However, for patients who have taken oral bisphosphonates for longer than three years, the statement urges prescribing physicians to stop

the drug three months before oral surgery and to restart it only after osseous healing has occurred. The authors acknowledge that these recommendations are consensus judgments that are not yet evidence-based.

How should primary care physicians respond to patients and dentists who are concerned about BRONJ? In our view, patients who are considering oral bisphosphonate therapy should be told that a small risk – which cannot be quantified yet and which probably emerges only after several years of treatment – is plausible. For most patients with documented osteoporosis and for those with osteopenia plus multiple risk factors for fracture, the potential benefits of bisphosphonate therapy outweigh the risks. However, some clinicians are prescribing bisphosphonates to patients with marginal indications (e.g., to recently postmenopausal healthy women with mild osteopenia). For relatively young patients, delaying bisphosphonate therapy seems prudent: We simply don't know whether years or decades of exposure to bisphosphonates might result in unanticipated harms.

What about the patient who is already taking an oral bisphosphonate and whose dentist recommends stopping it because an invasive procedure is necessary? Although the AAOMS recommendations admittedly are based on opinion and not hard data, stopping the drug temporarily is reasonable.

On the one hand, no proof exists to show that interruption of bisphosphonate therapy will make the dental procedure safer; but, on the other hand, a six or 12-month "drug holiday" is unlikely to alter fracture risk substantially in a patient who has taken a bisphosphonate for several years.[2] Indeed, the FLEX data suggested that a five-year drug holiday after five years of active treatment is reasonable for many patients.[4]

Practitioners and patients often are forced to make clinical decisions under conditions of uncertainty; the situation with BRONJ is no exception. Although bisphosphonates are extraordinarily valuable drugs with proven benefits, recent developments with cyclooxygenase (COX)-2 inhibitors, recombinant erythropoietins, rosiglitazone and postmenopausal hormone therapy should remind us to remain open-minded about unexpected effects of drug therapies.

– **Allan S. Brett, MD, and Peter B. Lockhart, DDS** (Dr. Lockhart is Chair of the Department of Oral Medicine and Director of the Oral Medicine Institute at Carolinas Medical Centre in Charlotte, North Carolina).

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Evidence That Counts

Antibiotics in acute rhinosinusitis: often prescribed, but rarely indicated

Journal Watch, Vol. 28, No. 10, May 15, 2008

About a third of patients who present with upper respiratory infections are diagnosed with acute rhinosinusitis, and 80% of patients with this diagnosis receive antibiotics, even though no known criteria distinguish between viral and bacterial aetiologies.

To determine whether a subgroup of patients could be identified that might derive benefit from antibiotics, researchers combined and reanalysed individual patient data from nine clinical trials that involved 2547 adults with clinical signs and symptoms of rhinosinusitis who were randomised to receive antibiotics or placebo. No patient had undergone imaging or culture before randomisation. Cure was assessed after eight to 15 days in all trials.

The odds ratio for cure in the antibiotic group was 1.37. The estimated number needed to treat (NNT) with antibiotics to achieve one additional cure was 15; the NNT was similar in all trials. Symptom severity, symptom duration, and age did not predict increased benefit from antibiotic treatment. Patients with purulent pharyngeal discharge derived somewhat greater benefit from antibiotics than did other patients, but the NNT for patients in this group was still 8.

Comment:

The authors conclude that adults with acute rhinosinusitis generally should not receive antibiotics, regardless of presenting signs and symptoms, and that guidelines that suggest antibiotic therapy after seven days of symptoms are not supported by evidence. Some clinicians might argue that an NNT of 8 or 15 is sufficient to warrant antibiotic therapy, but any benefits must be weighed against risks for adverse effects and increased antimicrobial resistance. Of course, patients with unusual signs or symptoms (e.g. high fever, periorbital oedema) suggesting a serious complication should be treated promptly with antibiotics.

— Bruce Soloway, MD

Young J et al. Antibiotics for adults with clinically diagnosed acute rhinosinusitis: A meta-analysis of individual patient data. Lancet 2008 Mar 15; 371:908.

Written Action Plans for Children with Asthma

Journal Watch, Vol. 28, No. 6, March 15, 2008

Written action plans are recommended by virtually every asthma guideline that has been produced by professional societies and government agencies, including the NIH. However, no consensus exists about whether plans based on symptoms are better than those based on peak-flow levels.

Canadian investigators conducted a meta-analysis of four randomised clinical trials (involving 355 school-aged children) that compared symptom-based action plans with peak-flow–based action plans. Other asthma interventions were similar in both groups, and follow-up ranged from three to twelve months.

Compared with peak-flow–based plans, symptom-based plans significantly lowered (by 27%) risk for unscheduled acute care visits (the primary outcome). Compared with symptom-based plans, peak-flow–based plans led to an additional half-day reduction in the number of

symptomatic days per week. However, no differences between groups were found in the number of patients who required rescue oral steroids or hospital admission or in school absenteeism, lung function, quality of life, or study withdrawals.

Comment:
Although the authors conclude that these data provide clear evidence that action plans based on symptoms are superior to those based on peak-flow levels, I do not agree. The primary outcome favoured symptom-based plans, but one secondary outcome favoured peak-flow–based plans, and most assessed outcomes were similar with the two plans. Thus, one could reasonably allow parents to choose the approach with which they are most comfortable.

— **Howard Bauchner, MD**

Zemek RL et al. Systematic review of random ized controlled trials examining written action plans in children. Arch Pediatr Adolesc Med 2008 Feb; 162:157.

Bandolier

Independent evidence-based thinking about health care

REMINDER - Bandolier has now ceased publishing its monthly print editions. The Bandolier website is now updated with new information

Bandolier Knowledge. In this section of the website, Bandolier collects good quality evidence under a variety of different headings. They search for systematic reviews of treatments, of evidence about diagnosis, epidemiology or health economics, and abstract it.

Go to http://www.jr2.ox.ac.uk/bandolier

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CVD Quickscreen

*A screenshot showing the number of fields required to calculate a 5-year CVD Risk using the new **CVD Quickscreen** module. Most of these fields will be pre-populated by the PMS.*

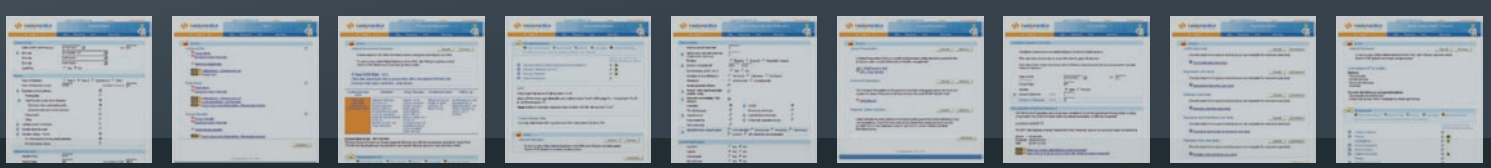
CVD Quickscreen

The new ***CVD Quickscreen*** module calculates 5-year CVD Risk using only the minimal number of fields required by the Framingham equation. Because many of these fields are prepopulated by the PMS, a CVD Risk can usually be determined in seconds.

New Modules

The ***Cardiac Atrial Fibrillation*** and ***CVD Quickscreen*** modules are newly released this month, taking the total of currently available modules to more than 20. There are a further dozen ready for release or in advanced development stages.

The screenshots below show some of the most popular of the currently available modules.



Diabetes | ***Nursing Management Guides*** | ***CVD*** | ***Chronic Kidney Disease*** | ***Atrial Fibrillation*** | ***Forms*** | ***Clinical Toolkit*** | ***Interactive Education*** | ***Healthy Children***

A weed from far away

Dear bpac,
Thank you for your article on Māori Health. It suprises me that it did not mention cannabis which in my opinion is one of the major factors underlying adverse physical and mental health of people who use it, and it reduces the individuals motivation to strive for good health.

I did your recommended search of my high risk Māori males >35, which was a very valuable and instructive exercise. Analysis of their notes revealed that most of them either smoke or have been smokers, and at least 33% of them smoke cannabis. Cannabis tends to induce a mental state of apathy, and it is not suprising that cannabis smokers are not highly motivated to improve their health while continuing to smoke it.

Why did your article not say anything about this extremely important adverse influence on Māori health?

We will never get to the root of ill health if we chose to ignore it.

He Taru Tawhiti - "a weed from far away"

GP, South Island

We agree that cannabis use is an important health issue for all communities and we will consider addressing this in a future edition of BPJ.

BPJ 13 Māori Health Edition – Immunisation

Dear bpac,
As a Pakeha having worked as a Tamariki Ora nurse at Whaiora Whanui Māori health provider in Masterton and Arai Te Uru Whare Hauora in Dunedin I am heartened to see Best Practice magazine acknowledge the diverse reality of Māori people.

I read with interest the item on bronchiectasis as I had not connected that disease with immunisation before. I noticed the statistics for Māori two year olds being fully immunised had increased significantly since the referenced figure of 42%. As of 24th April 2008, according to the National Immunisation Register, the national immunisation coverage rate for Māori at age two years is 68%, with a variance across the 21 DHBs between 56% and 90%. All 21 DHBs now have childhood immunisation outreach services and general practices are working hard to reduce disparity, going the extra mile to recall or refer those children who might otherwise miss out.

Our message in general practice is to encourage access to timely immunisation to avoid breakthrough disease, in particular from Hib, Pertussis and Strep. pneumoniae. On Page 3 of BPJ 13 I read the sentence ; 'Rheumatic fever is unlikely to be seen in children under three years because their immune systems are not fully developed'. I would prefer to see the terms 'inexperienced' or 'naïve' used in this context to avoid perpetuating the misperception that we should wait until children are older before immunising. Trained vaccinators are aware that the infant immune system is fully developed at birth and that the vaccines on the immunisation schedule in New Zealand make a miniscule contribution to the massive antigenic challenges in the natural environment. All of our communication ought to convey this message on the importance of timeliness as the alternative delaying immunisation leaves us open to criticism in the event of an unprotected child becoming ill from a vaccine preventable disease.

Kind regards

***Barbara Warren**, Coastal Otago Immunisation Coordinator*

Amiodarone monitoring

Dear bpac

In BPJ 2, December 2006, you ran an article on amiodarone monitoring requirements. The article suggested a range of tests that are not practical in a GP practice, for patients or GPs to manage. The article finishes with an editors note to revisit the requirements in the next issue – I cannot find any follow up.

GP, Waikato

We asked Cardiologist, Dr Stewart Mann to comment on the issue of monitoring patients on amiodarone. He says:

As a recent paper[1] suggested, many monitoring recommendations are not evidence based and some recommendations are over the top pragmatically with no evidence of more rigorous monitoring leading to fewer important side effects. For example, the usefulness of any routine monitoring of pulmonary function tests or chest x-ray to pick up early fibrosis is not at all established.

Some clinicians suggest more intensive monitoring may be justified with higher amiodarone maintenance doses (400mg/day +).

Reasonably pragmatic guidelines produced in 2000[2] suggest a number of baseline tests and follow-up clinical assessment (history and examination) every three months in the first year (Table 1).

It is likely that a cardiologist would follow a patient newly prescribed amiodarone three-monthly for the first year and be responsible for the recommended tests over that period. GPs may take responsibility thereafter if no cardiac follow-up is otherwise required.

Reference:

1. Stelfox H, Ahmed S, Fiskio J, Bates D. Monitoring amidodarones toxicities: recommendations evidence and clinical practice. Clin Pharmacol Therap 2004:75(1):110-22.
2. Goldschlager N, Epstein AE, Naccarelli G, et al. Practical guidelines for clinicians who treat patients with amiodarone. Arch Intern Med; 160(12): 1741-8.

Table 1: Recommended amiodarone monitoring

	Baseline	Follow-up	
		6 monthly	Annually
Electrocardiogram (ECG)	✓		✓
Chest x-ray (CXR)	✓		✓
Thyroid function tests (TFTs)	✓	✓	
Liver function tests (LFTs)	✓	✓	
Pulmonary function tests (PFTs)	Only if any symptoms of respiratory deficiency	Only for those with suspicious symptoms	
Eye examination	Only if visual impairment	Slit lamp assessment suggested for those with suspicious symptoms	

The use of SSRI's for treating depression: An Open letter

In February 2008, researchers at the University of Hull, UK, published an article which concluded that "there seems little evidence to support the prescription of antidepressant medication to any but the most severely depressed patients".

[Kirsch I, Deacon BJ, Huedo-Medina TB et al. Initial severity and antidepressant benefits: a meta-analysis of data submitted to the Food and Drug Administration. PLoS Med 2008;5(2):e45].

The following day, New Zealand newspapers ran the story; "Anti-depression drugs don't work".

In March 2008, the BMJ published an article by Turner and Rosenthal [BMJ 2008; 336-:516-7]. Turner and Rosenthal respond to the conclusions of Kirsch et al and point out that the efficacy of antidepressants is not an absolute measure but depends on how clinical significance is defined.

In an earlier article (January 2008), Turner and Rosenthal assessed FDA data on 12 antidepressant drugs, and derived an overall effect size of 0.31 (on a scale from 0-1). Kirsch et al used the same FDA data to examine four of these 12 drugs and calculated an overall effect size of 0.32. Despite the apparent agreement, interpretation of these results, by Kirsch et al on the one hand and by Turner and Rosenthal on the other, has been quite different. In contrast to the conclusion of Kirsch et al that antidepressants are ineffective, Turner and Rosenthal concluded that each drug is superior to placebo.

Turner and Rosenthal explain their different interpretation in relation to Kirsch's use of the criteria for clinical significance recommended by NICE. Clinical significance is important because drug trials can show benefit of drug over placebo that is statistically, but not clinically, significant. Tests of statistical significance (e.g. p values) tell whether the true effect size is zero or not (i.e. they use the null or no effect or no difference hypothesis) but say nothing about the size of the effect. Effect size is required to gauge clinical significance. By convention, values of 0.2, 0.5 and 0.8 represent small, medium and large effects respectively – the values chosen are relative (to each other) and they are arbitrary – i.e. they are no more reliable than intuition. Using this convention, NICE chose 0.5 as the cut-off below which they deem drug benefit to be not clinically significant. But this decision transforms a continuous measure of effect into an all or none (yes/no) measure and Kirsch's (fallacial) conclusion that the overall statistical effect size of 0.32 equates with NO benefit from antidepressant drugs.

Actually, 0.3 is a positive effect – not a full effect, but a significant effect between small and medium. Given this clinical interpretation of effect size, Turner and Rosenthal advise a circumspect but not dismissive approach to use of antidepressants – patient response is not all or none; partial response should be expected.

In the light of this appraisal of the original report by Kirsch et al, we suggest that GPs should take the decision to prescribe or not prescribe SSRI treatment, for patients with mild to moderate depressive illness, on a case-by-case basis.

***Dr Peter Cardon**, Chair of the South Link Health Clinical Advisory Committee*

Gastroprotection for people taking NSAIDs?

Dear bpac,
For patients who need long term NSAIDs (and we try to keep this to a minimum!) do you still recommend omeprazole 20mg/d (or equivalent PPI). Would ranitidine 300mg/d be an alternative?

Also do you recommend checking for H. Pylori *(by stool) first before starting long term NSAIDs?*

Is there any good evidence for the above or is it just expert opinion?

Thank You,

GP Peer Review Group, Nelson

NSAIDs can cause several problems in the gut such as bleeding, perforation, ulceration and dyspepsia.

The first thing to consider is minimising risk for all people about to start long-term NSAIDs.[1] For example, factors to consider are:

- Is there a safer alternative (regular paracetamol)?
- Can you choose an NSAID with a lower relative risk of GI toxicity, e.g. ibuprofen?
- Is the lowest effective dose being used?
- Has the person been informed of potential adverse effects and what to do should they occur?
- How long does the treatment need to continue? Regular review of risk status is needed.
- Is the person on aspirin? If so:
    - Has the additive effect of aspirin and any other NSAID been considered?
    - Is the person on a COX-2 selective drug? COX-2 drugs lose their selectivity in the presence of aspirin.

Co-prescription of agents that protect the gastrointestinal mucosa is recommended for those aged 65 years or over with one additional risk factor and for those aged less than 65 years with two or more risk factors (see box).[1]

Proton pump inhibitors (omeprazole 20mg/day or equivalent), or misoprostol (≥ 600mg/day if tolerated) may be considered for protection against NSAID-associated gastric or duodenal ulcers. Adverse effects such as colic and diarrhoea may limit the use of misoprostol.

H2-receptor antagonists may not be adequate for NSAID gastro-protection against the more severe complications but they do improve dyspepsia related to NSAID use.

The following characteristics predict the risk of adverse upper GI events in those taking NSAIDs:

Use of NSAID (includes aspirin, and COX-2 plus aspirin) **plus** any of the following:

- History of peptic ulcer
- History of upper GI bleeding
- Concomitant disease, especially coronary heart disease
- Increased frailty such as substantial arthritis-related disorder
- Previous NSAID gastropathy
- Concomitant use of corticosteroids, anticoagulants or bisphosphonates
- High dose of NSAID (includes NSAID + aspirin)
- *H. pylori* infection

Consider cardiovascular risk also

It is important to consider an individual's cardiovascular as well as gastrointestinal risk, before initiating any NSAID (especially a COX-2), and then to consider the questions of – with or without a gastroprotective agent, and with or without concomitant aspirin? See Table 1.

Table 1. Prescribing NSAIDs for people with cardiovascular or gastrointestinal risks

	No or low NSAID GI risk	NSAID GI risk
No CV risk (without aspirin)	Non-selective NSAID	COX-2 or non-selective NSAID + PPI COX-2 + PPI for prior GI bleeding
CV risk (with aspirin)	Naproxen* + PPI if GI risk of aspirin/ NSAID combination warrants gastroprotection	PPI irrespective of NSAID. Naproxen if CV > GI risk COX-2 +PPI for prior GI bleeding

* Naproxen is not associated with an excess risk of vascular events as are some other traditional NSAIDs

NSAID use and *H. pylori* infection are independently associated with an increased risk of gastrointestinal bleeding and ulceration. Treat known *H. pylori* infection in people about to start long-term NSAID therapy.

In people who are already taking an NSAID, eradication of *H. pylori* is unlikely to reduce the risk of NSAID-induced bleeding or ulceration.

In New Zealand *H. pylori* prevalence is generally low so routinely testing for it pre-NSAID is probably not worth it, particularly in those who are not at high GI risk.

However patients about to start long term NSAID therapy who have a history of GI complications may benefit from testing and eradication of *H. pylori* before initiation of treatment to prevent GI complications.[2, 3]

Thank you to Dr John Wyeth, Gastroenterologist and Clinical Leader, Capital and Coast DHB, for his contribution to this answer.

References:

1. New Zealand Guidelines Group. Management of dyspepsia and heartburn, 2004. Available from: http://www.nzgg.org.nz/ (Accessed May 2008)
2. British National Formulary (BNF). BMJ Publishing Group and Royal Pharmaceutical Society of Great Britain. March 2008.
3. Ables AZ, Simon I, Melton ER. Update on Helicobacter pylori treatment. Am Fam Physician 2007; 75(3): 351-358.

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