RISEDRONATE | LOW DOSE ISOTRETINOIN | SUDDEN UNEXPECTED DEATH IN INFANCY | PMS TIPS

# Best Practice

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## Assessing wheeze in pre-school children

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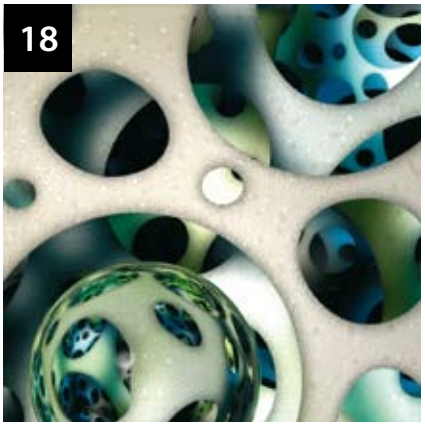

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# Sudden unexpected death in infancy: Where are we now?

Sudden unexpected death in infancy (SUDI, see: "New terminology", over page) refers to the death of an infant aged under one year, which is initially unexplained. Death usually occurs while the infant is asleep. The incidence of SUDI has declined significantly since public prevention campaigns began in New Zealand in the early 1990s. Prior to this, in the 1980s, the SUDI mortality rate was over 4 per 1000 live births.[1] This corresponded to over 200 infant deaths per year.[1] Now, the mortality rate is less than 0.8 per 1000 live births (across all ethnicities), with approximately 50 deaths per year.[1]

This success, however, masks several important factors. The first is that New Zealand still has one of the highest rates of SUDI in the developed world.[2,3] Australia, for example, has an incidence of 0.4 cases of SUDI per 1000 births, half New Zealand's rate.[4] The second is that the decline in infant mortality has not been equal among all New Zealanders. Māori infants now form the overwhelming majority of SUDI deaths, with a mortality rate five times that of European infants.[3] In 2009, Māori and Pacific infants accounted for 75% of all SUDI deaths (Māori 61.8%, Pacific 12.8%).[3] Infants born to families in lower socioeconomic areas also have a disproportionally high likelihood of SUDI.[5] Younger maternal age is another significant risk factor, with the majority of SUDI deaths occurring in infants whose mother is aged under 25 years.[6]

The increased incidence of SUDI among Māori in particular is thought to be due to greater levels of high-risk behaviour, such as maternal smoking, and poorer knowledge about SUDI

## Ministry of Health's recommendations for Safe Sleep

The Ministry of Health has published safe sleep recommendations for parents of young infants. It is emphasised to parents that SUDI is extremely rare when infants are protected by being put to bed in safe sleep conditions.

Parents are advised that they can protect their infant by doing the following things:[12]

- Place the infant to sleep on their back with their face up
- Ensure the infant's face is clear of bedding and they cannot become trapped or strangled. Pillows and bumper pads should be avoided, the infant should not be placed on soft surfaces or loose bedding and there should be no gaps in the bed.
- Put the infants to sleep in their own cot, bassinet, wahakura or Pepi-Pod, in the same room as the parent. Infants should not sleep in a bed with another person (either adult or child).
- Provide a smoke-free environment both during pregnancy and after birth
- Where possible, mothers should breast feed the infant

## New terminology, new understanding

Sudden unexpected death in infancy (SUDI) refers to the death of an infant aged under one year, usually while sleeping, which is initially unexplained. Sudden infant death syndrome (SIDS) refers to cases in which the death remains unexplained after a thorough investigation.[8] SUDI is a broader term, including deaths that can be explained, such as accidental asphyxiation, and those that cannot. This has prevented the artificial lowering of SIDS mortality rates as the accuracy of investigation and mortality coding increases. However, it is possible that the change in terminology may account for some of the decline in the reported incidence of SIDS/SUDI over the years.


risk factors, along with lack of access to this information.[7] What is less well understood is why Māori have poorer knowledge about SUDI. Maternal education about risk factors has been successful among Europeans and families in higher socioeconomic areas, but has failed to have a strong impact among the groups most at risk.[6] This may be partly attributable to less exposure to health education among Māori women, such as lower attendance at antenatal classes.[7] Educational messages that are not culturally tailored to Māori families also contribute to the problem.[7]

This gap in health knowledge presents a strong opportunity, and responsibility, for General Practice to help address disparities. The focus should be on explaining the factors which increase the risk of SUDI, why these factors present a risk and helping to find ways to minimise or overcome risks that are culturally and financially acceptable. Information, conveyed in a culturally relevant way, is the key to reducing rates of SUDI in New Zealand.

## The cause of SUDI: a trio of risks

Understanding of the causes of SUDI has grown over the last two decades. The current model is multi-factorial and relies on three aspects being simultaneously present:[9, 10]

- An infant in a critical developmental period
- An underlying vulnerability present since birth
- Exposure to an external stressor

**The critical developmental period** is from age one month to one year.[8] However, 90% of deaths occur in infants aged less than six months, with a peak between ages one and four months.[8]

**The vulnerabilities** that lead to SUDI are only now beginning to be understood. They include low birth weight (either from pre-term birth or low weight normal gestation births), abnormalities in the arousal system, serotonin receptor abnormalities and genetic polymorphisms, such as altered calcium channel genes which may affect cardiac rhythms.[11] Most of these vulnerabilities are genetic, present from birth, difficult to identify and non-modifiable.[10]

**The external stressors** for SUDI are factors which place the infant at a higher risk of asphyxiation, re-breathing of expired gases, overheating or other similar risks. The factors are usually modifiable and recognising them and creating public health messages around their avoidance has helped to reduce the burden of SUDI worldwide.[1] It is these risk factors that present the greatest opportunity for SUDI prevention in primary care.

## Vulnerabilities: modifiable risk factors for SUDI

### Sleep position

Infants should be placed to sleep in a supine rather than prone position, i.e. on their back, not on their front. The promotion of this behaviour has been the single greatest factor in reducing infant death while sleeping.[1] It is estimated that prone sleeping increases the risk of SUDI between 3 – 14 times.[13] Side-sleeping is also strongly associated with an increased risk of SUDI, as it increases the likelihood that the infant will roll into a prone position.[13]

Some parents may be hesitant to place infants in a supine position due to the belief that this is associated with choking, reduced muscle development or deformational plagiocephaly (the flattening of one side of the head). However, the risk of aspiration, apnoea and cyanosis are not increased when an infant is placed in a supine position, compared to a prone position.[13] Prone sleeping is not a recommended method to prevent gastro-oesophageal reflux in infants; raising the head of the bed may be helpful.[13]

Infants should be placed on their back for sleeping from birth until at least age twelve months. In order to help infants develop head control, upper body strength and reduce the risk of plagiocephaly, "tummy time", where the infant is placed on their front on the floor, is recommended when the infant is awake and under adult supervision.[13] Parents should be reminded to instruct all caregivers about the correct sleeping position for the infant.

### Bed sharing

Adults sleeping in the same bed as an infant, i.e. bed sharing or co-sleeping, increases the risk of SUDI.[8, 13] A retrospective study of SUDI cases in Wellington found that 50% of the reported deaths occurred while bed sharing.[14] A similar study in Auckland found that 67% of SUDI deaths occurred while bed sharing, and the vast majority of these deaths (97%) occurred in Māori and Pacific infants.[15]

The risk associated with bed sharing is highest in infants aged under three months.[13] The risk is further increased if the infant is sharing a bed with a person who smokes, has consumed alcohol or taken drugs.[8, 13]

There has been resistance to advice against bed sharing due to perceived advantages such as ease of breast feeding and night-time bonding.[9] In addition, bed sharing is viewed as a culturally important part of childrearing by many Māori and Pacific peoples, with important practical, psychological and spiritual benefits for the infant.[1, 7] While this issue can be sensitive, most consensus statements recommend that the risks of bed sharing should be discussed with the parents, regardless of ethnicity or cultural views, as parents have a right to evaluate the risks and benefits themselves.[13]

Ideally, infants should be placed in a cot, next to the parent's bed, until at least age six months.[8, 13] Interventions that allow safe bed sharing to take place are also acceptable, such as wahakura (woven flax bassinet) or Pepi-Pod (a basket-like device), (see: "Māori and Pacific sleep safe interventions", over page).

### Mattresses, mattress protectors and bumpers

Soft bedding and sleeping surfaces, such as pillows, quilts, comforters, sheepskins and mattresses not designed for infants, increase the risk of SUDI through airway obstruction, re-breathing of expired gases and overheating.[13] Mattresses should be new or in good condition, and tightly fitted to the cot. If a mattress is ill-fitting, an infant can become trapped between it and the wall of the cot.[13] Mattress bumpers should not be used and soft toys should not be placed in the cot.

In the past, some experts recommended wrapping the cot mattress in polythene, in theory to prevent toxic gases from the mattress reaching the infant. However, this theory has now been discredited,[8] and there is a potential risk of suffocation with the polythene.

### Over-heating

Over-heating is associated with an increased risk of SUDI.[1, 8] However, the risk is lessened if the infant is placed in a supine position.[1]

Infants should be dressed appropriately for the environment, with no more than one extra layer than an adult would wear to be comfortable.[8] Blankets and coverings should also be appropriate to the environment. Ideally, the temperature of the room the infant sleeps in should be between 18 – 22°C.[1]

### Swaddling

Swaddling refers to the practice of firmly wrapping young infants in light sheeting or muslin. It is thought to create a tranquil sleeping state and longer sleep periods.[16] Evidence is conflicting on the association between swaddling and the risk of SUDI.[16] It is thought that because swaddling increases sleep time and reduces arousal, it may increase the risk of SUDI.[16]

## Māori and Pacific sleep safe interventions

The emphasis of SUDI prevention has changed over time, from giving strict "rules" that help prevent SUDI, to providing education and interventions that allow for culturally important behaviours to continue in a safer way. One of the most effective of these interventions has been the wahakura, a flax bassinet that allows the infant to share the parent's bed while sleeping in their own space. The device prevents exposure to adult bedding, mattresses and pillows, and reduces the risk of the parent crushing or overlaying the infant.[20] Correct use of a wahakura, along with education relating to SUDI risk factors, appears to have been highly effective in pilot studies.[20]

Producing wahakura from flax is a skill-intensive process, and therefore they are not available commercially.[20] As a result, a similar device called a Pepi-Pod has been developed and is now being produced on a larger scale. A Pepi-Pod is essentially a wahakura made from recycled plastic (the bottom of a clothes basket) that comes with a fitted mattress and safe bedding. Pepi-Pods are currently being trialled in selected high-need groups around New Zealand, and have been shown to be highly effective and acceptable.[20]

The general practice team are encouraged to work collaboratively with Māori health providers, who are also working within their communities to reduce the incidence of SUDI. This may help to overcome the barriers to knowledge that have created the current ethnic disparities in SUDI incidence over the last two decades.

For more information on Pepi-Pods and similar interventions, see:
**www.whakawhetu.co.nz/pepi-pod.html**

Infants should not be swaddled in heavy material, which can cause over-heating, and infants who are swaddled should never be placed in a prone position for sleeping.[16]

### Cigarette smoke exposure

There is strong evidence that antenatal exposure to cigarette smoke and nicotine increases the risk of SUDI.[8, 13] *In utero* exposure to tobacco smoke increases the risk of intra-uterine growth retardation and pre-trem birth.[13] It also increases the recovery time from hypoxia, decreases heart rate variability and removes the normal relationship between gestational age at birth and predicted heart rate.[8] These factors represent the vulnerabilities outlined in the trio of risks.[9] There is also evidence that exposure to maternal smoking *in utero* reduces the frequency of arousal from sleep in the infant, which is a strong risk factor for SUDI.[9] Nicotine exposure may also alter serotonin receptors in the brain stem; brain stem abnormalities involving the serotonergic system are found in up to 70% of cases of SUDI.[8]

While General Practitioners rarely act as lead maternity carers, any consultation before or during pregnancy should be used to encourage smoking cessation. This should extend to all household members. Parents and family members who wish to stop smoking should be offered and encouraged to use smoking cessation supports, e.g. Quitline and NRT.

### Breast feeding

Breast feeding is thought to have a protective effect against SUDI and other causes of post-natal mortality.[13] Infants should ideally be breast fed exclusively until age six months, with continued breast feeding, alongside complementary foods, until the infant is aged at least one year.[17]

As of 2010, approximately 85% of infants in New Zealand were breast fed up to age six weeks.[1] However, there is a lower rate of breast feeding, and earlier breast feeding cessation, among Māori mothers.[7]

Barriers to breast feeding should be discussed, and education and support provided where necessary.

### Immunisation

Infants who are fully immunised have a decreased risk of SUDI.[13, 18] There may be a misconception among some parents that vaccines, particularly diphtheria-tetanus-pertussis (DTaP), are associated with an increased risk of SUDI. A series of studies over the last two decades has consistently refuted this association.[13]

Parents should be strongly encouraged to keep up to date with their child's immunisation schedule, and if any immunisations have been missed, plan a catch-up immunisation as soon as possible.

## Dummy (pacifier) use

There is a recognised association between the use of a dummy (pacifier) and reduced risk of SUDI.[13] The protective mechanism is not well understood, however, and dummy use is associated with several adverse effects, such as malocclusion of the teeth, increased risk of dental caries, otitis media and earlier cessation of breast feeding.[19]

If dummies are used, they should not be introduced before breast feeding is firmly established, usually at age three to four weeks.[8]

## Further resources for parents

Patient handouts and waiting room posters can be found at the Ministry of Health Safe Sleep website, see: **www.health.govt.nz** (Keywords: safe sleep)

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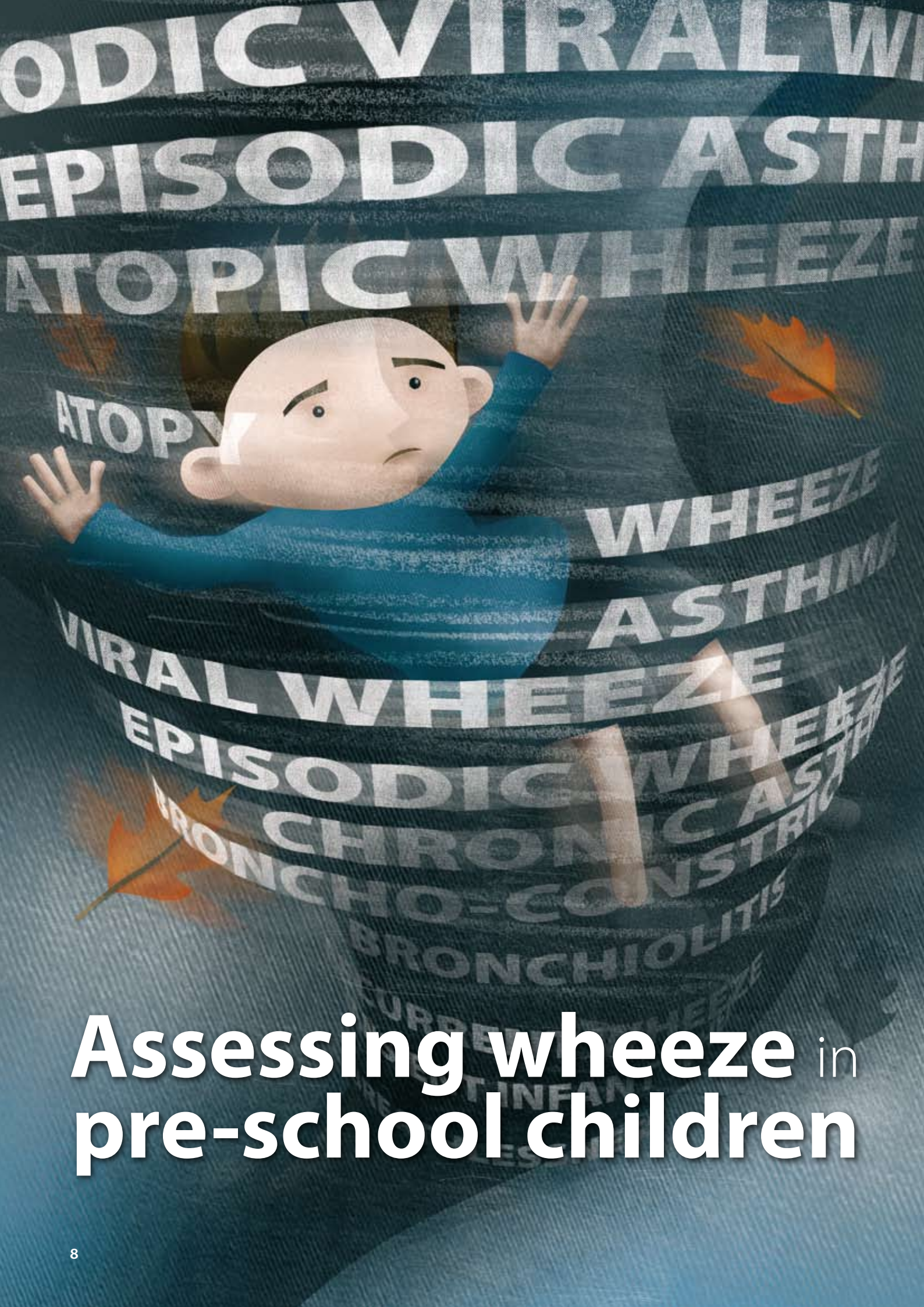



# Assessing wheeze in pre-school children

*Wheeze in children aged less than five years has many potential causes. Often it is regarded as the first sign of asthma, however, a substantial proportion of young children who wheeze will not go on to develop asthma.[1] In infancy, bronchiolitis is the most likely cause of wheeze. As children get older, episodic viral wheeze becomes more common. Atopic wheeze is most likely in children with risk-factors, such as a family history of asthma. By school-age, some of these children with wheeze will be diagnosed with asthma and others will have "grown out" of their symptoms. Therefore, rather than focusing on making a diagnosis when a young child presents with wheeze, it is more important to ensure the child receives appropriate management of their symptoms and that the parents receive education about their child's treatment and advice about vaccinations, infection prevention and maintaining a smoke-free home.*

## Not all that wheezes is asthma

Half of all children will have an episode of wheeze before school age.[2] Many will "grow out" of their symptoms by the time they attend school, but some will go on to have recurrent respiratory symptoms and a clear pattern of reversible airway obstruction that will be recognised as asthma.[2] It can be challenging for the clinician to differentiate those young children who will go on to have asthma from those who will not.[1]

Wheeze is clinically defined as a continuous, musical sound due to intrathoracic airway obstruction.[1] The small physical size of a young child's respiratory system, an immune system that is still developing and high levels of exposure to viral respiratory pathogens make wheeze both more common and more difficult to diagnose in young children than in older children.[3] Environmental factors also play a significant role in the development and severity of wheeze. Exposure to tobacco smoking, both before and after birth, significantly increases the likelihood of a child developing wheeze.[3] In addition, exposure to smoking in the household exacerbates respiratory symptoms.[3] There are many other environmental factors that can cause or aggravate wheeze in children, including damp homes, dust mites, pets, food allergies, air pollution and infections.

The usefulness of a diagnosis of asthma in pre-school children is debated. The signs, symptoms and treatment described by the terms "episodic viral wheeze" and "atopic wheeze" in pre-school children (Page 11) are very similar to "episodic asthma" and "atopic asthma", respectively.[2] Asthma is also commonly diagnosed in school-aged children who had previous recurrent wheeze. Because of this, some clinicians believe that wheeze and asthma are part of the same spectrum, and that giving the "label" of asthma leads to more appropriate treatment strategies.[4] Others are reluctant to make a diagnosis of asthma when the pathology in pre-school children is largely unknown, and there may be unintended social and psychological consequences of a diagnosis of asthma which later turns out to be transient.[2]

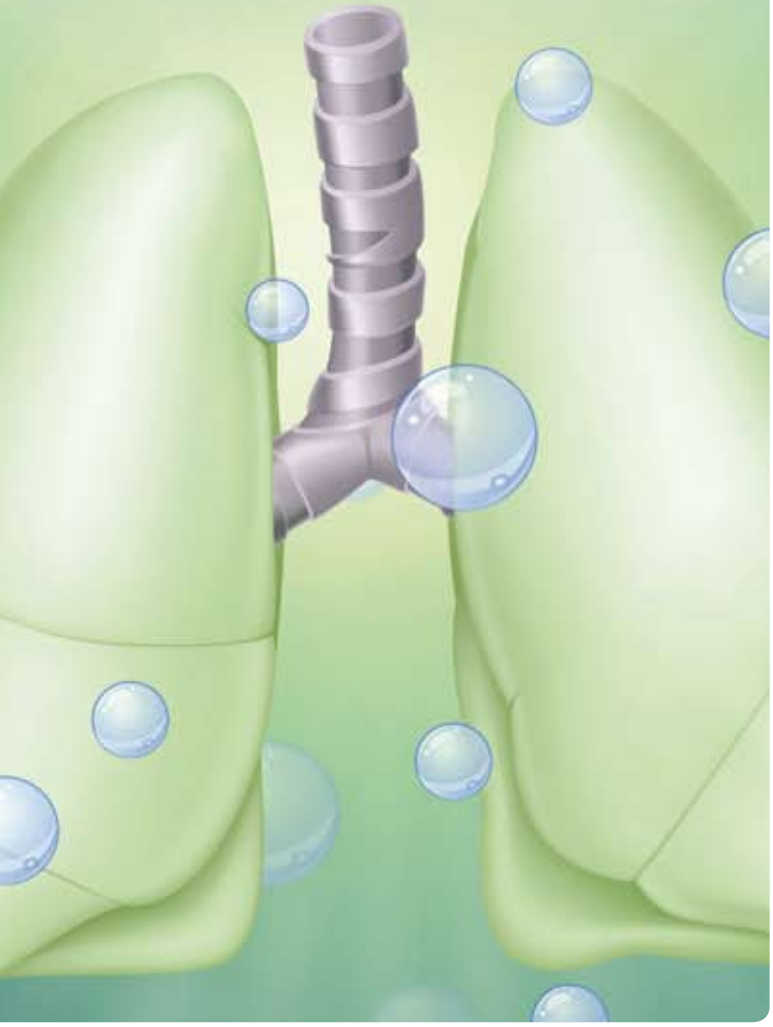
In practice, determining a definitive cause of wheeze in pre-school children requires a long-term approach, with consideration of the likelihood of the common causes, ruling out serious congenital or acquired conditions and assessment of the child's response to treatment. The goal for primary care should be to provide symptomatic control, to manage exacerbating factors and to monitor the child rather than provide a firm diagnosis, as the presentation may change over time.

## Transient infant wheeze

Transient infant wheeze is an epidemiological term for self-limiting wheeze that occurs in children aged up to three years.[4] The term has limited clinical utility but is commonly used in the literature to describe the group of children with recurrent wheeze who grow out of their symptoms by age three years.

Children in this group are generally born with smaller airways, have reduced lung function from birth, do not have an increased family history of atopy or asthma compared to children without wheeze and have often been exposed to tobacco smoke antenatally.[4] Prematurity and low birth weight may also be risk factors for transient infant wheeze.

In general, it is not possible to clinically differentiate transient infant wheeze from other forms of wheeze.



## The causes of wheeze in pre-school children

The first step in assessing wheeze in a young child should be to establish how long the symptoms have been present. If there is a recent, sudden onset of wheeze (i.e. that day or within a few days) and there is no obvious cause, e.g. viral illness, consider the possibility of an inhaled foreign body. If the wheeze is of recent onset, but a concurrent upper respiratory tract infection is present, consider episodic viral wheeze or bronchiolitis. If wheeze has been present for several weeks/months or the child has presented multiple times with wheeze, consider atopic wheeze. However, the symptoms may also be due solely to recurrent upper respiratory tract infections.

### Inhaled foreign body

An item which is sucked into the tracheobronchial system and becomes lodged can cause acute onset wheeze, dry cough and reduced lung sounds.[5] The key finding in a child with an inhaled foreign body is that symptoms began after an episode of choking or severe coughing.[5] However, this episode is not always observed and children may not volunteer the information.[5] If diagnosis is delayed other symptoms may be present, including dyspnoea and a wet cough with sputum production.[5]

Serious complications of an inhaled foreign body (including pneumonia, pneumothorax and subglottic oedema) are more likely when the diagnosis is made more than 24 hours after inhalation.[5] Long-term complications, such as recurrent pneumonia, lung abscesses and bronchiectasis, become more likely the longer diagnosis is delayed.[5]

Children with a suspected inhaled foreign body require immediate referral to a Paediatrician or emergency department.[5]

### Bronchiolitis

Bronchiolitis is an acute infection of the lower respiratory tract that primarily occurs in young infants, and is most common in winter.[1] It is usually caused by respiratory syncytial virus (RSV).[1]

In a child with bronchiolitis, tachypnoea, cough, hyperinflation of the chest and fine inspiratory crackles are likely.[6] A short, tight cough is likely to be present, and airway secretions play a significant role in obstruction.[6] The child may also have a low-grade fever (< 39°C).[6] A high-grade fever may indicate another diagnosis, such as pneumonia, although wheeze is rare in children with bacterial pneumonia.

Bronchiolitis is the most common cause of wheezing in children aged one to six months.[1] By age ten months, the incidence of bronchiolitis is much lower, and it is rare after age one year.[1]

For further information on bronchiolitis, see: "Bronchiolitis in infants", BPJ 46 (Sep, 2012).

### Episodic viral wheeze

Episodic viral wheeze, also referred to as non-atopic wheeze, is wheezing associated with viral upper respiratory tract infections (URTI).[2] Children with episodic viral wheeze do not usually display respiratory symptoms between episodes of viral infection. The most common causative viruses include rhinovirus, coronavirus, human metapneumovirus, parainfluenza virus and adenovirus.[2]

Symptoms include acute wheeze and dyspnoea, usually with cough, shortly after the onset of an upper respiratory illness. Children with acute viral wheeze are unlikely to have chest crackles, as seen in children with bronchiolitis. In addition, bronchiolitis is usually a single episode of acute disease, compared to recurrent infections with viral wheeze.

Episodic viral wheeze is most common in children from age ten months up until age three years.[1] Children who develop wheeze as a symptom of viral infection will usually have fewer episodes over time,[2] and most children with wheeze without concurrent atopy will grow out of their symptoms by, or soon after, school age. However, some children with episodic viral wheeze will go on to have a confirmed diagnosis of asthma.

### Atopic wheeze

Atopic wheeze, or multiple-trigger wheeze, is recurrent/persistent wheeze, associated with atopic features and multiple exacerbating factors, e.g. cold air, night time, exercise or allergen exposure.[2] Symptoms occur when the child does not have a viral illness, and more severe exacerbations will be present when the child has a viral illness.[2]

Children with atopic wheeze are typically found to have bilateral, widespread wheeze and/or rhonchi, that are most prominent on exhalation, alongside cough, dyspnoea, prolonged expiration, increased respiratory rate and chest tightness.[1,7] A child with recurrent wheeze with signs of atopy or eczema, positive skin-prick tests or a family history of asthma or atopy can be considered to have atopic wheeze.[1]

Atopic wheeze is unusual in a child aged under two years (although it does occur), but becomes the dominant form of wheeze after age three years.[1] In practice, it appears that almost all children with atopic wheeze go on to be diagnosed with asthma after reaching school-age. Occasionally, a child who initially has wheeze that only occurs with viral respiratory infections will develop interval symptoms over time and wheeze in response to other triggers, before eventually being diagnosed as having asthma later in childhood.

## Assessing a child with wheeze

**The history** is the most important aspect of the assessment of wheeze in a young child.[2] It is important to describe wheeze to the parent/caregiver and check that this fits their description of the child's symptoms. Most people use the word "wheeze" to describe a wide range of audible breath sounds, however, the clinical definition is specific – a high-pitched, musical or whistling sound coming from the chest.[1]

Enquire about:

- The nature and duration of the wheeze, including whether it is present constantly or intermittently
- The presence of other respiratory symptoms
- Exacerbating factors and triggers
- Previous episodes
- The smoking status of the household
- Whether the child has ever had eczema or other symptoms or signs of atopy
- Whether there is a family history of atopy

**The physical examination** is primarily used to help identify potentially serious causes of wheeze. Ideally, the child's wheeze should be assessed during the examination to confirm that it fits the clinical definition of wheeze, but this will not always be possible.[2]

The examination should include a general assessment of the child, including respiratory rate, heart rate, temperature and oxygen saturation (if pulse oximetery is available). In a child with acute wheeze, the examination should assess whether concurrent upper respiratory infection is present, e.g. otitis media or pharyngitis.[8] Observe the child's chest to assess for signs of hyperinflation and respiratory distress, e.g. intercostal in-drawing and accessory muscle use. Perform auscultation of the child's chest and note any wheeze or crackles and whether there are focal sounds.

**Laboratory and respiratory investigations** are generally not used for assessing wheeze in pre-school aged children.[2] Further investigation, such as chest x-ray, is generally reserved for children in whom symptoms have been present since birth

or for children with wheeze that is unusually severe, does not respond to a trial of treatment or is accompanied by unusual clinical features.[2]

Peak-flow, spirometry and other assessments of lung function are generally not used in children aged under five years as they cannot provide a reliable, consistent result between tests.[7]

## The management of wheeze in children

The management of pre-school children with wheeze should begin with a clear discussion with the parents/caregivers about the likely prognosis of the child's illness and limitations of treatment. Explain that the diagnosis will usually become clearer with time and that pharmacological treatment can be used to relieve symptoms, but does not alter the natural history of the child's wheeze, nor prevent the development of asthma.[1–3] Regular re-evaluation of the child's symptoms will be necessary as the type of wheeze can change over time, before age five years.

### Lifestyle interventions for preventing exacerbations

A young child presenting with wheeze provides a good opportunity to encourage all adults in the household to stop smoking. Give smoking cessation advice and support where necessary and record the smoking status of family members. Maternal smoking during pregnancy should also be strongly discouraged.

Exacerbating factors, such as damp housing and inadequate heating in winter, should be discussed and parents assisted with solutions where possible, as these have been found to reduce childhood respiratory symptoms.

Infection prevention strategies should be discussed, particularly for children with episodic viral wheeze. Children should be up to date with their immunisation schedule and receive the influenza vaccine each year. Regular hand washing and good hygiene practices should be encouraged to avoid transmission of infections in the household or daycare environments.

Allergen avoidance has long been discussed as an early intervention for wheeze and asthma. However, there appears to be limited benefit to attempting allergy avoidance and the intervention can be difficult and costly.[9]

For further information on smoking cessation, see: "Encouraging smoke-free pregnancies: the role of primary care", BPJ 50 (Feb, 2013) and "Update on smoking cessation", BPJ 33 (Dec, 2010).

### Treating acute episodes of wheeze

**Bronchodilators**

Infants with **bronchiolitis** should not generally be treated with bronchodilators, as they provide minimal benefit.[3]

Children aged under five years with **episodic viral** or **atopic wheeze** can be trialled on a short-acting bronchodilator for symptomatic control.[3]

**Where required, bronchodilator treatment should be with a short-acting beta-agonist (SABA).[2, 3] Salbutamol, 100 micrograms, as required, to a maximum of 800 micrograms per day, is recommended for children.**[10]

This should be given by inhalation using a spacer and mask. Instruction on proper use and cleaning of the device should be given.

Long-acting beta-agonists (LABAs), while potentially effective for young children with wheeze, are generally not recommended as there are few strong studies in young children illustrating their benefit or safety.[2]

Theophylline is not recommended for use in children with wheeze or asthma.[11]

**Oral corticosteroids**

In a child with acute severe wheeze requiring hospitalisation, oral corticosteroids are recommended, and may be given while awaiting transfer. In a child with acute severe wheeze, who does not require hospitalisation, the use of oral corticosteroids is less clear, and should be based on clinical judgement. Evidence of the efficacy of oral corticosteroids in children aged under five years is limited and often conflicting, and most studies focus on older children.[2, 3, 12]

**If required, oral prednisolone can be given at 1 – 2 mg/kg per day, up to maximum of 40 mg, for three days.**[10]

The practice of giving parents a "back-pocket" prescription for corticosteroids is not recommended in pre-school children, as it has not been shown to prevent exacerbations or hospital admission in this age group.[2, 3]

Oral corticosteroids are associated with a range of adverse effects when used for short periods, including appetite, mood and behaviour changes. When used for longer periods (more than three months) adverse effects can be severe, including reduced growth, changes to skin, muscle weakness, Cushing's syndrome, bone weakening and increased risk of diabetes.[10]

## Preventing symptoms between episodes

**Inhaled corticosteroids**

In children with **atopic wheeze** who have symptoms between viral episodes, consider the use of inhaled corticosteroids (ICS).[2]

In children with **episodic viral wheeze** treatment with ICS is less effective and is not commonly recommended.[2]

Treatment with ICS in children aged under five years with wheeze is for symptom control only, and has no effect on the long-term natural history of the condition, and does not reduce the likelihood that a child will develop persistent wheeze or progress to asthma over time.[1–3] The response to treatment with ICS in younger children is usually less than that seen in older children.[2]

**The recommended ICS in children aged under five years is fluticasone 50 – 100 micrograms, twice daily, via a spacer and mask device, for up to three months.**[10]

The ICS should be stopped (after tapering) rather than just reduced, once interval symptoms resolve. Short-term treatment with ICS is as effective as continuous use in pre-school aged children, and may limit adverse effects.[13]

The use of corticosteroids in young children may cause several adverse effects. The most significant is reduced height growth, with studies finding approximately 1 cm less height (which may persist) in children treated with ICS for two years compared to placebo.[2] Adrenal suppression has also been observed in children taking ICS,[2] but impairment on adrenocorticotropic hormone (ACTH) stimulation tests may be more common than currently recognised.

## Montelukast has a role in managing episodic and atopic wheeze

Montelukast, a leukotriene receptor antagonist, is an appropriate treatment for symptom and exacerbation control in children with **wheeze of any type**. The medicine is currently subject to Special Authority criteria for subsidy (see: "Montelukast Special Authority has changed"). The Special Authority criteria allows for use in children with intermittent wheezing, which is currently an "off-label" use.

In pre-school aged children with wheeze, continuous use of montelukast appears to moderately reduce episodes of wheeze, and intermittent use, when the first signs of an upper respiratory tract infection appear, may help control symptoms and reduce the number of visits to primary care.[2]

## Montelukast Special Authority has changed

In November, 2013, the Special Authority requirements for subsidy of montelukast were updated. The changes occurred due to feedback that the previous requirements, particularly the requirement that exacerbations be severe enough to require hospitalisation, were too restrictive.

The Special Authority may be applied for by any relevant practitioner. For the initial application for managing wheeze in children aged under five years, both of the following criteria must be met:

1. Montelukast is to be used for the treatment of intermittent severe wheezing (possibly viral)
2. The patient has had at least three episodes of acute wheeze in the previous 12 months severe enough to seek medical attention

Renewal can be from any relevant practitioner. Approvals are valid for two years where the treatment remains appropriate and the patient is benefitting from treatment.

Montelukast may be used alone for preventing and managing exacerbations of wheeze, or can be used alongside ICS, to avoid having to increase the dose of ICS for effectiveness. It is available in a chewable tablet. The recommended dose of montelukast in children aged two to five years is 4 mg, once daily.[10] The ideal duration of treatment is unclear. Twelve months of continuous treatment appears to be effective in preventing exacerbations and controlling interval symptoms. However, short-term dosing, such as seven day cycles initiated by the parent or caregiver when symptoms occur, are also effective at controlling exacerbations and episodes and may reduce the overall combined dose of the medicine.[3]

No clinically relevant adverse effects have been reported in children taking montelukast.[2]

**ACKNOWLEDGEMENT:** Thank you to **Dr Philip Pattemore**, Associate Professor of Paediatrics, University of Otago, Christchurch for expert review of this article.

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# Prescribing update:
# Low dose isotretinoin for acne?

New evidence is increasingly suggesting that isotretinoin may be best prescribed using a lower daily dose, with the regimen tailored to the individual patient, the severity of their acne and their response to the medicine.[1–6] Low-dose isotretinoin appears to be as effective as higher doses for resolving acne, and may present a safer, more patient-centred approach to prescribing a medicine that is associated with significant adverse effects, e.g. photosensitivity, liver abnormalities and eczema.[3]

Current guidelines recommend that isotretinoin treatment is calculated based on body weight, usually 0.5 – 1 mg/kg per day, prescribed for long enough to reach a cumulative dose of approximately 150 mg/kg.[7, 8]

Based on this newer evidence, a suggested regimen would be to initiate isotretinoin at a dose of 10 – 20 mg/day, continued until all acne lesions have resolved,[3] which generally occurs between three to five months.[2] Treatment would then continue for a further two to four months to reduce the risk of relapse and help with resolution of acne scarring.[3] This second stage of treatment might be at a further reduced dose, e.g. 5 – 10 mg per day.[3]

## If what we do now works, why change?

There are several problems with prescribing isotretinoin based on a high daily dose to reach a cumulative total amount, including:[3]

- There is no clinical difference in effectiveness between high and low daily doses
- Adverse effects are more significant at higher doses
- There is limited evidence for directing treatment based on a cumulative dose
- Treatment duration is not based on the patient's response to the medicine
- Cumulative, weight-based dosing can be difficult to calculate and monitor

## How is isotretinoin currently being prescribed in New Zealand?

Current national dispensing data shows that there are two "peaks" of isotretinoin doses being prescribed. At present, 63% of people taking isotretinoin are dispensed 10 – 20 mg per day, and 22% are dispensed 80 – 90 mg per day, with the remainder prescribed intermediate doses.[9] It is difficult to conclude whether the 10 – 20 mg peak already represents low dose prescribing or whether it represents the traditional weight-based prescribing, but using lower doses for longer in response to adverse effects. It is likely that the higher dose group represents traditional weight-based prescribing. The current total average daily dose of isotretinoin is 42 mg.[9]

**Low doses are as effective as high doses**

Research has shown that isotretinoin at doses of 0.1 mg/kg per day is as effective as doses of 1 mg/kg per day in terms of acne clearance.[2, 3] A recent study found that acne clearance rates were between 92 – 95% in people taking isotretinoin 20 mg per day for six months (equivalent to 0.28 mg/kg per day, with a cumulative dose of 52 mg/kg).[1] This is comparable to the rate of clearance achieved with a traditional regimen of 0.5 – 1 mg/kg per day, with a cumulative dose of 150 mg/kg.[1]

**Adverse effects increase with increasing dose**

Adverse effects of isotretinoin are dose dependent and become more common, and more severe, with higher doses.[1, 3] At 1 mg/kg per day, 98% of patients report adverse events, such as eczema, impetigo and photosensitivity, while at doses below 0.25 mg/kg per day, 50% of patients report adverse effects, which are generally less severe.[3]

**There is little evidence to support cumulative dosing**

The duration of treatment with isotretinoin is currently based on the calculated cumulative dose. This method is used because several early studies suggested that relapse one to two years after a single 16-week course of isotretinoin was more common in people treated with 0.1 mg/kg per day than those treated with 1 mg/kg per day.[3] This was interpreted to mean that the strongest long-term response from isotretinoin was obtained if the cumulative dose reached 120 – 140 mg/kg.[3]

Subsequent research, however, has not supported cumulative dosing.[3] Long-term follow-up studies show rates of relapse between 40 – 52% several years after treatment.[3] These studies have concluded that relapse risk is determined by age, severity of acne and seborrhoea after treatment, but not by daily dose, duration of treatment or cumulative dose.[3]

**Duration should be based on patient response**

There are no studies that have specifically assessed the most appropriate duration of treatment to clear acne.[3] In practice, based on recent research and opinion, isotretinoin is continued until acne has cleared (defined as no active acne lesions), and then for another three to four months to limit recurrence.[1, 3] This approach tends to result in a shorter duration of isotretinoin treatment than with most cumulative dosing regimens,[3] while maximising patient outcomes and minimising adverse reactions.

**ACKNOWLEDGEMENT:** Thank you to **Dr Amanda Oakley**, Dermatologist and Clinical Associate Professor, Tristram Clinic, Hamilton for expert review of this article.

## Changes to the *bestpractice* Decision Support module

The current Special Authority criteria for prescribing subsidised isotretinoin recommend that a computer-based decision support tool is used when initiating and renewing the medicine. The *bestpractice* Decision Support Module for prescribing isotretinoin has recently been updated to reflect the new research which shows that lower doses are appropriate.

The module now recommends that the dose of isotretinoin should be based on the patient's response to treatment and not on a cumulative dose:

> "Use a starting dose of 10 – 20 mg and continue until there is a resolution of active acne lesions. Treatment dosages can then be halved and continued for a further two to four months".

The default isotretinoin capsule dose in the Decision Support module is now 10 mg.

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# Risedronate now fully subsidised:

## What is its place in practice?

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# Hazardous Substances

## Hazardous Substances Disease & Injury Notification

The Hazardous Substances & Lead Notifications reporting form is a new electronic notification system designed by BPAC Inc for general practices to report incidents related to exposures to hazardous substances.

A hazardous substance is anything that can explode, catch fire, oxidise, corrode or be toxic to humans, as defined in the *Hazardous Substances and New Organisms Act 1996*. The Act requires medical practitioners to notify cases of injury or disease caused by exposure to a hazardous substance to the Medical Officer of Health.

The form is available to health professionals at no cost, funded by the Ministry of Health.

**bestpractice**
DECISION SUPPORT FOR HEALTH PROFESSIONALS

# Beating the blues

*Contributed by: Dr Fiona Bolden, General Practitioner*

"Beating the Blues" is a web-based cognitive behavioural programme for people with symptoms of mild or mild-moderate depression, with good social support. They may have some degree of disruption to daily function as well as possible sleep and mood disturbance. If depression scoring tools are used, this would be equivalent to a patient with a PHQ-9 score of 10 – 14 or a Kessler 10 score of <16.

Once the General Practitioner has established that the patient has mild or mild-moderate depression, they can then:

- Give general advice and support
- Refer them to other services as appropriate, e.g. budget/ employment services
- Give them written information about depression or referral to a recommended reading list
- Refer them to online resources such as: **www.depression.org.nz**
- Refer them to Beating the Blues

In order to participate in the Beating the Blues programme, the patient needs access to a computer, the internet and an email address. They also need to be able to commit 50 minutes a week to doing the programme. There are eight sessions in the programme and patients can leave the programme at anytime; most people do not complete all eight sessions.

The *bestpractice* Decision Support depression modules contain links to Beating the Blues. The Beating the Blues programme is accessed via "Manage My Health".

Patients who do not have the resources available to them to participate in the programme, patients with less social support or patients with a higher degree of disorder of their mental health can be referred to a counsellor, psychologist or primary mental health coordinator for additional support.

For further information, visit: **www.beatingtheblues.co.nz**

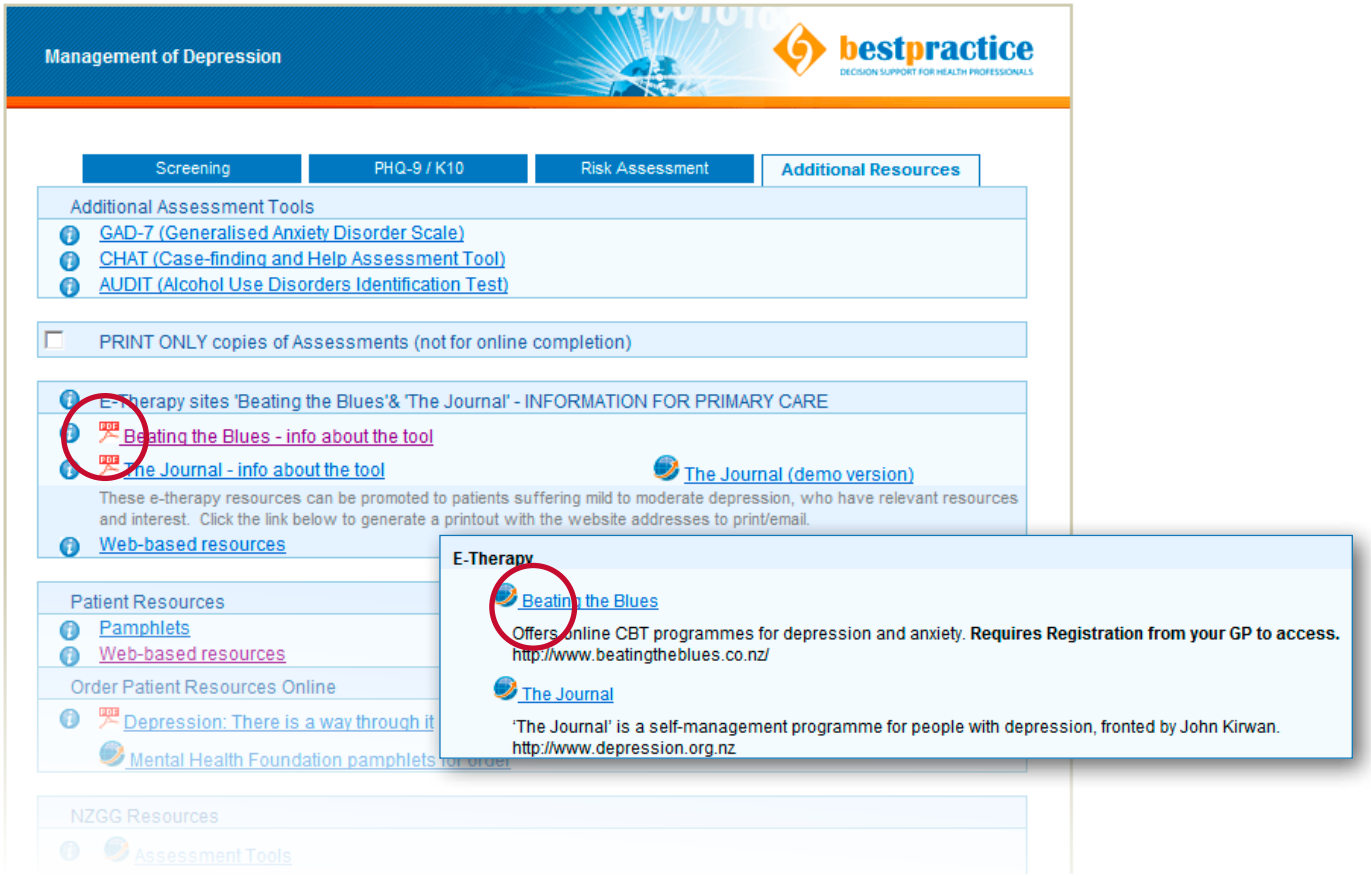
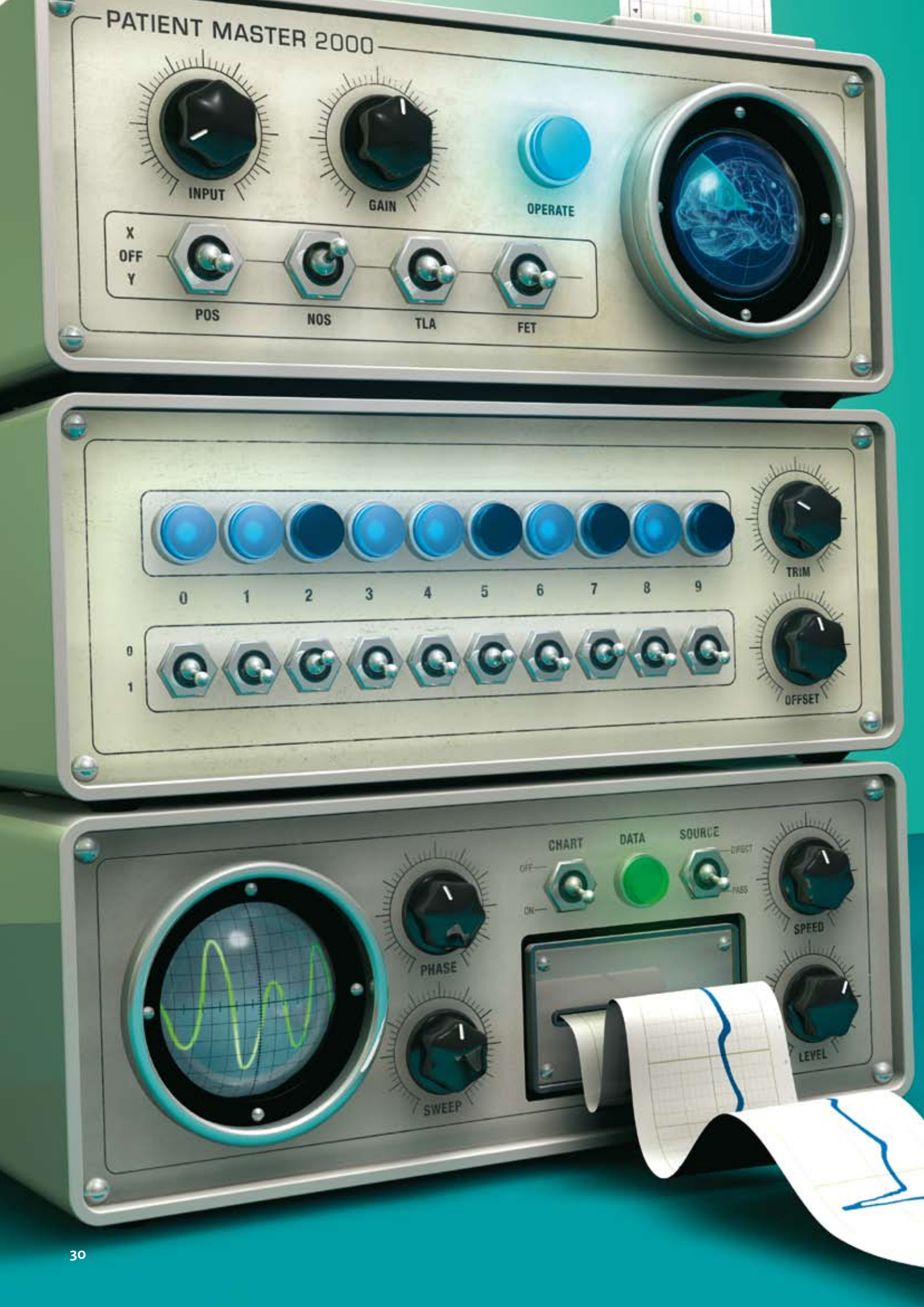



**Figure 1:** The *bestpractice* Decision Support depression module showing links to Beating the Blues.


PATIENT MASTER 2000
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SOURCE
OFF
ON
DIRECT
PASS
SPEED
PHASE
LEVEL
SWEEP

# 5 tips for getting the most out of your Practice Management System

Your Practice Management System (PMS) is a powerful tool. When used well, consistently and linked to decision support software, it can enhance understanding of a practice population's health and help to improve patient outcomes. Once a practice has quality information about its population, targeted care and monitoring can help improve the services offered by the practice, increase quality of care and reduce health disparities. Your PMS is also the best tool you have to help you meet the goals of the current PHO Performance Programme (PPP).

These goals are to:

- Improve the health of people enrolled in general practices
- Reduce inequalities in health outcomes for high need populations

Achieving PPP targets and having an information plan for the practice supports clinical governance, assists patients at an individual level and can be financially rewarding to primary care when targets are met across the PHO as a whole.

A good example of what the PPP has achieved is illustrated in the cardiovascular disease (CVD) risk assessment indicator. Since the CVD risk assessment indicator was introduced to the programme in 2008, assessment rates for the high need and total population have risen from under 20% to over 67% as of June, 2013.[1] From a population perspective we now have a very good understanding of cardiovascular risk within our practices and how this relates to New Zealand as a whole.

So what are the top five things you need to make sure you and your practice team are doing to get the most out of your PMS?

## 1. Make sure your demographics are correct

Ensure that the information you have about your practice population is accurate. Check that all of your patients are identified correctly if they are part of the high need population, which comprises Māori and Pacific peoples and people living in the lowest (NZDep deciles 9 and 10 or quintile 5) socioeconomic areas. To capture this information your PMS needs to have recognised ethnicity codes linked to patient demographic information. To ensure your population has the appropriate deprivation index the geo-coding functionality needs to be up to date and working correctly (see: Best Practice Tip). This is important as the high need population accounts for a large part of the funding that your PHO receives.

Ask patients what ethnicity they identify with when they present for a consultation. Some patients may be reluctant to indicate a preference but explaining the implications may help, i.e. that ethnicity is an important piece of health information that impacts on clinical decisions and helps to address specific health needs.

**Best Practice Tip:** To confirm your demographic coding is operating, create a new patient record and then check if dwelling location and ethnicity coding options are automatically displayed.

Recognised ethnicity codes are available from: **www.dhbsharedservices.health.nz/Site/SIG/pho/Technical-Documents.aspx**

## 2. Know the targets and what data is required to meet them

The correct data needs to be exported from your PMS to your PHO in order to assess performance against PPP targets. Therefore it is important that you know what the PPP targets are and what data the targets require. There should be a staff member within the practice who is responsible for ensuring that the correct data is being entered into the PMS. This helps to guarantee that the effort and clinical expertise of the team is reflected in your PPP performance. It also means that the practice receives all the funding that it's entitled to. The PPP targets, and the data that your PHO requires to meet them, is outlined in the document "Indicator definitions for PHOs". An article in support of the PPP is also published in each edition of Best Practice Journal (indicated by the PPP logo).

To access the latest version of the indicator definitions for PHOs, see: **www.dhbsharedservices.health.nz/Site/SIG/pho/Operational-Documents.aspx**

## 3. Right data, right format, right place...

The coding of clinical information, using diagnostic Read codes, ensures that the recording of health information is the same across the health care system, and that this information can be used for improving funding decisions and research.

It is also important to be aware of the codes that are used for the PPP. Recording patient outcomes and co-morbidities is an important factor for the diabetes and ischaemic heart disease indicators. When the wrong codes are used or misused your practice's performance will be inaccurate and you can miss out on achieving the targets.

All practices should develop a set of commonly used, recognised codes for their PMS from the PPP data format standard document (see below). These should be used by all clinicians within your practice.

**Best Practice Tip:** Run a query in your PMS to check that the codes you use in your PMS are approved for the purposes of PPP. If there are unapproved codes being used some systems allow mapping of local codes to approved codes for PPP purposes.

Recognised Read codes for the purposes of PPP can be obtained from the document "Code mappings for data transfer specification and clinical performance indicator data format standard document" available from: **www.dhbsharedservices.health.nz/Site/SIG/pho/Technical-Documents.aspx**

The data required for the PPP indicators is stored in a number of different areas within the PMS. The PPP collects practice-based data via an extraction process. To ensure that all the data the practice records contributes to the PPP target, it is important that the data extractor is mapped to all sources of the information. For example, in MedTech smoking status can be incorrectly linked to cardiovascular risk assessment, i.e. a screening code linked to an incorrect PPP code, potentially leading to an incorrect assessment of your practice's performance. It is important to check that the extractor mechanism has been mapped correctly to extract all the information stored within your system.

To ensure that your PMS's data extractor is set-up correctly contact your local PHO.

## 4. Implement practice-wide processes

To get the most out of your PMS it is important that everyone in the practice is using it the same way. Good practice-wide processes help to structure tasks across the practice. Identify key team members who have the responsibility for ensuring that patients are registered properly and have an identified geocode, and that the practice is receiving funding for the patients. A process should also be put in place to ensure that patients who have not been seen in the last three years, and are about to be removed from the practice register, are contacted and offered an assessment. Patients who are due for an intervention, e.g. a CVD risk assessment or cervical smear, should be recalled and followed up if they do not respond initially. Investigation results, such as mammography reports, should be captured in the system so that recalls are automatically generated for follow-up. Together, such processes should increase the PPP achievement rate for the practice.

As an example of a good practice-wide process, set a policy for clinicians to ask everyone who comes to the practice about their smoking status and record the information so that it can be captured by the PPP. This can dramatically improve the "smoking status recorded" indicator. This could be introduced as a team goal. For example, review the number of times each staff member records a smoking status or a CVD Risk assessment. The clinician who has achieved the most can then be rewarded at the end of each month. This approach creates a healthy competition within the team to achieve better recording of information.

## 5. Make use of electronic support tools

Support tools are usually "add-on" applications that are integrated into the PMS. Their main purpose is to ensure best practice is followed and any missing data is collected. These tools help practices to maximise the benefits of their PMS.

Support tools have three main functions:

1. Dashboard or patient prompt tools
2. Electronic decision support tools
3. Reporting applications

When all of these support tools are set-up to work together, consistency occurs with coding and the storing of information within primary care systems. The data contained in the PMS then has greater meaning and ensures more accurate population information. Clinicians can then focus on clinical issues and not be concerned about how to record and check that the information for the PPP is accurate and retrievable.

When PHOs implement these integrated tools across their regions a greater understanding of population health and practice performance is captured. Practices implementing good processes can be identified and their methods can be shared with other practices to adopt the successful approaches.

### Dashboard or patient prompt tools

When the patient's file is opened on the PMS the prompting tools assess the information within the systems and look for missing information, such as smoking status, or whether previously collected information is now out of date, such as looking for a cardiovascular risk within the last five years or a cervical smear in the last three years. These tools are becoming increasingly more sophisticated and can now highlight progressive chronic kidney disease and integrate with electronic decision support tools and enable the use of electronic health pathways and smart referrals.

### Electronic decision support

Decision support tools are integrated into your PMS to help facilitate cardiovascular risk and diabetes assessments. They have evolved from simple data collection tools for administrative purposes into key solutions for providing best practice guidance on managing cardiovascular disease, diabetes, chronic kidney disease and many other long-term conditions. Decision support applications are enhancing the role of primary care.

### Reporting applications

Population reporting applications give practices and PHOs an understanding of disease prevalence and associated co-morbidities. They provide information on how practices are performing in relation to the PPP and to other practices. The ability of the reporting applications to receive data on a daily basis helps practices to reflect more accurately on their achievements and to visualise their improvement when they change or introduce new processes into their practice.

Practices can clearly identify patients who have not received an intervention or require review. Using the information within these reports allows practices to schedule appointments for patient with long-term conditions and target care for those at greatest risk.

**Reference**

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# Common coding conundrums

Coding of health information is a bit like marmite; you either love it or hate it! Coding systems can be complex, and poor understanding of how coding systems are structured can lead to errors. Conversely, improved coding leads to more accurate reporting of health information, more efficient practice systems and, in the case of the PPP, improves your performance and funding.

Coding of information allows for the standardisation of clinical terms, e.g. the terms "heart attack" and "acute myocardial infarction" are linked by the mapping of these two terms to a single code root: in the Read thesaurus this is G30. The different terms, heart attack and acute myocardial infarction, are extensions of the code G30. Extensions or synonyms are defined by the numbers after the period as illustrated below:

G30.14 Heart Attack

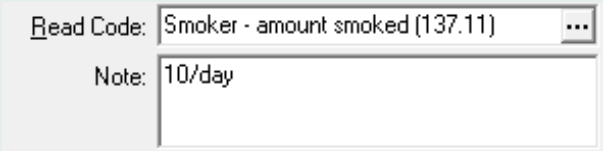
G30.00 Acute Myocardial Infarction

The linking of these medical terms provides uniformity through the G30 Read code. Uniformity then allows for data to be collated and used for evaluation of care, reporting, statistics, funding, planning and clinical research.

However, problems can arise when coding is not well understood. Common issues that occur in practice include:

## 1. Codes are used that are not recognised by the PPP

For example, the smoking code 137.00 – Tobacco consumption is not part of the code used to identify smoking status. The synonym of the code 137, 137.11, is called "Smoker – amount smoked". This is frequently recorded in practice as follows:



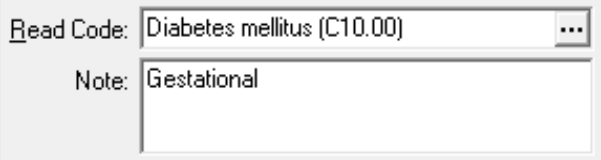

This code is not extracted for the PPP so will not be recognised. The other mistake is the use of the note section to change the meaning or reference of the code being used. A more appropriate code would be to use:

1374.00 Moderate smoker – 10 –19 cigarettes/day

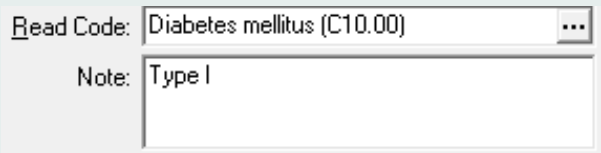
## 2. Changing the meaning of the code by adding text to the note section

If the note section is used in an attempt to change the meaning of the code, the addition is lost when the code is interpreted and aggregated by a computer. This will lead to unreliable reports and performance.

For example:




Or




In the first example the patient will be included in the PPP reports, but should not be as people with gestational diabetes are excluded from the PPP.

In the second example there will be no affect on the PPP reports, but there would be no ability to differentiate between two different types of diabetes with two different pathophysiologies if all patients with diabetes were coded with the C10 code and only identified by type within the note section.

A more precise approach which would lead to an improved understanding of population health and more accurate PPP reports would be to use the following codes:

| | |
|---|---|
| L1809 | Gestational diabetes |
| C108 | Type I diabetes mellitus |
| C109 | Type II diabetes mellitus |

### 3. The over-use of local codes

Some practices have devised their own codes when the current coding system does not match their needs. In the example below, at first glance everything looks fine, the term clearly refers to an individual with type I diabetes, but the code linked to the term is a made up local code and will not be recognised by the PPP.


Local codes are best avoided unless they have been adopted nationally as in the case of the new 'ZP' smoking codes.

**ACKNOWLEDGEMENT:** Thank you to **Glen Knol**, Facilitator, *bestpractice* Decision Support, Dunedin for contributing to the development of this article.

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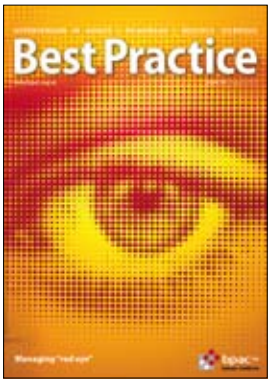



## Choice of medicines for hypertension

*Dear Editor*

*There were a few problems with the article: "Hypertension in adults: the silent killer", BPJ 54 (Aug, 2013). I usually find the bpac*[nz] *resources well written and evidence based. In this review there were a number of key errors:*

1. ***Start with an ACE inhibitor or calcium channel blocker.*** *Ironically there is data that neither of these medications are more effective than chlorthalidone – a thiazide-like diuretic: see ALLHAT study, JAMA 2002;288:2981-97, which found, albeit for the secondary but important outcome of combined cardiovascular disease, that chlorthalidone was more effective than lisinopril and amlodipine. However, it was pleasing to see that chlorthalidone and indapamide were mentioned in your article as they are the probably the best diuretics in New Zealand. Now that PHARMAC has driven down the price of ACEs, chlorthalidone is slightly more expensive than lisinopril but your article did not seem too focused on cost.*

2. ***The NICE guidelines suggest that those aged < 55 years should start with ACEs and those older start on a calcium channel blocker.*** *This does not make sense given the ALLHAT results. Also I did check the NICE guidelines in their earlier version (no references on the latest one) and the ABCD model of treating blood pressure was based on blood pressure lowering rather than hard outcomes.*

3. ***Don't give ACEs and ARBs together without the recommendation of a nephrologist or diabetologist.*** *I was bewildered by this statement. I am not sure what they would say beyond monitor the potassium and creatinine. Where does this notion come from?*

***Professor Bruce Arroll****, General Practitioner*
*Professor of General Practice and Primary Health Care, University of Auckland*

**Hypertension guidelines versus individual studies: Should we hang our hat on ALLHAT?**

Recommendations in Best Practice Journal are tailored to the needs of primary care health professionals by incorporating information from guidelines, and where necessary, adapting this to a New Zealand context. Naturally, this guidance will sometimes differ from conclusions that are based on individual studies. "Hypertension in Adults: The silent killer", BPJ 54 (Aug, 2013) was largely based on the United Kingdom National Institute of Health and Care Excellence (NICE) guidelines for the clinical management of primary hypertension in adults (2011).[1] The discrepancies highlighted between the recommendations in the Best Practice Journal article and the results of the Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) represent differences in clinical/expert opinion rather than "key errors".

1. We agree that the ALLHAT trial published in 2002 did not show evidence of superiority for thiazide-like diuretics over ACE inhibitors or calcium channel blockers. ALLHAT reported that all three medicines were equally effective in terms of primary outcomes.[2] However, ALLHAT has previously been criticised for not reflecting "real world practice".[3] Upon entering the trial, patients had all previous anti-hypertensive medicines withdrawn (including diuretics). Patients who were randomised to ACE inhibitor or calcium channel blocker treatment were then prevented by the protocol from receiving diuretic treatment, unless it was indicated by a definitive diagnosis of heart failure. Patients in the diuretic groups had no similar therapeutic restriction. The principle advantage of diuretics in treating hypertension, as reported from the ALLHAT study, was in reducing the risk of heart failure.[2] This is unsurprising given the design of the trial. Furthermore, the ALLHAT data showed a significant increase in adverse metabolic effects associated with the use of diuretics.[2] Therefore ALLHAT did not provide convincing evidence that diuretics should be used first-line in patients with diabetes, dyslipidaemia or gout, even if the metabolic adverse effects did not translate into an increased number of cardiovascular events. Also, one of the key considerations for ALLHAT investigators in recommending diuretics was their lower cost in comparison to other antihypertensive medicines; this is no longer the case.

2. ALLHAT excluded patients aged under 55 years and is therefore of limited use in helping to guide treatment decisions for hypertension in this patient group.[2] The recommendation to use calcium channel blockers to treat hypertension in patients aged over 55 years was based on the NICE guideline.[1] However, this "cut-off" should not replace clinical judgement and in some patients, e.g. in people with heart failure, treatment options include diuretics, ACE inhibitors and beta blockers. Furthermore, a "cut-off" approach to treatment was not central to the Best Practice Journal article recommendations. Instead, the article discussed the importance of hypertension alone, but also as part of the overall cardiovascular risk and the need for multiple medicines to achieve blood pressure targets. By highlighting a combination approach to treatment, the importance of which class of medicine to initiate first is reduced. This approach is strongly supported by recent European guidelines.[4] We acknowledge the important role that thiazide-like diuretics will continue to play in the reduction of cardiovascular risk and agree that chlortalidone (chlorthalidone) has a strong evidence-base for its effectiveness.

3. The recommendations in the Best Practice Journal concerning the combined use of ACE inhibitors and angiotensin II receptor blockers (ARBs) may require further clarification. A number of guidelines recommend against combining these two medicines for the treatment of hypertension due to an increased risk of complications, including patients developing end-stage renal disease.[1, 4, 5, 6] General Practitioners are therefore not recommended to combine ACE inhibitors and ARBs unless this has been recommended by a Nephrologist or Diabetologist, e.g. to reduce protein loss in patients with diabetic nephropathy. However, this indication is controversial. New Zealand Guidelines and Best Practice Journal recommendations acknowledge that this combination of medicines will rarely be initiated.[7] New Zealand guidelines also note that combination treatment with an ACE inhibitor and an ARB in people with chronic kidney disease is not currently supported by outcome evidence.[7]

We thank the correspondent for feedback on these points and acknowledge that the management of hypertension is a controversial area, and not all experts will agree with the recommendations. The goal of Best Practice Journal is to present clear, evidence-based and above all, practical, guidance for primary care clinicians to apply to their patient populations. As with all recommendations, management of individual patients may differ and clinical judgement must always be applied.

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## Use of dopamine agonists in restless legs syndrome

*Dear Editor,*

*I am a consistent "user" of the Best Practice Journal and New Zealand Formulary. I have used the material on restless legs syndrome (BPJ 49, Dec, 2012) and found it to be excellent for creating discussion within GP peer groups. Two consistent queries have come out of the discussion across the nine groups we have. The first is around schedules for tapering dopamine-like treatments and the second is around "intermittent" medicines use.*

*1. Tapering - it is clear that there is an increased risk of neuroleptic malignant syndrome (and rhabdomyolysis in the case of L-dopa) and that ropinirole, pramipexole and levodopa/carbidopa or*

*levodopa/benserazide should not be stopped abruptly. The BPJ article suggests tapering over one month but the GPs find that unhelpful unless there is a dosing schedule available (a table within the document would have been helpful). I have checked the data sheets for all of these products and only pramipexole provides a down titration schedule of any use. The New Zealand Formulary states abrupt cessation should not occur but again offers no recommendation of what a "down titration" schedule might look like for these products.*

*2. Linked to the above query, GPs were interested in the statement that intermittent use is possible. They were talking of PRN use and applying treatment in that fashion - as you would paracetamol for pain! I am sure that this is not what the authors are meaning; just that over time the severity of the RLS may change and so over a longer period of time use can fluctuate. Is this the case?*

***Dr Shane Scahill, PhD***
*Clinical Advisory Pharmacist*
*Auckland*

As the correspondent states, dopamine agonists should not be withdrawn abruptly due to the risk of potentially life-threatening neuroleptic malignant syndrome. This risk is higher in the context of withdrawing anti-Parkinsonian medications, particularly levodopa.[1] Recommended doses of dopamine agonists, such as pramipexole, are considerably lower for the treatment of restless legs syndrome than in Parkinson's disease. The National Prescribing Service (which publishes Australian Prescriber) suggests that at the doses typically used for restless legs syndrome, pramipexole can be stopped without tapering.[2] While at low doses (pramipexole ≤ 1 mg daily, ropinirole ≤3 mg daily) dopamine agonists can be abruptly discontinued, it is recommended that higher doses be halved and maintained for a week.[3] If this is tolerated, the medicine can be stopped. However, if withdrawal effects (anxiety, depression, irritability, orthostatic dizziness, diaphoresis) develop then the dose should be tapered gradually to zero over an extra two to three weeks.[3]

The practice of intermittent dopamine agonist use can be problematic if the patient is exposed to fluctuating dopamine levels, but it is recommended as a treatment strategy for restless legs. Pharmacological treatment options for restless legs syndrome is dominated by off-label prescribing of medicines, and treatment is driven by consensus rather than national guidelines. Again, there is clear distinction between the treatment of restless legs syndrome and Parkinson's disease. NICE specifically recommends against medication holidays in the treatment of Parkinson's disease,[4] whereas in the treatment of restless legs syndrome, they recommend that levodopa can be used intermittently when symptoms occur or in anticipation of symptoms for patients with intermittent symptoms (less than three times per week).[5] Levodopa with carbidopa has only a short duration of action of four to six hours.[6] The use of levodopa, pramipexole and ropinirole for intermittent symptoms is also supported by in North American[6] and European[7] recommendations. Therefore, to answer the second part of the correspondent's question, guidance does recommend that levodopa can be administered "as required" for restless legs syndrome.

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## A different take on restless legs and nocturnal cramp

*Dear Editor,*

*I found your articles on Restless Legs Syndrome and Nocturnal Leg Cramps (BPJ 49, Dec 2012) disappointing, because no clear causes were outlined and the treatment options were poor.*

*For the following reasons I believe the working hypothesis to use in our medical practice, as to the primary cause of both conditions, should be magnesium deficiency:*

1. *No other simple explanation has been proposed*
2. *Magnesium is an essential factor for the healthy function of nerves and muscles*
3. *Widespread soil mineral deficiencies and mineral losses in food preparation combine to make the magnesium intake inadequate for many people. The requirement for magnesium is large, with the human body needing about half the mass of its sodium requirement.*
4. *The statement that: "Magnesium supplementation has no benefit in the treatment of nocturnal cramps" is an inaccurate summation of the conclusions of the research quoted in the Cochrane review. Of the four studies referenced, only two were of published studies relating to oral supplementation. These studies by Frusso et al 1999 and Roffe et al 2002, both noted a significant period effect i.e. improvement in cramp occurrence with time as magnesium treatment continued. However all six oral studies quoted, including those in pregnancy, were flawed because they used too low a dose of magnesium and/or poorly absorbed magnesium, for too short a time. Any serious attempt to treat the symptoms of magnesium deficiency with oral supplementation to raise the total body magnesium content requires months of magnesium amino acid chelate (glycinate) or perhaps magnesium of marine origin, in a dose of at least 500 mg elemental magnesium per day if tolerated.*
5. *Anecdotal accounts of benefit of magnesium supplementation for both conditions are widespread*

*In a review of my practice database covering the last 11 years and eight months, 99 current adult patients with a Read code diagnosis of cramp (N2472.00) were found. Of these, 92 had received advice on the use of magnesium supplementation, and in subsequent consultations cramp had settled in 88. In four there was a reduced amount of cramp, and of the four patients who reported ongoing cramp, two were found to have been taking no magnesium. All eight patients over the last three months have received further advice to take a higher dose of a better absorbed magnesium preparation, and will be reviewed again in due course.*

*In the last year only two patients have been prescribed quinine. One was a short supply for a patient with severe cramp occurring in multiple sites, to use over the time needed for a magnesium supplement to take effect. The other and only patient requiring an ongoing supply has chosen to take the advice of a specialist who initiated the prescribing of quinine.*

*Diuretics increase renal loss of magnesium, and appear to increase the tendency to cramp and restless legs syndrome. Therefore in this practice in order for benefit from magnesium supplementation to not be sabotaged by diuretic action, the use of frusemide, bumetanide and thiazides is minimised and where possible replaced by spironolactone.*

*I have made the following observations in clinical practice and have assumed they are common knowledge, but they were also omitted from the restless legs article:*

1. *The sleep deprivation restless legs causes becomes in itself a major cause of the restless legs syndrome; i.e. it becomes self-perpetuating, with the increased fatigue from the inability to get to sleep increasing the restless legs condition the next night*
2. *The most effective acute management of restless legs is cooling, and in particular running cold water over the legs in the bath or shower*
3. *Much safer and cheaper medicines than those suggested in the article are effective in controlling restless legs, such as ¼ to 1 tablet of dihydrocodeine (DHC) 60 mg, each evening. Later, after magnesium supplementation takes effect, if needed restless legs may be controlled with paracetamol 500 mg plus codeine 8 mg tablets, or clonidine 25 – 50 mcg nocte.*

*Observations made in my clinical practice over decades have contributed to the above hypotheses and conclusions, and there should be research to confirm them. However there is a great deal of health knowledge which has been gained in general practice*

*by doctors listening carefully to what patients tell us, and this huge source of information and learning should not be ignored.*

***Dr Ralph Brock-Smith,*** *General Practitioner*
*Lower Hutt*

**Editorial comment:** There are few robust studies on the use of magnesium for nocturnal cramps or restless legs syndrome. Studies include only a small number of participants and have shown limited evidence of effectiveness. Reviews of the balance of evidence have concluded that magnesium is unable to be recommended as an effective treatment for nocturnal cramps or restless legs syndrome. The data reported by the correspondent undoubtedly demonstrates an association between patients taking magnesium and experiencing an improvement in their symptoms of cramp/restless legs. However, what this data does not definitively reveal is causality. The patients' symptoms may have remitted spontaneously over time, or because of other non-pharmacological interventions the patients may have undertaken. The debate, therefore, centres on whether giving magnesium to patients with nocturnal cramps or restless legs may cause harm. Magnesium is considered safe at doses no greater than the upper recommended level of intake for supplements of 350 mg/day for adults.* Adverse effects associated with excessive use of magnesium, i.e. hypermagnesaemia, include diarrhoea, nausea, vomiting and thirst, and in more serious cases, hypotension, arrhythmias and respiratory depression. Perhaps of greater concern are the limitations on the use of diuretic medicines in these patients. In addition, although some of the medicines recommended for unremitting restless legs syndrome and nocturnal cramps are associated with adverse effects, dihydrocodeine and clonidine are not without potentially significant adverse effects also.

* Australian Government Department of Health and Ageing, National Health and Medical Research Council. Nutrient reference values for Australia and New Zealand. 2006. Available from: www.health.govt.nz (Accessed Oct, 2013).

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