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"Seventh age itch": Preventing and managing dry skin in older people

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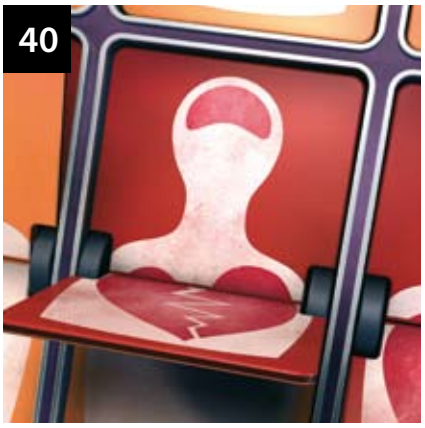
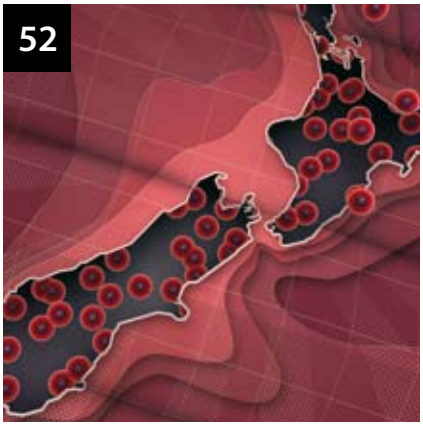
CONTENTS

Issue 63 September 2014

CONTENTS

Issue 63 September 2014





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Good IT Practices: Recommendations for good IT management for general practices

General practices are increasingly dependent on their IT systems for managing their patients and their business. Up to date and effective IT systems are an essential part of protecting electronic patient information against the risks of data loss or unauthorised access.

As other technology solutions move forward it is also important to ensure that the IT platforms that you operate are able to support new IT systems aimed at helping the practice to improve performance and patient outcomes. The following are some recommendations for good practice IT management.

1. Effective backups

- Effective and properly managed backups are the most important defence against loss of data that could result from many different issues, including hardware failure, computer virus infection or physical events such as fire
- Backup schedules should be performed at a minimum of daily (differential – where all changes since the previous backup are captured), weekly (full backup – where all data is captured) and end of month (full backup)
- These should be stored offsite in a secure location on a daily basis
- Backups should be validated to check that they have been successfully completed and tested at least once a month to ensure that they are valid and recoverable

2. Antivirus, security updates and firewalls

- A subscription based reputable anti-virus solution should be deployed (e.g. Symantec, ESET, McAfee) and each workstation should have a full scan scheduled on a weekly basis
- Updates (such as Windows Updates) should be regularly applied to ensure that systems are fully patched against vulnerabilities
- Use firewalls to protect your systems from unauthorised network traffic
- Use an email security system such as MailMarshal

3. Use of up to date IT hardware and operating systems

- All IT equipment should be kept current. Old equipment has a higher likelihood of failure.
- As a guide, any equipment older than five years should be considered for replacement
- IT operating systems (such as Windows Server or Windows for the PC) should be kept within current supported versions
- Microsoft XP support ended in April 2014 and Microsoft Server 2003 support will end in July 2015. Desktop and server computers using these operating systems will become vulnerable as no new security updates will be provided. Practices should be working with their IT provider on a plan for upgrading any computers using these operating systems.

4. Practice management systems – database management

- Appropriate database management should be undertaken for the practice management system (e.g. Medtech32) and the required maintenance schedule followed as prescribed by the relevant software vendor

5. Security, awareness and vigilance

- System access should be protected by robust passwords which are kept secure and regularly changed (at least every 90 days). Passwords should be at least 8 characters long (the longer, the better), contain a mixture of upper and lower case characters, numbers and symbols and should avoid words that could easily be associated with the user.
- Care should be taken with any suspicious emails, and particularly before opening any attachments included with suspicious emails. Suspect emails should be deleted.
- Practices should have policies in place to manage risks associated with copies of sensitive data held on portable media and devices (such as USB data sticks, mobile phones, tablets and laptops). At a minimum, access should be protected by passwords or PIN numbers and where possible, avoid copying sensitive data to these devices.

6. Trusted and reputable IT service providers

- Engage a trusted and reputable IT supplier, and ensure that your IT provider has the necessary experience
- All support agreements with IT providers should be in writing. The contract should be clear on what services they will provide and on what terms, the service levels that they are committing to and any exclusions that may be in the "fine print". Practices should be able to confirm that their IT provider actually complies with all terms of the contract.
- Any vendor delivering IT services for primary health that includes maintenance of Medtech32 should be MedTech or equivalent certified within the last two years

Encouraging **children** to **swallow tablets** or **capsules**

Many health professionals will encounter children who are reluctant or unable to swallow tablets and capsules. This can present a challenge when children require a particular medicine where limited formulation options are available. Simple techniques can be discussed with parents to help teach their child this skill.

Most school-aged children are able to learn to swallow tablets and capsules

In general, children are able to learn to swallow tablets or capsules from approximately age six years, and most master the technique on their own by age ten years. Some children, particularly those with chronic conditions requiring daily medicines, can be taught to swallow pills at a younger age.

Parents frequently report barriers for children learning to swallow tablets and capsules, however, these can often easily be overcome. For example:

Anxiety – Children who fear swallowing pills are likely to be tense when attempting to do so, therefore making the process more difficult. This tension, particularly in the throat, neck and chest can make the child feel like they are having trouble breathing which may, in itself, cause further anxiety. Learning strategies to swallow tablets and capsules effectively, and practicing these techniques, can help to reduce the child's anxiety and make them feel more relaxed, therefore increasing their chance of success. Children who are anxious about swallowing medicines often have parents who are also reluctant or unable to swallow medicines themselves. Therefore it is important that parents lead by example and demonstrate that swallowing pills or capsules is easy.

Strong gag reflex – Children who are fussy eaters or who gag frequently on food and drink can often struggle with swallowing medicines. Getting the child to take a deep breath before putting a tablet or capsule in their mouth can help them to suppress their gag reflex.

Texture, size and shape – The size and shape of the tablet or capsule, and the nature of the coating can affect the ease of swallowing. Using yoghurt or a thick drink, such as a milkshake, may help to reduce a child's awareness of a tablet or capsule being swallowed.

Techniques for swallowing tablets and capsules

There are many techniques for swallowing tablets and capsules, and it is appropriate for children to find one which works for them. In some circumstances, such as when a child needs to take ongoing medicines, it may be appropriate to train them to swallow tablets and capsules, e.g. using lollies (see: "Practice makes perfect", below). A study of 33 children aged 2 – 17 years with pill-swallowing difficulties found that after 14 days practice, all children were able to swallow tablets or capsules.[1]

In general, it is best not to throw a tablet or capsule towards the back of the mouth. This is because it can actually make swallowing more difficult.

A recommended technique for swallowing a pill or capsule is to:

1. Ask the child to have a drink of water or their favourite drink to moisten their mouth
2. Place the tablet or capsule into the centre of the child's mouth
3. Ask the child to take a big sip of their drink, and then swallow

Yoghurts and thick drinks, such as milkshakes, can help ease tablets or capsules down. Using a straw to drink, with a tablet or capsule already in the mouth, may also help by getting the child to concentrate on the suction of the straw rather than thinking about the tablet or capsule going down.

Another technique is to put the capsule into a small spoonful of apple sauce or ice cream. This can help capsules to slip down the throat more easily.

The physical properties of capsules may cause them to float in the mouth when taken with water. Leaning forward when swallowing can help the capsule go down.[2] This technique may not be comfortable for everyone, but some children may wish to try this:

1. Ask the child look down at the floor instead of up at the ceiling
2. Slip the capsule into the centre of the child's mouth.
3. Ask the child to take a big sip of their drink while still looking at the floor. The capsule should float to the back of the child's mouth and roll down their throat with the drink.

Other options for administering medicine

When children cannot yet swallow whole tablets or capsules, an option could be to crush a tablet or empty the contents of a capsule into food or drink. Some tablets or capsules can be compounded into a suspension, but there can be concerns about stability, bioavailability and dose accuracy.

This is not possible for all medicines, for example long-acting medicines and those with special coatings cannot be crushed or opened, and some medicines cannot be mixed with certain foods. These options must be checked with a Pharmacist first.

References

1. Kaplan BJ, Steiger RA, Pope J, et al. Successful treatment of pill-swallowing difficulties with head posture practice. Paediatr Child Health 2010;15:e1–5.
2. Medsafe. Helping medicine capsules go down. Prescriber Update 2003;24. Available from: www.medsafe.govt.nz (Accessed Aug, 2014)

Download this quick guide for clinicians and parents to encourage swallowing of pills and capsules in children. This guide can be printed out and used as a take-home resource. Available from: **www.bpac.org.nz/Supplement/2014/September/pillswallowing.aspx**

“Seventh age itch”:
Preventing and managing dry skin in older people

As skin ages, increased transepidermal water loss leads to dry skin (xerosis) and reduced barrier function. Dry skin is often itchy and prone to dermatitis. Repeated scratching can lead to chronic wounds and infections, particularly on the lower legs and especially if treatment is delayed. Older patients should be asked regularly about skin symptoms and periodically examined for signs of poor skin health. Encourage older patients to use emollients (which hydrate and soften the skin) and to avoid products which irritate the skin, e.g. standard soap, to improve skin health. If patients do experience skin rash or injury, e.g. skin tears, they should be advised to seek help for this early on to reduce the risk of complications developing.

Delaying skin deterioration in older patients: managing dry skin

As skin ages, the epidermis and dermis become thinner and flatter and the skin's mechanical strength declines.[1] There is a reduction in the number of cutaneous blood vessels and nerve endings, and in the amount of connective tissue, which contains collagen and elastin.[1] The skin has a decreased ability to retain moisture, to control temperature and to sense the surrounding environment.[1] Environmental factors, such as exposure to UV radiation, also have a detrimental effect on skin health over time.

Skin management in older people focuses on preventing or delaying damage and monitoring skin regularly. Prevention includes looking after the health of the skin and maintaining a balanced diet, with sufficient intake of protein, vitamins and fatty acids.[2] The ability of skin to regenerate once damaged is reduced in older people. Immune function also declines with age, therefore older people have an increased susceptibility to infection, e.g. in wounds caused by scratching.

For further information, see: "Skin and the biology of ageing", Page 14.

Identify dry skin early

Dry skin (xerosis) is a common dermatological feature in older people.[3] This is caused by water loss from the stratum corneum, and as a consequence the skin is more likely to crack, which can result in itching, bleeding and asteatotic dermatitis.[4] Troublesome dry skin is often under-reported and patients may wait many years before asking a health professional for advice.[4]

All older patients should have their skin periodically assessed for signs of excessive dryness. Possible causes of dry skin include:[3]

- Cold, dry air during winter
- Direct skin exposure to fan heaters
- Excessive immersion in water
- Use of alkaline soaps and detergents with ingredients that damage the skin barrier
- Chronic sun damage
- Papulosquamous skin conditions, e.g. eczema, psoriasis
- Chronic illnesses, e.g. hypothyroidism, chronic kidney disease (CKD)
- Genetic inheritance, e.g. ichythyosis vulgaris, characterised by persistently dry, thickened, rough skin
- Systemic disease, e.g. lymphoma, malnutrition, resulting in acquired ichythyosis

Excessively dry skin is often scaly or cracked. This is frequently seen on the legs in older people, where skin may have the appearance of cracked porcelain, referred to as eczema craquelé (Figure 1).

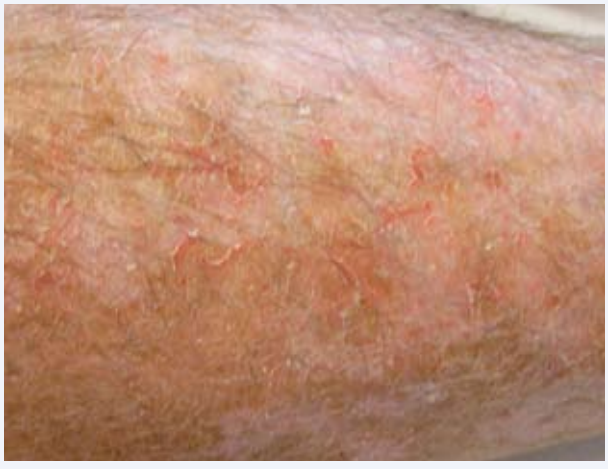


Figure 1: Eczema craquelé; characteristic "cracked porcelain" appearance of dry skin. Image provided by DermNet NZ.

Removing triggers for dry skin may reduce the need for treatment

Take a history from patients with dry skin to identify any obvious causes. Advise the patient to avoid soap and other alkaline cleansers and any topical products containing alcohol or fragrance. Wearing loose clothing and avoiding woollen fabrics may reduce skin irritation.

Reducing the time spent in the shower or bath, and avoiding very hot water decreases lipid loss from the skin and may limit skin irritation.

Emollients are used to treat dry skin

Patients with dry skin should apply emollients at least twice daily, ideally within minutes of washing the skin, and at other times if necessary.[1] These are available in a range of different formulations, including creams, ointments and lotions (Table 1).[1]

Emollients can hydrate the skin by two mechanisms:

1. Drawing water from the dermis to epidermis due to the presence of humectants, e.g. urea or glycerol
2. Preventing water from escaping with an oily layer, e.g. ointments

Emollients generally have a neutral or low pH which helps to maintain the barrier function of skin,[3] and are also reported to have anti-inflammatory properties.[4]

The choice of emollient is based on how dry the patient's skin is and their preference; patients are most likely to be adherent to a treatment if they are using a preparation they are comfortable with. A process of trial-and-error may be required, however, **an emollient with a 10% urea content is a reasonable starting point** (Table 1).[3] Some patients may report a mild stinging or itchy rash associated with the use of urea.[5] Emollients containing urea should not be used on areas of dermatitis or broken skin, on any area that is infected or where there is severe or widespread erythema.

Emollient products may need to be changed depending on the season, the patient's lifestyle and disease severity.[4] Patients who experience drier skin during winter may benefit from temporarily using an emollient with a thicker lipid film, e.g. an occlusive ointment. Due to their high oil content, ointments can be messy and may stain clothes, and therefore are best applied at night. Greasy emollients may lead to infected or irritant folliculitis. If this occurs, the patient should be switched to a less greasy product. Antibiotics may also be required if infection is present, e.g. flucloxacillin if *Staphylococcus aureus* is suspected.

A patient using a leave-on emollient can also be prescribed a wash-off emollient, e.g. aqueous cream BP or emulsifying ointment (see: "Aqueous cream or emulsifying ointment should not be used as leave-on products", Page 10).[4]

An evidence-based approach to skin care is important and it should not be assumed that all naturally occurring oils are beneficial to human skin. For example, olive oil has historically been recommended as an emollient for infants with dry skin. However, this practice has been shown in adults to be detrimental to skin hydration and to adversely affect skin integrity.[6]

Recommend non-soap based bath products

Soaps that contain lanolin and glycerine, or moisturising soaps, are less likely to cause skin flaking than standard products. However, it is best that patients with dermatitis avoid all soaps. In addition, the use of lanolin may cause allergic contact dermatitis in some patients.[7] Non-soap based bath and shower products are available on prescription (Table 1) and over-the-counter (OTC). Emollient bath additives, e.g. oatmeal colloidal (unsubsidised), can be added to bath water and skin hydration improved by soaking for 10 – 20 minutes.[8] Advise patients that bath and shower products, especially oils, can make surfaces slippery and care needs to be taken. Non-slip mats and/or the installation of a rail in the shower box or next to the bath, may help reduce the risk of falling.

Emollients should be prescribed in sufficient quantity to have an optimal effect, and the amount will vary depending on which areas of the body are affected (Table 2, over page).[4] If the patient spends a significant portion of their time away from home, an additional smaller-quantity pack will allow them to use the product during the day.

Fire hazard with paraffin-based emollients

Emulsifying ointment, petroleum jelly, or 50% liquid paraffin and 50% white soft paraffin, can be ignited when present on clothes or dressings. This risk is greatest when large areas of the body are being treated, or when dressing or bandages become soaked in the ointment. Patients should keep away from open fires and flames, e.g. candles, and be advised not to smoke when using these products.

Table 1: Emollient selection aide for subsidised products used for the treatment of dry skin in older patients*[8, 9]

		Type of skin product	Description	Product name/ components	Brand name and pack sizes	Comments
LEAVE ON EMOLLIENTS	Increasing severity of dry skin ↓	**Mineral oil lotion**	Light, non-greasy lotion	Lanolin (wool fat) with mineral oil 3% lotion hydrous	BK lotion, Alpha-Keri Lotion, DP lotion, Hydroderm Lotion 250 mL bottle, 1000 mL bottle	These products are only partially subsidised. Cost or part charge will depend on the product and quantity prescribed; check with the patient's pharmacy for an accurate cost. Allergic contact dermatitis reactions may occur in people sensitive to lanolin. This is more common in people who are atopic and may appear as intense swelling and redness from hours to one or two days after treatment initiation. Hydroderm lotion is to be delisted 1 Dec, 2014; the cost of the part charge for all mineral oil lotions may change at this time.
		Emollient/ simple moisturiser	Slightly greasy cream	Cetomacrogol cream BP†	Cetomacrogol (PSM) cream BP 500 g jar	Also known as non-ionic cream
		Emollient with humectants	Moderately greasy cream	Cetomacrogol with 10 % glycerol cream	Pharmacy Health Sorbolene with Glycerin 500 g jar, 1 kg jar	
			Moderately greasy cream	Urea Cream 10%	healthE Urea Cream 100 g tube	Do not use urea on broken, oozing or infected skin, or where there is severe widespread reddening of the skin.
		Occlusive emollient cream	Moderately greasy cream	Oil in water emulsion	healthE Fatty Cream 500 g jar	
		Occlusive emollient ointment	Very greasy ointment	Water-in-oil emulsion products available		Non-subsidised products are: healthE Liquid Paraffin in WSP, petroleum jelly (Vaseline) or Duoleum
WASH OFF EMOLLIENTS		**Bath and shower products (soap substitutes)**	Leave a film on the skin	Aqueous cream BP†	Aqueous Cream BP AFT 500 g jar	The subsidised products contain sodium lauryl sulphate and should not be left on the skin as this can be an irritant; some unsubsidised versions do not contain sodium lauryl sulphate and therefore may be left on the skin (see: "Aqueous cream BP should not be used as a leave-on product"). It is recommended that emulsifying ointment BP be added to bath water, initially using hot water to disperse before adjusting temperature of bath water. When showering a heaped teaspoon of emulsifying ointment can be dissolved into a jar of hot water and used as soap substitute.
				Emulsifying ointment BP†	Emulsifying Ointment BP AFT 500 g jar	

† Some products contain 'BP' in their name. This stands for British Pharmacopoeia, which is a detailed collection of standard specifications and formulae for compounded medicinal and pharmaceutical products.

* NOTE: the quantity of supply by the dispensing pharmacy will be the full three-month quantity prescribed, unless the prescriber endorses each skin product on the prescription clearly with the words "Trial Period" or "Trial", and specifies the maximum quantity to be dispensed at any one time; liaison with the patient's pharmacy is helpful.

Table 2: Recommended quantities of topical emollients to be prescribed for an adult, with twice daily application for one week to specific body areas[8]

Body area	Creams and ointments	Lotions
Face	15 – 30 g	100 mL
Both hands	25 – 50 g	200 mL
Scalp	50 – 100 g	200 mL
Both arms or both legs	100 – 200 g	200 mL
Trunk	400 g	500 mL
Groin and genitalia	15 – 25 g	100 mL

Emollients are best applied by dotting the product on the skin then spreading it using a downward stroking motion.[1] It is not necessary to spread the product until it is all absorbed.

A clean spatula should be used to extract the emollient from the tub so pathogens are less likely to be introduced into the preparation. Alternatively, the emollient can be decanted into a clean, empty pump dispenser. In patients who are older, obese or have arthritis the assistance of a partner or carer may be necessary when applying the emollient.

Emollients can be used in conjunction with other topical treatments. If an emollient and a topical corticosteroid are prescribed at the same time, e.g. for a patient with atopic dermatitis, the emollient should be prescribed and applied at ten times the quantity of the steroid.[4] The use of emollients in conjunction with topical corticosteroids can reduce the need for corticosteroid use without a loss of treatment efficacy (by improving skin health).[4] Ideally one product, i.e. emollient or corticosteroid, should be applied at least 30 minutes before the other; the order of application is not important.[4]

Cosmetic products containing fragrance and colour have no therapeutic value and should be avoided as these can alter skin pH, irritate, cause contact allergy and worsen dry skin.

Aqueous cream or emulsifying oinment should not be used as leave-on products

Aqueous cream BP first appeared in the British Pharmacopoeia in 1958 and its original formulation has been unchanged since. It was intended for use as an emollient wash product,[10] however, it has been frequently prescribed as a leave-on emollient for patients with atopic dermatitis in New Zealand and in other countries.[11] In 2009 it was reported that aqueous cream BP accounted for approximately one-quarter of all topical emollients prescribed in the United Kingdom.[11] A growing body of evidence now suggests that for some patients, aqueous cream BP should not be used as a leave on emollient and that doing so can cause significant damage to the skin barrier.

Aqueous cream BP is formulated using emulsifying ointment. Emulsifying ointment contains emulsifying wax, which in turn contains sodium lauryl sulphate (1% w/v in aqueous cream). Sodium lauryl sulphate is a surfactant used in a wide range of products due to its solubilising, wetting, suspension stabilising, emulsifying and frothing properties.[12] Sodium lauryl sulphate is known to increase the permeability of the skin barrier and is often used as a model irritant in experiments.[12]

Concerns about the possible adverse effects of aqueous cream BP were first noted in studies assessing children in which over half of patients reported a cutaneous reaction described as a "stinging" sensation when the product was applied.[12] Several small studies in adults have reinforced these concerns and led to a number of dermatologists recommending that aqueous cream BP should not be used as a leave-on emollient in patients with atopic dermatitis.[11]

In one study of six volunteers, with no history of skin disease, aqueous cream BP was applied to the forearm, twice daily for four weeks.[12] A significant reduction in skin thickness and an overall increase in transepidermal water loss was reported.[12] Another study involved 13 volunteers with a previous history of atopic dermatitis, but without current symptoms.[11] Again, aqueous cream BP was applied to the forearm, twice daily, for four weeks. Aqueous cream BP was described as causing severe damage to the skin barrier.[11] The minimum recommended doses were used in these studies and therefore more frequent use of aqueous cream BP, or use of the product by patients with more severe forms of dermatitis, may cause more extensive skin damage.

Aqueous cream and emulsifying ointment are therefore not recommended as leave-on emollients, but are still suitable as soap substitutes because in this situation, the product is washed off and is only in contact with the skin for a short time.

Managing dry, itchy skin in older patients

In older people, dry skin is frequently associated with pruritus due to the reduced lipid content in the skin, the decreased production of sweat and sebum, and diminished vascular perfusion.[3] Dry skin and pruritus commonly occur without a visible inflammatory rash and are sometimes referred to as "winter itch" or "seventh age itch". Scratching can lead to secondary dermatitis with dry scaly plaques (lichen simplex).

The patient's history is likely to contain clues to the source of their symptoms. Considerations for health professionals when discussing dry and itchy skin with a patient include:[3]

1. Are the patient's symptoms general or localised? Generalised skin symptoms are more suggestive of a systemic cause. Localised pruritus without a primary rash may be of neuropathic origin.
2. Has the patient noticed a rash? Itchy rashes are most often due to eczema, scabies, urticaria, and insect bites, however, there are many more possible diagnoses.
3. Is there any time of day when the symptoms are worse? For example, night time is often associated with worsening symptoms in patients with scabies.
4. Have any of the patient's family members or contacts developed similar symptoms that suggest an infectious condition, e.g. scabies.
5. Is the patient aware of anything that can improve their symptoms, e.g. a specific treatment?
6. Did the onset of the patient's symptoms coincide with the initiation of any medicines?
7. Could the patient's symptoms be related to gardening or another outdoor activity? Plant dermatitis (phytodermatitis) can occur after contact with plants such as the Rhus tree (*Toxicodendron succedaneum*), Primrose (*Primula obconia*) or Chrysanthemum.[13]

A diagnosis of atopic dermatitis (eczema) generally occurs early in life and is more likely in patients with another atopic condition, i.e. asthma or allergic rhinitis. Atopic dermatitis is reported to affect approximately 3% of the adult population and may persist into older age.[14] In adults, the dermatitis is often persistent and localised to the hands, eyelids, flexures and nipples. The skin is often more dry and lichenified than seen in children with eczema.[15] Recurrent staphylococcal infections may also occur.[15] Irritant contact dermatitis due to exposure to water or detergents is more likely to occur in patients with atopic dermatitis.[15]

For further information see: "Managing eczema", BPJ 23 (Sep, 2009).

Psoriasis can start later in life and is reported to affect approximately 2 – 4% of the population.[16] The condition is often mild and may or may not be itchy.[16] Approximately 5% of these patients will develop psoriatic arthritis involving single or multiple joints, which can be debilitating.[16] Psoriasis appears as red, scaly plaques with well defined edges and silvery-white scale, and is often symmetrical.[16] Scale may be less obvious if the patient has been using emollients regularly.[16]

For further information see: "The treatment of psoriasis in primary care", BPJ 23 (Sep, 2009).

Consider other causes of dry, itchy skin

Other skin disorders frequently encountered in older patients that cause dry, itchy skin include:[3]

- Adverse reactions to medicines, e.g. antibiotics
- Localised contact irritant dermatitis, e.g. vulval dermatitis due to urinary incontinence (see:"Vulvovaginal health in post-menopausal women" Page 16)
- Localised contact allergic dermatitis (occurs less frequently), e.g. hair dye dermatitis
- Venous eczema associated with leg oedema and lipodermatosclerosis
- Localised inflammatory skin disorders, e.g. lichen sclerosus (For further information, see: **www.dermnetnz.org/immune/lichen-sclerosus.html**)
- Grover disease – mainly affects older males and may appear as itchy crusted papules on the trunk. It is more common in winter and although frequently itchy there may be no other symptoms (For further information, see: **www.dermnetnz.org/scaly/grovers.html**).
- Bacterial infections, e.g. cellulitis
- Intertrigo due to seborrhoeic dermatitis
- Fungal infections, e.g. *Candida albicans*

When examining the patient's skin, pay particular attention to their feet, especially in older patients with diabetes. Examine the soles of the feet for scale and between the toes for maceration and fissuring suggestive of tinea pedis. Nail changes can help in the diagnosis of fungal infections and psoriasis. Cellulitis is painful and should be suspected in patients with erythema, swelling and skin that is hot to the touch. Referral and/or skin biopsy should be considered for patients with skin lesions that cannot be classified on examination. Further investigations may be appropriate in patients with itchy skin and additional features consistent with an underlying condition. For example, HbA_{1c} testing should be considered in patients with pruritus, polyuria and polydipsia, or liver function testing in a patient with pruritus and other symptoms of liver dysfunction.

A general approach to pruritus

Scratching can exacerbate pruritus. It can also result in secondary lesions that may mask the primary cause of the patient's pruritus. Excoriations or bruising may be present on areas such as the back if the patient has used a hair brush or doorway to alleviate their itch, therefore a thorough inspection of the skin is necessary.

Resisting scratching can be as stressful as pruritus itself. Some patients may find pressing on the affected area, rather than scratching, provides relief. The use of a damp cloth, reduction of night-time heating and tepid showers may be beneficial. Suggest patting skin dry rather than vigorous towel-drying following washing to reduce the risk of further skin damage. Some patients may be able to tolerate wearing gloves or mittens while they sleep to prevent further skin damage.

Topical products may provide limited relief of pruritus

Over-the-counter topical products containing menthol or phenol cause nerve fibres to transmit a cold, itch-relieving sensation when they evaporate.[3] Menthol is safe and non-toxic when applied to unbroken skin; emollient products containing approximately 1% menthol are suitable for older patients, although care should be taken to avoid mucus membranes and genitals.[3] There are no subsidised proprietary products that contain menthol, but menthol in other bases, e.g. aqueous cream, 10% urea cream, wool fat with mineral oil, 1% hydrocortisone with wool fat and mineral oil lotion, or glycerol, paraffin and cetyl alcohol lotion, can be prepared by pharmacists if prescribed.

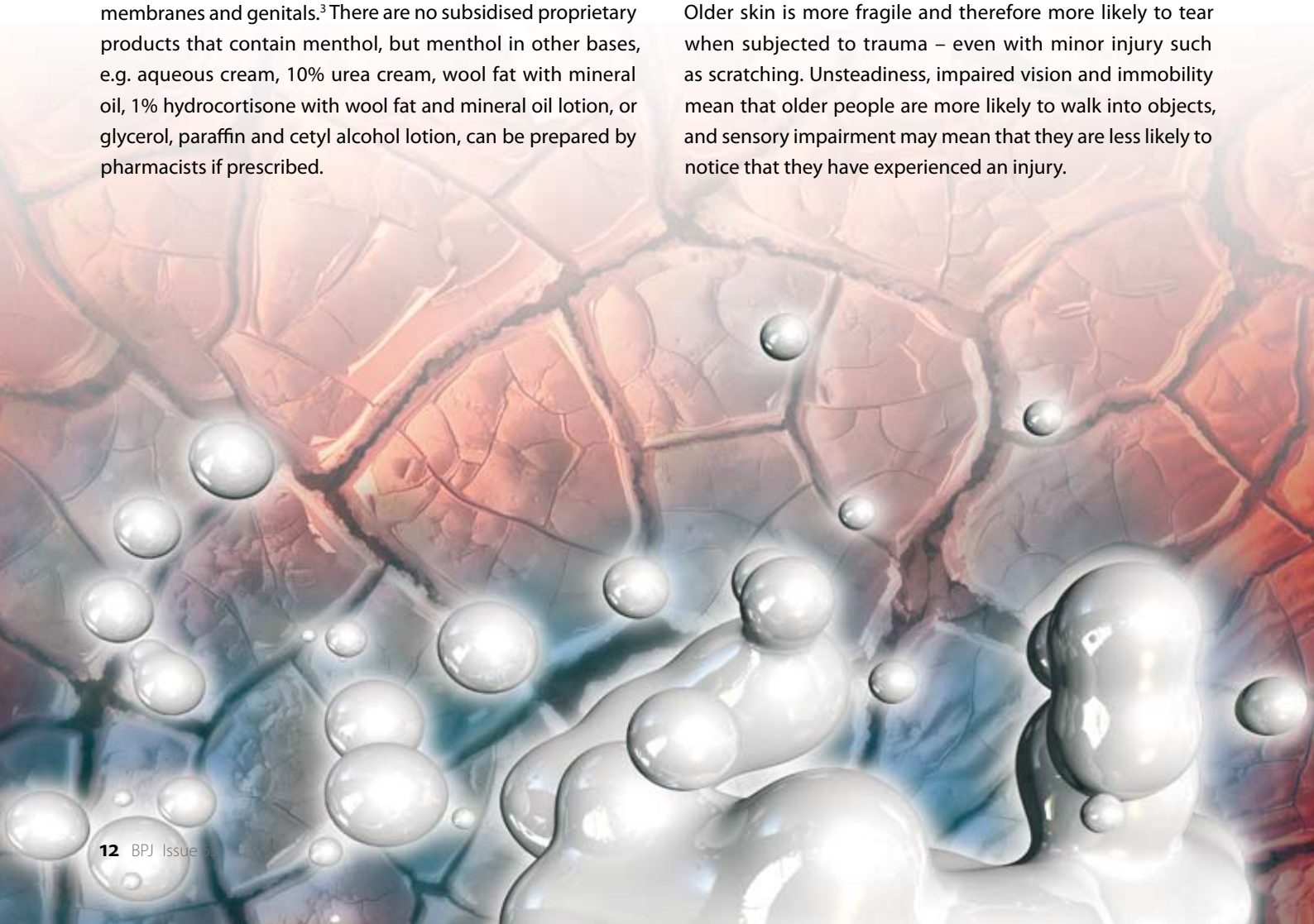
Tar (2.3%) with triethanolamine lauryl sulfate (6%) (Pinetarsol solution) is indicated and fully-subsidised for the treatment of dry, itchy, inflamed or flaky skin conditions, e.g. eczema, dermatitis and psoriasis. Bathing for five to ten minutes in a warm to tepid bath with 15 – 30 mL of Pinetarsol added may provide relief for the patient.[8]

Capsaicin cream (0.075%) can desensitise sensory nerve fibres and exert an antipuritic effect.[3] This treatment may be beneficial for neuropathic, systemic and dermatological pruritus, although pain, burning and stinging at the site of application may mean that treatment is not tolerated by the patient.[3] Capsaicin cream is only subsidised for patients with post-herpetic neuralgia, diabetic neuropathy or osteoarthritis (Special Authority criteria apply). It can be purchased OTC.

Local anaesthetics and topical antihistamines are only slightly effective in treating pruritus and can occasionally cause sensitisation.[8] Calamine aqueous cream (containing zinc oxide) and calamine lotion (containing phenol 0.5% and zinc oxide) are fully subsidised, but can increase skin dryness and are therefore not recommended.[8]

Managing skin tears due to dry, itchy skin

Older skin is more fragile and therefore more likely to tear when subjected to trauma – even with minor injury such as scratching. Unsteadiness, impaired vision and immobility mean that older people are more likely to walk into objects, and sensory impairment may mean that they are less likely to notice that they have experienced an injury.



Skin tears in older people often occur on the upper and lower limbs and on the back of the hands.[17] Where a patient requires assisted lifting or bathing by a carer the risk of skin tears in other areas may be increased. Older people and their carers can reduce the risk of skin tears by keeping their nails well trimmed, not wearing jewellery and placing padding around corners of objects, e.g. beds. Gardening and interacting with pets are also common causes of skin tears in older people. Encourage older patients to wear gloves and protective clothing when working outside and to keep pets claws trimmed and discourage them from jumping up where possible. Advise patients to report and seek treatment for any significant skin tears as early treatment reduces the likelihood of complications developing.

Assessing skin tears

When assessing a skin tear it is important to note any underlying conditions that may influence the patient's rate of healing, e.g. diabetes or venous insufficiency. The patient's tetanus vaccination status should also be considered.

A baseline assessment of the wound allows the healing process to be monitored. It is recommended to include the:[17]

- Date that the injury occurred and its anatomical location
- Length, width, depth and presence of skin flap
- Wound bed characteristics and percentage of viable tissue: eschar (scab) is usually seen as hard and black, and slough (dead tissue) is generally cream or yellow in colour
- Extent of any flap necrosis
- Type and quantity of exudate
- Presence of bleeding or haematoma
- Surrounding skin integrity
- Presence and severity of any pain
- Signs of infection

Best practice Tip: A photographic record with a ruler to demonstrate wound size can be useful for monitoring wounds.

Treating skin tears

The goals of skin tear management are to:[17]

1. Preserve the skin flap and protect the patient's surrounding tissue
2. Reapproximate the wound margins without overly stretching the skin
3. Reduce the risk of infection

Dressings that encourage a moist wound healing environment should be selected.[17]

Clean the wound with either warm saline or water to flush the wound and remove any debris or residual haematoma.[17] The surrounding skin should be patted dry, taking care not to cause further injury.

Any viable skin flap should be gently replaced using a dampened cotton tip, gloved finger, careful use of tweezers or a silicone strip.[17] A moistened non-woven swab can be applied to the flap for five to ten minutes to soften it and make it easier to align.[17]

A barrier cream, e.g. dimeticone 5%, can be applied as appropriate to prevent the surrounding skin breaking down due to periwound moisture.[17]

There is no single dressing that is recommended for all skin tears. The choice of dressing will be influenced by the location and type of tear and clinical experience. An optimal dressing for a skin tear injury will:[17]

- Provide an anti-shear barrier
- Optimise healing by providing a moist environment with bacterial exclusion and optimal pH
- Be flexible and moldable to contours
- Attach securely to the patient without causing ischaemia
- Be durable
- Not cause trauma on removal
- Allow for movement
- Be cosmetically acceptable

If an adhesive wound-closure strip is considered, e.g. steri-strips, then sufficient space should be left between each strip to allow drainage and swelling to occur.[17] Care should be taken to prevent tension over flexure sites which can cause ischaemia.[17] Tissue glue can be used to hold skin in contact with skin, but should not be used under the skin flap. Calcium alginate, foam or fibre dressings may help with exudate absorption.[17] Sutures and staples are generally not recommended in patients with fragile skin, however, these may be necessary when treating deep or full-thickness lacerations.[17]

Dressings should be left in place for several days to allow the skin flap to adhere.[17] When using an opaque dressing an arrow indicating the direction of removal may be useful.

Skin and the biology of ageing

The process of ageing involves genetics, physiological processes and environmental factors. As ageing progresses, body function is reduced at all levels, from cells to organs, including the skin.[20]

The outer most layer of the epidermis, the stratum corneum, forms the skin barrier that restricts water loss and prevents entry of pathogens, irritants and allergens. Healthy skin contains densely packed corneocytes, which are flattened dead cells without a nucleus or organelles. These cells are embedded in extracellular lipids, such as ceramides.[4] Corneocytes contain natural moisturising factors, including urea, which attract and hold water inside the cell.[4, 5] In healthy skin, transepidermal water loss is minimised by the secretion of sebum onto the skin surface from the sebaceous glands.[2]

Over a life-time the epidermis and dermis becomes thinner and flatter; this is associated with a reduction in the number of cutaneous blood vessels and nerve endings, and a reduction in connective tissue containing collagen, elastin and ground substance (Figure 3).[1] Cumulative exposure to ultraviolet (UV) radiation damages DNA and causes extracellular proteins to degrade. The skin's mechanical strength declines and there is a decreased ability to retain water, control temperature and sense the environment.[1] Production of sebum also declines making corneocytes more susceptible to desiccation and shrinkage.[1]

Skin accumulates advanced glycation end products (AGEs) over time, which are the product of non-enzymatic reactions between reducing sugars, e.g. glucose, and proteins, lipids or nucleic acids.[20] AGEs are reactive compounds that cause proteins, such as collagen, to crosslink, resulting in a loss of skin elasticity.[20] AGEs are of dietary origin and endogenously produced. The rate of AGE formation is elevated in people with diabetes and they are also increased by smoking and food preparation methods, e.g. fried food generally has a far higher AGE content than steamed or boiled food.[20] Exposure to UV radiation may accelerate the formation of AGEs.[20] Various receptors interact with AGEs that are involved in inflammation, immune responses, cell proliferation and gene expression.[20] Accumulation of AGEs is associated with angiopathy and solar elastosis (a thickening and yellowing of the skin due to sun damage).

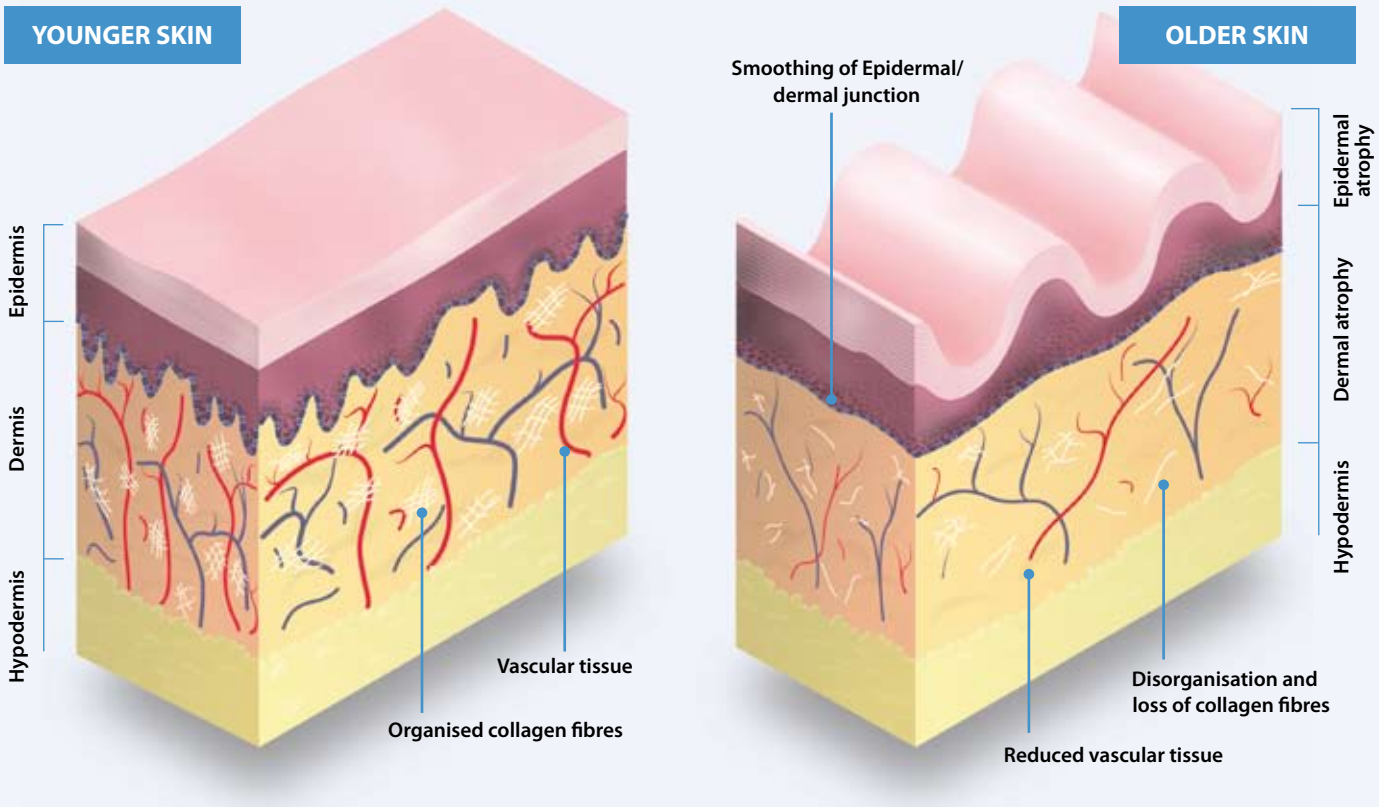


Figure 3: Comparison of skin anatomy in a younger person and an older person

If the patient is at increased risk of infection, e.g. the patient has diabetes or has a history of skin infections, then empiric oral antibiotics may be considered, e.g. flucloxacillin (erythromycin if penicillin-allergic). Topical antibiotics or antiseptics should not be routinely used for wound management.

Review and reassess the wound at each dressing change. The dressing should be gently worked away from the attached skin flap. Soaking dressings in saline or using silicon-based adhesive removers can reduce trauma to the healing wound.[17] Cleaning should not disrupt the skin flap. The wound bed should be assessed for changes. If the skin or flap is pale and darkened, it should be re-examined after 24 – 48 hours.[17] If the skin flap is not viable then debridement is usually necessary.[17]

Increased pain may indicate that the patient's wound has become infected. However, routine microbiological assessment of wounds is not necessary as swabbing a wound that is not infected can result in the identification of organisms colonising the wound rather than those causing an infection. Consider swabbing a wound if there are clinical signs that the wound is infected and it is deteriorating, increasing in size or failing to heal.[18] In patients with diabetes, or another condition associated with reduced perfusion and immune function, a lower threshold for swabbing the wound may be appropriate as the classical clinical signs of infection may not be present.[19]

Oedema may cause levels of exudate to be increased. Patients with skin tears on their legs may need to elevate the wound to reduce the likelihood of oedema complicating wound healing. However, regular movement of the leg in patients who are able to exercise should also be encouraged to improve circulation. In patients without peripheral artery disease and diabetes mellitus, graduated compression therapy should be considered early in the treatment process in order to prevent the development of a chronic leg ulcer.

 For further information see: "Microbiological assessment of infected wounds: when to take a swab and how to interpret the results", BT (Jun, 2013).

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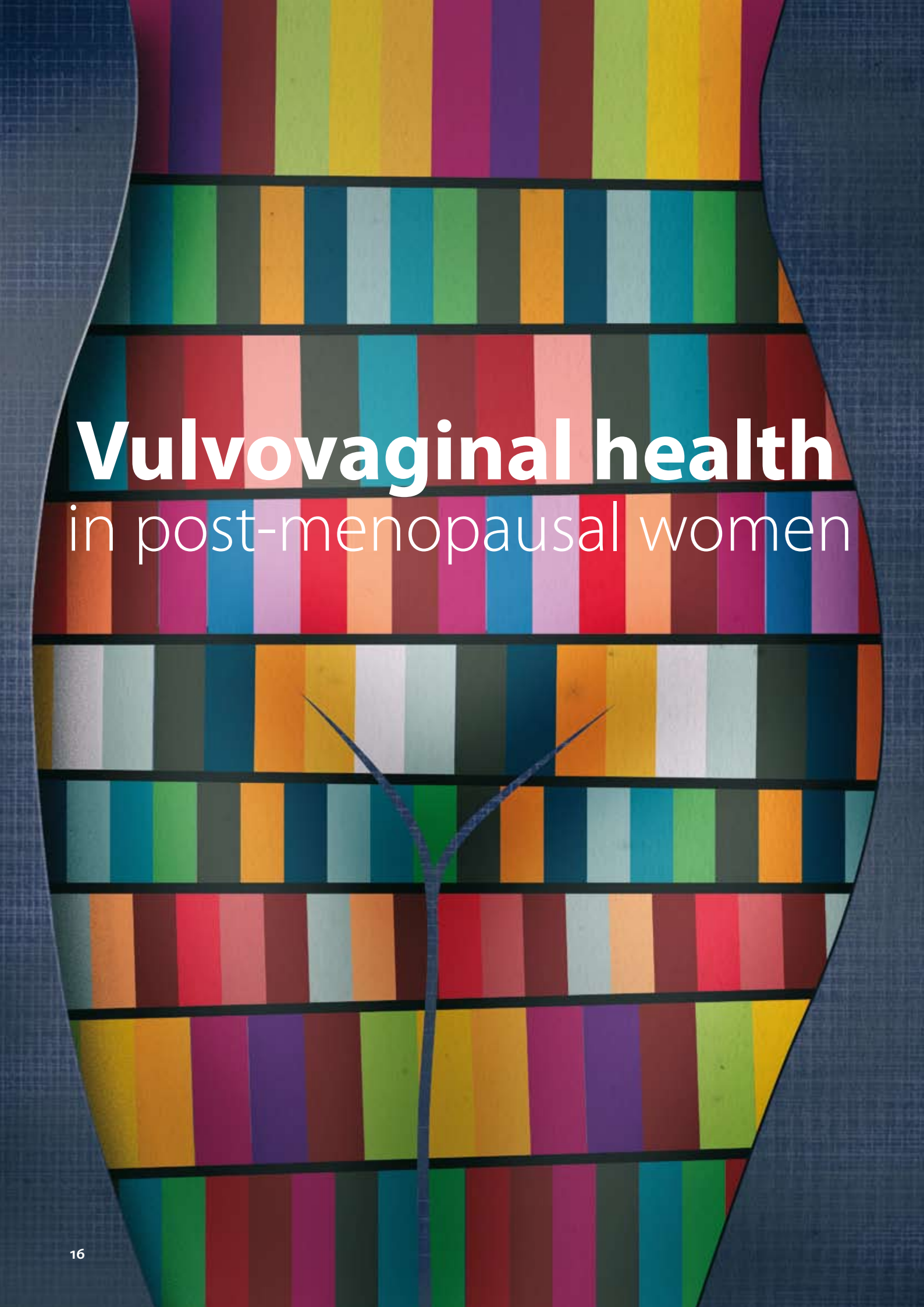
References

1. Best practice statement: Care of the older person's skin. 2012. Available from: www.woundsinternational.com/pdf/content_10608.pdf (Accessed Aug, 2014).
2. Cowdell F. Promoting skin health in older people. Nurs Older People 2010;22:21–6.
3. Cohen KR, Frank J, Salbu RL, et al. Pruritus in the elderly: clinical approaches to the improvement of quality of life. P T Peer-Rev J Formul Manag 2012;37:227–39.
4. Moncrieff G, Cork M, Lawton S, et al. Use of emollients in dry-skin conditions: consensus statement. Clin Exp Dermatol 2013;38:231–8.
5. DermNet NZ. Urea. DermNet NZ, 2013. Available from: www.dermnetnz.org/treatments/urea.html (Accessed Aug, 2014).
6. Danby SG, AlEnezi T, Sultan A, et al. Effect of olive and sunflower seed oil on the adult skin barrier: implications for neonatal skin care. Pediatr Dermatol 2013;30:42–50.
7. DermNet NZ. Allergy to wool alcohols. DermNet NZ, 2013. Available from: www.dermnetnz.org/dermatitis/wool-alcohols-allergy.html (Accessed Aug, 2014).
8. New Zealand Formulary (NZF). NZF v25. 2014. Available from: www.nzf.org.nz (Accessed Aug, 2014).
9. Counties Manukau DHB. Eczema – Emollients available for use in eczema. 2012. Available from: www.healthpoint.co.nz/public/paediatrics/kidz-first-outpatient-care-general-paediatrics/im:308393/ (Accessed Aug, 2014).
10. Medsafe. Aqueous cream – moisturiser or irritant? Prescr Update 2012;33:4.
11. Danby SG, Al-Enezi T, Sultan A, et al. The effect of aqueous cream BP on the skin barrier in volunteers with a previous history of atopic dermatitis. Br J Dermatol 2011;165:329–34.
12. Tsang M, Guy RH. Effect of Aqueous Cream BP on human stratum corneum in vivo. Br J Dermatol 2010;163:954–8.
13. DermNet NZ. Plant dermatitis. DermNet NZ, 2013. Available from: www.dermnetnz.org/dermatitis/plant-dermatitis.html (Accessed Aug, 2014).
14. DermNet NZ. Guidelines for the managment of atopic dermatitis. DermNet NZ 2014. Available from: www.dermnetnz.org/doctors/guidelines/atopic-guidelines.html (Accessed Aug, 2014).
15. DermNet NZ. Atopic eczema. DermNet NZ, 2014. Available from: www.dermnetnz.org/dermatitis/atopic.html (Accessed Aug, 2014).
16. DermNet NZ. Psoriasis. DermNet NZ, 2014. Available from: www.dermnetnz.org/scaly/psoriasis-general.html (Accessed Aug, 2014).
17. Stephen-Haynes J, Carville K. Skin tears made easy. Wounds Int 2011;2:1 – 6.
18. Edmonds M, Foster A. ABC of wound healing: Diabetic foot ulcers. BMJ 2006;332:407.
19. Bergin S, Gurr J, Allard B, et al. Australian Diabetes Foot Network: Management of diabetes-related foot ulceratioin - a clinical update. Med J Aust 2012;197:226–9.
20. Gkogkolou P, Böhm M. Advanced glycation end products: Key players in skin aging? Dermatoendocrinol 2012;4:259–70.



Vulvovaginal health
in post-menopausal women

Age-related changes in women can result in an increased occurrence of vulvovaginal dermatological conditions such as vulval dermatitis and lichen sclerosus, along with associated issues such as incontinence, recurrent urinary tract infection and sexual dysfunction. Atrophic changes during and after menopause due to declining oestrogen levels can result in a range of symptoms, including vaginal dryness and irritation as well as increase susceptibility to vulvovaginal trauma and infection.

Vulvovaginal changes after menopause

Vulvovaginal atrophy occurs due to decreasing oestrogen levels

Oestrogen is the primary hormone that regulates the physiology of the vulvovaginal tissues. As a woman ages, the progressive decline in circulating oestradiol, beginning in the peri-menopausal period, results in a number of changes that can affect the health of the genitourinary tract. The inherent sensitivity of the vulvovaginal skin, progressive oestrogen deficiency and the close proximity of the urethral opening and the anus, combined with skin changes due to ageing make conditions affecting the vulvovaginal skin common and a cause of distress for many post-menopausal women.

For further information, see: "Skin and the biology of ageing", Page 14.

Changes that occur with increasing age and decreasing oestrogen levels include:[1]

- Atrophy of vulval tissues – thinning of the skin, atrophy of subcutaneous fat, decreased hair growth
- Atrophy of the vagina – narrowing and shortening of the vagina with constriction of the introitus. The lining of the vagina tends to become thinner, less elastic and smoother due to a decrease in the rugal folds
- Atrophy of all other oestrogen-dependent tissues, e.g. pelvic floor muscles, urethral mucosa, uterus, ovaries
- Decreased vascularity
- Decreased vaginal secretions
- Alterations in the vaginal microflora – decreased glycogen from vaginal epithelial cells results in a change in the pH of the vagina from acidic to more basic (typically > 5.0). The change in pH is detrimental to the survival of acid-producing bacteria (e.g. lactobacilli) and can lead to further alterations in the pH and the microflora.

Vulvovaginal atrophy is the term used to describe the specific atrophic changes of the vulva and vagina that occurs progressively in all women after menopause. It is also regarded as a condition in itself because the characteristic changes due to declining oestrogen can result in a range of symptoms, such as vaginal dryness, irritation and discomfort (Page 18). The atrophic changes also make the vulvovaginal skin more vulnerable to trauma and infection.[1]

Other vulvovaginal conditions become more common after menopause

In addition to vulvovaginal atrophy, a number of other conditions become more common after menopause, such as vulval dermatitis, lichen sclerosus and less frequently, lichen planus (Page 22). Lichen simplex may also occur in post-menopausal women, however, it is more frequently observed in younger women. The pattern of symptoms from these conditions can often be similar, with the majority of women having itch as their primary symptom. The non-specific nature of the presenting symptoms, however, can make distinguishing between the various conditions difficult.

In some women, more than one vulval condition may be present simultaneously or there may be a more generalised underlying dermatological condition, e.g. psoriasis. Itching from a primary dermatosis may lead to scratching and excessive use of hygienic measures, leading to secondary lichen simplex and irritant contact dermatitis. Other diagnoses should be considered, therefore, if an initial treatment regimen has failed to produce an improvement in symptoms.[2] Making a diagnosis can be difficult in some patients, so it is generally recommended that referral to a Dermatologist or a Gynaecologist (preferably with a special interest in vulval dermatoses) should be considered for confirmation of a diagnosis if the vulval disorder has failed to respond to initial treatment.

Atrophy of oestrogen-dependent tissues can contribute to other gynaecological problems for women who are post-

menopausal, including uterine prolapse, urinary incontinence (see: "Incontinence is a risk factor for skin lesions", Page 22) and recurrent urinary tract infections (see: "Recurrent UTIs", Page 22).[3] Women who are post-menopausal may also continue to have problems with vulvovaginal candidiasis and bacterial vaginosis.

For various reasons, sexually transmitted infections (STIs) are often not considered as a diagnosis in older women.[4] However, many post-menopausal women remain sexually active and may have a higher risk of STIs due to increased susceptibility to infection (as a result of atrophic change) and a lack of condom use, particularly in women who are "newly single".[4, 5] Women may also have concerns about sexual function, as this can be affected by vulvovaginal atrophy and vulval skin conditions (see: "Sexual health for older women", Page 25).

Ask about vulvovaginal health

Many women may be reluctant to talk about vulval or vaginal problems with a health professional and may initially use over the counter products in an attempt to relieve vulvovaginal symptoms. It is estimated that only 25–50% of women with vulvovaginal symptoms seek help from their General Practitioner.[1, 6] Research has shown that there are many reasons why women do not ask for help including:[6]

- The feeling that it is an embarrassing, uncomfortable or private matter
- The belief that it is a normal part of getting older
- Not being aware that there are treatments available
- Not knowing how to initiate a conversation about these issues

Acknowledging that changes in vulvovaginal health are an expected part of ageing and initiating a conversation about the presence of any symptoms may encourage women to share their concerns and be more receptive about the options for treatment.[6] Some women may not reveal that they have a skin disorder affecting the vulva because they are uncomfortable or embarrassed by the need for a clinical examination of the vulvovaginal area. Their concerns should be acknowledged and if appropriate, other options could be offered, e.g. seeing a female General Practitioner in the practice if their regular General Practitioner is male.

The management of common vulvovaginal conditions in post-menopausal women

Vulvovaginal atrophy

Symptoms of vulvovaginal atrophy include irritation, vaginal dryness, dysuria and other urinary symptoms, dyspareunia and abnormal vaginal discharge.[1] Atrophic vaginitis is the term often used when inflammation accompanies atrophic change, resulting in patchy redness and tenderness of the vaginal introitus.[1] In a woman with vulvovaginal atrophy without inflammation, the tissues tend to be thin, pale and dry. Fissuring of the posterior fourchette (the fold of skin forming the posterior margin of the vagina) is often seen and may also occur as a result of even minimal stretching during vulval or vaginal examination.

Local oestrogen treatment is usually the preferred treatment option, rather than oral or transdermal oestrogen treatment, when the sole aim of treatment is the relief of vulvovaginal symptoms.[6] Treatment with topical oestrogens (e.g. estriol 0.1% cream or 500 microgram pessaries) is regarded as safe and effective.[2] The initial advice should be to use one application or pessary daily in the evening until there is improvement in symptoms (often two – three weeks) and then to reduce the frequency to one evening, twice a week.[1, 6] The use of progestogens for endometrial protection is not usually necessary when using topical oestrogens.[1] Patients with vaginitis should be warned that initially the use of oestrogen cream or pessaries may cause stinging or burning, but that this should improve within approximately two weeks. A non-oestrogen containing vaginal moisturising bioadhesive gel, e.g. Replens (unsubsidised), may be used in conjunction with a topical oestrogen but it is less effective at relieving symptoms on its own. A water-based vaginal lubricant may be required to alleviate vaginal dryness and friction-related trauma during sexual intercourse, however, lubricants may also cause transient stinging or burning if the woman has vaginitis or fissuring.[1]

Uterovaginal prolapse (pelvic organ prolapse)

Women who are peri- or post-menopausal may present with symptoms due to pelvic organ prolapse. The symptoms include a dragging sensation in the pelvis, urinary incontinence or difficulties with micturition and defaecation. Examination will usually reveal bulging of the vaginal walls due to prolapse of the uterus, rectum or bladder and in some women descent of the cervix (or vaginal vault in women following hysterectomy) that depending on the stage of the prolapse may extend through the introitus with straining. Treatment options include pelvic floor exercises (often guided by a physiotherapist), topical oestrogen, use of a vaginal ring pessary or surgery.

Vulval dermatitis

Vulval dermatitis in post-menopausal women is more likely to be contact dermatitis due to exposure to an irritant such as soap, fragrance, over-washing or urine, than to be atopic dermatitis.[2] Irritants produce inflammation of the skin, which is often aggravated by vulvovaginal atrophy, and cause itch, burning or non-specific irritation. The clinical findings on examination may vary – a woman with mild dermatitis may have redness, swelling and scaling of the affected area, whereas a woman with more severe dermatitis may have skin that is markedly red and swollen with obvious erosions or ulceration.[7] Women with chronic dermatitis can develop lichenification (see: Lichen simplex, below).

Initial management relies on the avoidance of contact with irritants (see: "Strategies to reduce vulvovaginal irritation", Page 20) and the use of emollients.[7] Low-potency topical corticosteroids, e.g. 1% hydrocortisone, can be trialled to reduce inflammation. In women with severe itch, an oral sedating antihistamine or tricyclic antidepressant may be required at night.[7] Vaginal swabs are appropriate if there is abnormal discharge or malodour, as there may be co-existing infections or symptomatic bacterial vaginosis that should be treated appropriately. The use of topical oestrogen can increase the incidence of *Candida albicans* vaginitis, which is otherwise uncommon in post-menopausal women.

Lichen simplex

Lichen simplex arises as a result of excessive scratching and rubbing of an area affected with an underlying condition, e.g. contact dermatitis or neuropathic pruritus. This leads to lichenification of hair-bearing skin, usually on the labia majora or perineum, where the skin becomes thickened with increased skin markings and follicular prominence (Figure 1). Lichen simplex is itself intensely itchy, therefore excoriations and broken off hairs are also frequently seen. Pruritus results in a characteristic itch-scratch-itch cycle with symptoms often worse at night or aggravated by heat, humidity, soaps or the presence of urine or faeces on the affected areas.[8] In addition to itch, sometimes women describe a feeling of burning or pain. Symptoms can be intermittent or persistent and the history may extend back for months or years.[8] Lichen simplex can occur anywhere on the body but the vulval area is one of a number of sites more commonly affected, others being the lower legs, forearms, wrists and the back of the scalp and neck.[9] On the vulva, lichen simplex can be localised to one area or widespread, although mucosal or glabrous (hairless) areas are not affected.[8]

Management, which aims to reduce itch and allow healing, involves a number of steps, along with advice on vulval care (see: "Strategies to reduce vulvovaginal irritation", Page 20). The steps are to:[9, 10]

- Identify and manage the condition that has produced the primary itch, e.g. dermatitis from an irritant or allergen, lichen sclerosus (see below). Neuropathic pruritus due to pudendal nerve entrapment or radiculopathy may explain symptoms if a primary dermatosis cannot be identified.
- Prescribe a sedating oral antihistamine or low-dose tricyclic antidepressant at night to break the itch-scratch-itch cycle and to assist with sleep
- Prescribe a potent topical corticosteroid (e.g. betamethasone valerate ointment) to be applied once daily to thickened skin to reduce lichenification. Reduce the potency or frequency of the topical corticosteroid as the plaques resolve, usually after four to six weeks depending on the extent and severity of lichen simplex. If treatment with betamethasone valerate ointment does not appear to be beneficial then referral to a Dermatologist is recommended. Ultra-potent topical corticosteroids such as clobetasol propionate ointment can be used but should ideally be prescribed only for specific indications when a diagnosis has been confirmed, and their use should be monitored.
- Explain how and where to apply the ointment; application of potent topical corticosteroids on non-affected skin risks steroid-induced cutaneous atrophy

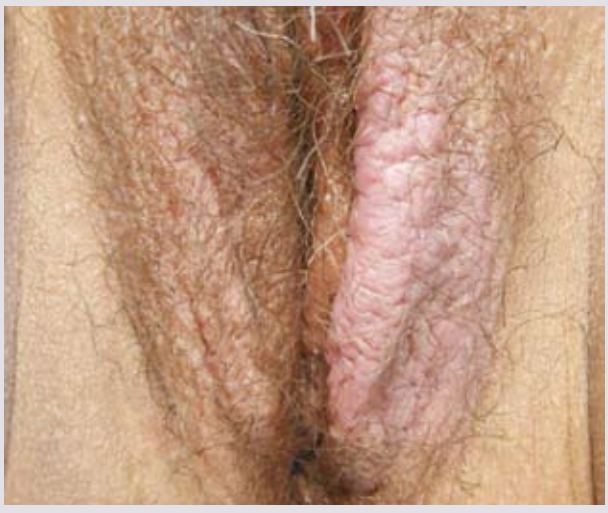


Figure 1: Lichen simplex showing asymmetrical lichenification of labia majora. Image provided by DermNetNZ

Strategies to reduce vulvovaginal irritation

Eliminating any aggravating factors is an important step in the management of women with conditions affecting the vulvovaginal area.[8] Aggravating factors include scratching and rubbing, products and routines used for cleansing, exposure to urine or faeces and medicines or products used to reduce symptoms from the underlying condition.[8] Women who are post-menopausal are more likely to be affected by these factors than younger women, as the barrier that the vulvovaginal skin forms is more vulnerable due to oestrogen deficiency.

Women can be advised to:[8]

- Avoid scratching and rubbing if possible – non-pharmacological methods for the relief from itch include cool gel pads, refrigerated petroleum jelly on a pad or cloth, or applied directly to the vulva. Petroleum jelly also acts as a moisturiser and a barrier preparation. It can be applied frequently. Sitting in a lukewarm bath may also be soothing. Advise women to keep their fingernails short or to wear gloves at night.
- Wash the vulva with water and avoid the use of soaps, bubble baths, bath oils and salts or perfumed products.
- Use white, unscented soft toilet paper – some of the inks on the printed papers can irritate skin. Avoid using baby or personal wipes; these wet products have a high-concentration of preservatives (such as methylisothiazolinone) to which contact allergy is increasingly reported.
- Tell their doctor if they have urinary incontinence so that this can be treated. Ensure pads or underwear designed for incontinence are used rather than pads intended for menstrual use. If leakage occurs, ideally the urine should be rinsed off the vulva with water followed by the application of petroleum jelly to provide a barrier.
- Wear comfortable underwear and avoid pantyhose. Avoid wearing underwear at night unless specific incontinence products are required.
- Avoid using over-the-counter products on the genital area, e.g. topical antifungal medicines or douches, as these can cause pain or irritation and may alter normal or desirable vaginal microflora

In addition, cool packs to control itch short-term, and emollients to reduce dryness and itch, can be applied frequently and may be helpful. Erosions and fissures can be caused by scratching and, although uncommon, can predispose the patient to secondary bacterial infections which may require oral antibiotics.[8] Treatment can often result in complete resolution of symptoms, however, this relies heavily on an effective approach to the elimination of vulval irritants and being able to stop the itch-scratch-itch cycle. For some women, lichen simplex can become chronic and cause significant distress. Long-term use of a tricyclic antidepressant, and intermittent applications of topical corticosteroid ointments (e.g., as weekend pulses), may be required in these women.

Follow-up is essential to ensure symptoms are controlled and treatment is used effectively and safely

Lichen sclerosus

Lichen sclerosus is an inflammatory skin disorder, thought to be of autoimmune origin, but with influences from genes, hormones, irritants and infection.[8, 11] It can occur in women of any age, but most frequently in those aged over 50 years.[11] Lichen sclerosus primarily affects the glabrous (hairless) vulval, perineal and perianal skin but does not involve the vagina itself. Longstanding disease can extend to involve the labia majora and inguinal folds. Approximately 10% of women with vulval lichen sclerosus will also have non-genital areas of skin affected,[11] and up to 20% may have another autoimmune disease, such as thyroid dysfunction, vitiligo, psoriasis or pernicious anaemia.[8, 11]

The most common symptom in women with lichen sclerosus is severe itch, although many are asymptomatic. Women may also complain of pain, which may be aggravated by the development of fissures secondary to scratching or friction from sexual intercourse. Chronic lichen sclerosus can cause distortion of the genital anatomy, including adhesions, resorption or partial fusion of the labia minora, and narrowing of the vaginal introitus causing dyspareunia compounded by post-menopausal changes from atrophy and loss of elasticity.[8] Scarring and fissure development around the anus can cause pain or bleeding and aggravate constipation.

On examination, the affected areas of skin may appear white and thickened and there may be ecchymoses, petechiae or purpura (Figure 2). Scratching can result in fissures and, rarely, secondary infection.

Referral to a specialist in vulvovaginal disease (usually a Dermatologist or a Gynaecologist with an interest in vulval

disorders) is recommended for confirmation of the diagnosis and, when management is complex, shared long-term care. It is not always easy to distinguish lichen sclerosus from other conditions affecting the vulval area and a biopsy is often required for an accurate diagnosis. Lichen sclerosus is rarely curable, although can usually be improved, therefore it is important that a long-term plan is established for treatment and follow up.[8] In addition, lichen sclerosus is associated with the development of vulval intraepithelial neoplasia (VIN) and invasive squamous cell carcinoma, with an incidence of approximately 5%.[8] In women with lichen sclerosus, ideally the vulval skin should be reviewed at least annually, or more often if symptoms persist despite treatment, so that an alternative diagnosis can be considered or if malignancy develops it is detected early (see: "Malignant vulval skin lesions", Page 24). Education is essential to explain the long-term nature of the disorder, the need for on-going, at least intermittent, treatment and follow-up.

Treatment with a potent or ultra-potent topical corticosteroid ointment, e.g. betamethasone valerate ointment or clobetasol propionate applied at night to affected areas for up to three months, is the usual initial choice and is aimed at reducing symptoms to a tolerable level.[8] Ensure that the woman is aware of the specific areas of affected skin that should be treated. The duration of daily treatment depends on the initial severity and the response to treatment. The frequency of application or potency of the topical corticosteroid should then be slowly reduced once the symptoms have begun to settle, e.g. used one to three times a week. More limited use of a potent or ultra-potent corticosteroid (e.g. a maximum of two weeks) is recommended in women with lichen sclerosus affecting the perianal skin because this is more susceptible to thinning.[8]

The majority of post-menopausal women with vulval lichen sclerosus should also be treated with intravaginal oestrogen cream. The response to corticosteroid treatment can be quite variable, with itch reducing within a few days but the appearance of the skin not returning to normal for weeks or months.[11] Maintenance treatment is required in many women, e.g. a topical corticosteroid used on a weekly basis, to prevent reoccurrence of symptoms and reduce the progression of scarring.[8] If scarring has already occurred, this is not reversible with corticosteroid treatment. If there is narrowing of the vaginal introitus, the use of vaginal dilators can be trialled. These are used progressively, starting with a small size and increasing in size as tolerated. Surgery is sometimes the best treatment option, particularly if the woman experiences difficulties with micturition (due to labial fusion causing obstruction of the urethra) or if the use of vaginal dilators has not resolved problems with sexual intercourse.[8,11]

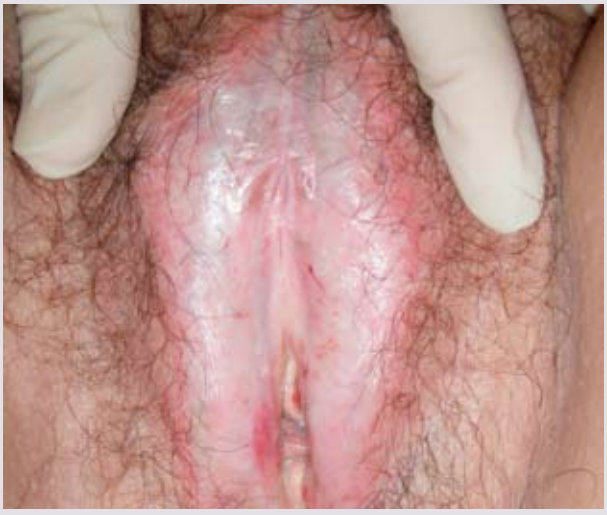


Figure 2: Lichen sclerosus showing whitening of the vulva extending towards the perianal skin with typical distortion, fusion and resorption of labia minora and ecchymosis. Image provided by DermNetNZ

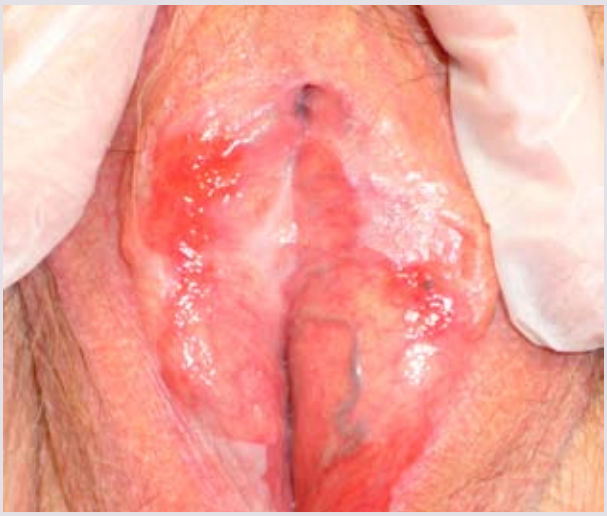


Figure 3: Erosive lichen planus showing characteristic redness and erosions of vulvovaginal skin. Image provided by DermNetNZ

Incontinence is a risk factor for skin lesions

The presence of urine and/or faeces on the skin creates an alkaline pH due to bacteria digesting urea and producing ammonia.[14] This increases the activity of proteases and lipases which can cause skin irritation and dermatitis.[14] Skin breakdown becomes more likely in older women if their skin remains moist for extended periods. Vulvovaginal atrophy, scratching and inappropriate cleansing can exacerbate this problem by further diminishing the skin's barrier function.

For further information, see: "Urinary incontinence in adults", BPJ 55 (Oct, 2013)

Recurrent UTIs are more common in older women

Older women are more susceptible to recurrent urinary tract infection (UTI) due to factors such as vulvovaginal atrophy (which increases risk of trauma and infection), incontinence, use of catheters and living in a residential care setting. It is estimated that each year 8% of postmenopausal women will have a UTI and 4% may have recurrent infections.[3]

Asymptomatic bacteriuria is very common in older women and does not require antibiotic treatment (or testing). The diagnosis of UTI in older women should therefore be made based on clinical signs and symptoms, as well as the results of urine culture. Urine culture should be requested in older women who have recurrent infection, or signs of significant infection such as fever > 38°C, worsening urgency or frequency, suprapubic pain, urinary incontinence or gross haematuria.

For some women recurrent urinary tract infections may be prevented by the use of topical oestrogen treatment.[3]

Lichen planus

Women with vulval lichen planus may present with itch and pain, similar to the symptoms of lichen sclerosus, however, it is less common than lichen sclerosus, is more likely to affect other areas of the body and also affects mucosal skin, e.g. of the vagina and mouth.[12] Lichen planus, like lichen sclerosus is also thought to be an inflammatory skin condition of autoimmune origin. Lichen planus most often affects women from age 30 – 60 years.[10]

The severity of vulval lichen planus tends to vary depending on the subtype. Subtypes include a cutaneous form (purplish or brown papules in hair-bearing areas), a mucosal form (painless, often itchy, white streaks) or the more common erosive form, affecting the vaginal introitus, characterised by marked redness and erosions with a characteristic white hyperkeratotic border (Figure 3).[8, 13] Erosive lichen planus can result in severe distortion and scarring of the affected areas with pain rather than itch being the main symptom.[10, 12] Unlike lichen sclerosus, lichen planus often affects the vaginal mucosa causing a bloody vaginal discharge.

The diagnosis of lichen planus can often be made based on the history and clinical findings, however, it is recommended that women be referred to a specialist in vulvovaginal disease for confirmation of the diagnosis, usually with biopsy although the histopathology may be nonspecific. The histological changes in lichen planus are often subtle and site-dependent with central areas less likely to show classical features than the samples taken from the margins.[13] In addition, as with lichen sclerosus, there is a risk of development of vulval malignancy and the condition can be more challenging to manage.[8, 12, 13]

Initial treatment for lichen planus is the same as for lichen sclerosus, but some women may require oral corticosteroids or immunomodulatory medicines, such as methotrexate, if the use of topical corticosteroids has not improved their symptoms.[10, 12]

Seborrhoeic dermatitis and psoriasis in post-menopausal women

Although more often diagnosed in younger women, seborrhoeic dermatitis and psoriasis may affect women of any age. These two conditions may occur simultaneously and when they are difficult to distinguish, "sebopsoriasis" may be diagnosed.

Seborrhoeic dermatitis tends to affect skin folds (e.g. inguinal, crural and interlabial creases), and hair-bearing areas (e.g. mons pubis, labia majora, perianal areas) and causes mild symptoms such as itch, scale and fissuring. Most women

with seborrhoeic dermatitis give a history of pityriasis capitis (dandruff) and seborrhoeic dermatitis affecting the scalp, eyebrows, retroauricular and nasolabial folds where they have ill-defined pink, flaking patches. Seborrhoeic dermatitis is treated with intermittent application of a topical antifungal (e.g., ketoconazole shampoo, twice weekly in the shower) and a low-potency topical corticosteroid (e.g., 1 % hydrocortisone cream) when symptomatic. This combination works well for seborrhoeic dermatitis, but is less effective for sebopsoriasis, which may require short term treatment with more potent corticosteroids (see below).

Psoriasis affecting the vulvovaginal area can be part of a more widespread type of psoriasis (usually plaque psoriasis), however, in 2 – 5% of patients, it may affect the genital area only.[15, 16] Women with psoriasis of the vulvovaginal area often present with well-circumscribed, bright red plaques that are symmetrically distributed in the vulva.[15] Other flexural sites are also commonly affected, e.g. natal cleft, umbilicus, axillae and under the breasts or an abdominal apron (if the flexures are involved it is referred to as flexural psoriasis).[16] Itch can vary from minimal to severe. On examination, scale can be a prominent feature, but it is often absent in moist areas resulting in a shiny smooth appearance to the affected skin (Figure 4). Psoriasis can be colonised by bacteria and yeasts, leading to symptomatic maceration and fissuring.

Treatment is usually with intermittent courses of low to moderate potency topical corticosteroids.[15] Flexural psoriasis usually responds well to the use of topical corticosteroids, however, it is often recurrent and may require repeated but intermittent use of a topical corticosteroid.[16] Education is therefore essential to explain to women that psoriasis tends to recur or that it may persist and to ensure that they use topical corticosteroids safely. Topical corticosteroids are absorbed to an increased extent by genital skin and this can result in thinning of the skin.[15] The use of more potent topical corticosteroids should be limited to a few weeks only and stepped down to a less potent corticosteroid once the psoriasis is improving.[15]

Stronger topical treatments used for psoriasis affecting other parts of the body (e.g. dithranol cream, coal tar preparations) may be too irritating for use in the vulvovaginal area, although they can be used for short periods and washed off or diluted in an emollient.[15] Oral medicines (e.g. methotrexate) are usually not required for psoriasis that is limited to the genital area, and the use of phototherapy should be avoided.[15]

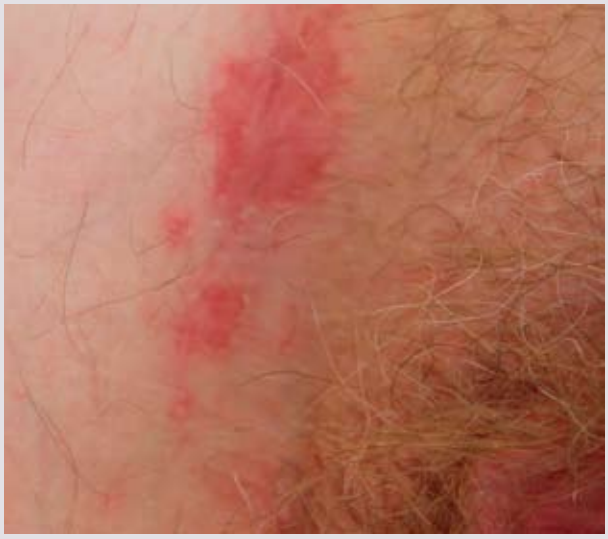


Figure 4: Sebopsoriasis in skin flexure showing red shiny skin with an absence of scale. Image provided by DermNetNZ

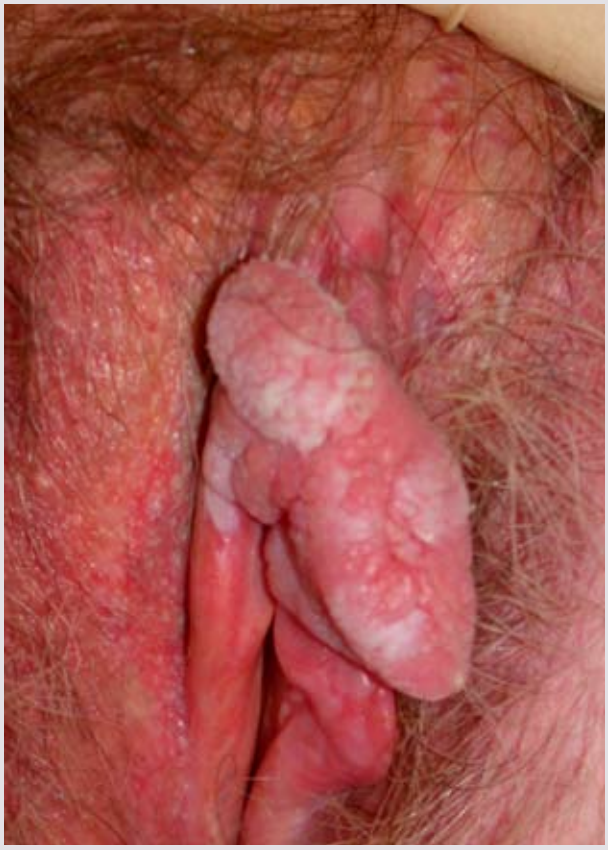


Figure 5: Squamous cell carcinoma (SCC) affecting the labia minora. SCC are variable in appearance, e.g. they can be warty, fleshy or ulcerated. Image provided by DermNetNZ

Less common vulvovaginal conditions in post-menopausal women

Mucous membrane pemphigoid (or cicatricial pemphigoid)

This is a rare autoimmune disease that causes blistering of mucous membranes, e.g. of the mouth, eye, nose and vulva.[17] It usually affects older people (age > 70 years) and is more common in women.[17] When it involves the vulva it can cause severe scarring resulting in distortion of the vulval anatomy.[18] Clinically it may be difficult to distinguish from other conditions affecting the vulva, such as lichen sclerosus or erosive lichen planus. Referral to a vulvovaginal specialist is recommended for an accurate diagnosis because although mucous membrane pemphigoid can respond to a potent topical corticosteroid it is often a very difficult condition to treat successfully and requires oral corticosteroids or an immunosuppressant medicine.[17]

Pemphigus vulgaris is another blistering autoimmune disease that can affect the genital area although more commonly the oral mucosa. Vulval pemphigus is extremely rare in New Zealand.

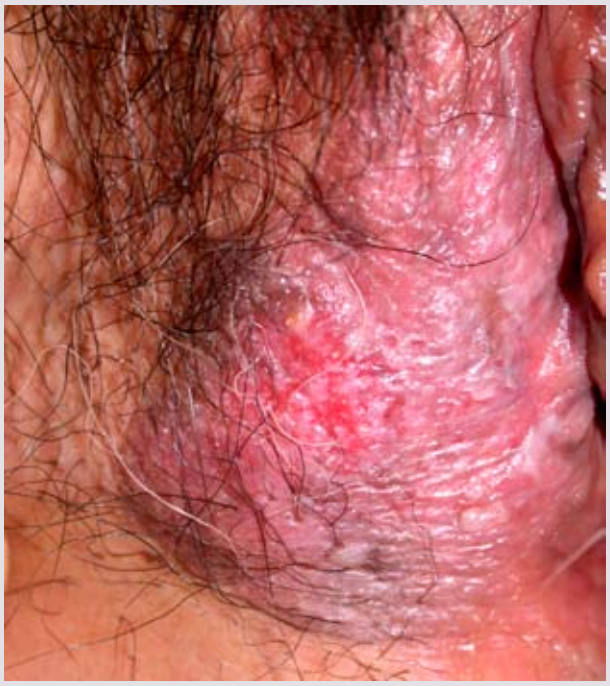


Figure 6: Paget disease of the vulva showing characteristic red thickened (fleshy) appearance. Image provided by DermNetNZ

Malignant vulval skin lesions

Most malignancies involving the vulval area occur in post-menopausal women, although vulval intraepithelial neoplasia (VIN) may begin prior to menopause and is occasionally diagnosed in younger pre-menopausal women.[19] VIN has the potential to progress to invasive carcinoma of the vulva and women with suspicious lesions require referral to secondary or tertiary care for biopsy and treatment. Approximately 90% of vulval cancers are squamous cell carcinomas (Figure 5), however, other types of malignant lesion may occur in the vulval area including, melanoma, basal cell carcinoma, sarcoma and rarely, Paget disease of the vulva (below) and adenocarcinoma of the Bartholin gland.[20]

Compared to benign dermatoses, malignant lesions are usually asymmetrical, unifocal or multifocal papules, plaques, erosions and ulcers. As with malignant lesions elsewhere on the body, those on the vulva typically have an irregular shape, structure, colour and distribution. Most vulval cancer starts in glabrous or mucosal sites rather than in cutaneous areas.[20] Many women with malignant lesions of the vulva do not present with an obvious mass. Symptoms of vulval cancer vary with the extent and the specific type of cancer involved. For example, itch or pain are associated with squamous cell carcinoma in approximately 50% of women, lesions due to Paget disease of the vulva may cause a burning sensation and itch, while other women with malignant lesions may be asymptomatic.[20] Women with symptomatic vulval invasive cancers may present with itch, an obvious lump, pain, ulceration or bleeding.

Women with suspicious lesions or those that have not responded to treatment for conditions, such as lichen sclerosus should be referred urgently to a specialist for examination, biopsy and further investigations as appropriate. Risk factors for vulval cancer include smoking, VIN, lichen sclerosus, lichen planus, cervical cancer or intraepithelial neoplasia, previous HPV infection and positive HIV status.[20] Vaginal or anal intraepithelial neoplasia (VAIN, AIN) or invasive cancer of the vagina and anus are less common than vulval malignancy.

Paget disease of the vulva

Paget disease of the vulva (also referred to as extramammary Paget disease) is a rare malignant condition, primarily affecting older women, that can be difficult to distinguish clinically from other skin conditions affecting the vulva.[21] The clinical features include itch and sometimes pain arising from thickened areas of skin around the vulva that become red, scaly and crusted (Figure 6). Typically, the skin lesions will have been present for some time as initially they are asymptomatic or cause minor irritation only. If Paget disease is suspected,

referral to a vulvovaginal specialist is recommended because an accurate diagnosis relies on the results of a biopsy. Other investigations, e.g. colposcopy or pelvic imaging, are likely to be required because there is an association with other underlying malignancies. For example, Paget disease around the anus is associated with an underlying colorectal cancer in approximately 25 – 35% of people.[21]

Management usually involves surgical excision of the lesion, however, recurrence is common (up to 50%) and further surgery is often required.[10, 21] Mohs micrographic surgery is the preferred option, if it is available, as it is associated with lower rates of recurrence and less extensive surgical excision.[10, 21] Non-surgical treatments include the use of laser ablation, topical fluorouracil, imiquimod or photodynamic treatment.[10, 21]

Benign skin lesions

A number of benign skin lesions may be found in the vulvovaginal area including:

Seborrhoeic keratoses: appear as "stuck on" warty papules on hair-bearing skin. They are benign but may be symptomatic or confused with malignant lesions. Removal (e.g. shave/curette/diathermy or cryotherapy) is generally only indicated if the lesions are painful, increasing in size or to rule out malignancy (excisional biopsy).

Skin tags (acrochordon, soft fibroma): appear as pendulous lesions on a narrow stalk. More common in areas of friction (medial thighs), and in women who are obese. Removal by shave excision or cryotherapy is only necessary if painful irritation or inflammation occurs.

Epidermal inclusion cysts: are common on the hair-bearing skin of the labia majora. Treatment is only required if the cyst becomes infected (with incision and drainage, and an oral antibiotic if appropriate) or if the cyst is large and symptomatic when surgical excision is usually required, provided any infection has settled.

Melanocytic naevi (moles): typically appear as skin- to dark-coloured, soft macules or papules. They are mostly under 6 mm in diameter, and uniform in shape, colour and structure. However, naevi that are larger, irregular in shape or colour are not uncommon in pubic or genital sites. Examine the patient's overall pattern of naevi to determine whether a particular spot is different from others, i.e. an ugly duckling. If uncertain, arrange dermatoscopic examination by an expert (usually a Dermatologist). Removal is only necessary for cosmetic reasons or to exclude malignancy.

Angiokeratomas: are solitary or more often multiple red, purple, blue or blackish papules <5 mm, located on labia majora. Women may present with these lesions because of bleeding or painful thrombosis, or because they are alarmed by the appearance. Reassurance is appropriate. Larger lesions can be distinguished from malignant lesions because of their uniform shape, structure and colour. Dermatoscopy reveals single or multiple red, purple or blue clods (lacunes) unless thrombosed, when they are black (and soon resolve).

Lipomas: appear as a freely moving, well-defined, sub-cutaneous mass. Excision is only indicated if painful, increasing in size or to exclude malignancy. They are rare in the vulvovaginal region.

For further information and images of these lesions, see: **www.dermnetnz.org**

Sexual health for older women

Questions about sexual health are a routine aspect of general practice. While this is most commonly considered in younger patients, it is important that sexual health is discussed with all patients, regardless of their age. The purpose of a sexual health history is not only to assess risk of sexually transmitted infections, but also to identify problems with sexual function and to assess overall wellbeing and knowledge about sexual health.

Talking about sexual health can be awkward or embarrassing for women of all ages so it is important to ensure that the patient feels comfortable and that the tone of the consultation is appropriate. Consider using an opening statement such as "We routinely discuss sexual health with all our patients, is it ok if I ask you some questions?" This could be followed by more direct questions that can lead into a more detailed discussion about sexual health in older females:

- Are you sexually active?
- Do you have any questions or problems with sex that you would like to discuss?

Sexual response and what is considered normal varies from person to person. In general, a sexual health dysfunction should be only considered a problem if it causes distress to the person or their partner. For example, vaginal dryness or loss of libido may not be an issue for a woman who is not sexually active, however, if the woman meets a new partner, this may be something she seeks help for.

Sexual problems for older women may include:

- Loss of libido; identify any contributing factors such as medicines or unmanaged co-morbidities, offer referral for counselling
- Vaginal discomfort and dryness; recommend use of lubricant or consider use of topical oestrogen
- Vaginal/vulval pain; investigate and treat any cause, recommend use of lubricant, pelvic floor exercises
- Incontinence; manage symptoms and modifiable factors, recommend incontinence wear, pelvic floor exercises
- Effect of co-morbidities and medicines on sexual function; where possible, reduce doses or avoid medicines which decrease libido, e.g. antidepressants, manage co-morbidities
- Lack of privacy, e.g. in a residential care setting; encourage discussion with carers
- Self-esteem issues; encourage discussion and coping strategies, offer referral for counselling
- Relationship issues, e.g. new partner, pressure to have sex; encourage discussion, consider referral for counselling
- Inadequate knowledge about STIs; educate about STIs, testing, appropriate protection and possible symptoms

The North American Menopause Society has produced a useful resource for women experiencing sexual health issues after menopause, available from: **http://www.menopause.org/for-women/sexual-health-menopause-online**

General Practitioners with an interest in the area of vulvovaginal health may wish to join the Australian and New Zealand Vulvovaginal Society, which holds an annual conference and update meetings for health professionals. Their website provides a list of specialists with an interest in vulval disorders, information about upcoming meetings and conferences, website links and patient information about vulval disease. See: **www.anzvs.org/index.html**

Also see: "Vulvovaginal health in pre-menopausal women", BPJ 41 (Dec, 2011).

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References

1. North American Menopause Society. Management of symptomatic vulvovaginal atrophy: 2013 position statement of The North American Menopause Society. Menopause 2013;20:888–902.
2. Kingston A. Vulval disease in the postmenopausal patient: a guide to current management. Menopause Int 2010;16:117–20.
3. Parish S, Nappi R, Krychman M, et al. Impact of vulvovaginal health on postmenopausal women: a review of surveys on symptoms of vulvovaginal atrophy. Int J Womens Health 2013;5:437–47.
4. Drew O, Sherrard J. Sexually transmitted infections in the older woman. Menopause Int 2008;14:134–5.
5. Bourne C, Minichiello V. Older people are at risk of sexually transmitted infections. Australas J Ageing 2009;28:32–6.
6. Reiter S. Barriers to effective treatment of vaginal atrophy with local estrogen therapy. Int J Gen Med 2013;6:153–8.
7. Margesson L. Contact dermatitis of the vulva. Dermatol Ther 2004;17:20–7.
8. Thorstensen K, Birenbaum D. Recognition and Management of Vulvar Dermatologic Conditions: Lichen Sclerosus, Lichen Planus, and Lichen Simplex Chronicus. J Midwifery Womens Health 2012;57:260–75.
9. DermNet NZ. Lichen simplex. DermNet NZ, 2014. Available from: http://dermnetnz.org/dermatitis/lichen-simplex.html (Accessed Aug, 2014).
10. Doyen J, Demoulin S, Delbecque K, et al. Vulvar skin sisorders throughout lifetime: about some representative dermatoses. Biomed Res Int 2014;[Epub ahead of print].
11. DermNet NZ. Lichen sclerosus. DermNet NZ, 2013. Available from: http://dermnetnz.org/immune/lichen-sclerosus.html (Accessed Aug, 2014).

12. DermNet NZ. Lichen planus. DermNet NZ, 2014. Available from: http://dermnetnz.org/scaly/lichen-planus.html (Accessed Aug, 2014).
13. Simpson R, Thomas K, Leighton P, et al. Diagnostic criteria for erosive lichen planus affecting the vulva: an international electronic-Delphi consensus exercise. Br J Dermatol 2013;169:337–43.
14. Best practice statement: Care of the older person's skin. 2012. Available from: www.woundsinternational.com/pdf/content_10608.pdf (Accessed Aug, 2014).
15. DermNet NZ. Genital psoriasis. DermNet NZ, 2014. Available from: http://dermnetnz.org/scaly/genital-psoriasis.html (Accessed Aug, 2014).
16. DermNet NZ. Flexural psoriasis. DermNet NZ, 2013. Available from: http://www.dermnetnz.org/scaly/flexural-psoriasis.html (Accessed Aug, 2014).
17. DermNet NZ. Cicatricial pemphigoid. DermNet NZ, 2013. Available from: http://dermnetnz.org/immune/cicatricial-pemphigoid.html (Accessed Aug, 2014).
18. Goldstein A, Anhalt G, Klingman D, et al. Mucous membrane pemphigoid of the vulva. Obstet Gynecol 2005;105:1188–90.
19. DermNet NZ. Vulval intraepithelial neoplasia. DermNet NZ, 2013. Available from: http://dermnetnz.org/site-age-specific/vulvar-intraepithelial-neoplasia.html (Accessed Aug, 2014).
20. DermNet NZ. Vulval cancer. DermNet NZ, 2013. Available from: http://dermnetnz.org/site-age-specific/vulvar-cancer.html (Accessed Aug, 2014).
21. DermNet NZ. Extramammary Paget disease. DermNet NZ, 2013. Available from: http://dermnetnz.org/site-age-specific/extra-mammary-paget.html (Accessed Aug, 2014).

Helping patients cope with **chronic non-malignant pain:** it's not about opioids

The role of opioids in the management of chronic non-malignant pain is a controversial subject due to concerns over the long-term efficacy and safety of treatment, including the risk of misuse and addiction. In the past, opioids featured prominently in many treatment guidelines for chronic non-malignant pain. However, this advice has been reconsidered in more recent times and the current opinion is that opioids have a very limited role in the management of patients with chronic non-malignant pain. Non-pharmacological methods for helping patients cope, and come to terms, with their pain should be the mainstay of treatment. Non-opioid analgesics may be considered for periods when pharmacological treatment for pain is necessary. Opioids should only be considered as a treatment of "last resort", and should be used for the shortest possible time, at the lowest effective dose, using the least potent opioid possible.

Why opioids should not be used for chronic non-malignant pain

Opioid analgesics are often used in the treatment of patients with chronic non-malignant pain, despite a lack of evidence supporting their effectiveness in this setting. There is now a growing, consistent body of evidence that suggests that opioids should play a much smaller role than previously thought in managing these patients.[1,2] This evidence suggests that the long-term efficacy of opioids is not proven and that opioid treatment is associated with a well established risk of adverse events and addiction.

Management of patients with chronic non-malignant pain involves a complex interplay of biological, psychological and social factors, therefore treatment needs to incorporate all of these aspects. Psychological factors in particular play a major role in determining the success or failure of treatment in patients with chronic non-malignant pain. It is important that clinicians understand and empathise with the emotions the patient is experiencing in order to best manage their pain (see: Recognising the importance of the patient's emotional wellbeing", Page 31).

The long-term effectiveness of opioids is not proven

Most clinical research on opioids has studied their effect on pain for relatively short-term treatment only. For example, a meta-analysis and a systematic review evaluated the effectiveness of opioids used to treat patients with chronic non-malignant pain.[3,4] The studies included in the analyses showed that patients were treated with opioids for a mean of five weeks (range 1 – 16 weeks). Opioids, which included oxycodone, morphine, fentanyl, tramadol and codeine, were associated with a modest short-term analgesic benefit, however, the authors cautioned that this finding should not be extrapolated to long-term treatment with opioids.[3,4] Opioid-induced hyperalgesia and tolerance have been found to be major limiting factors for long-term opioid treatment.[5] Another systematic review (that included 21 randomised studies) found that there was no evidence that opioids (including oxycodone, morphine and tramadol) were effective in managing chronic non-malignant pain in any of the conditions studied (including back pain and osteoarthritis). The only exception was "intermediate/fair" evidence for tramadol in patients with osteoarthritis.[6]

Opioids are associated with significant adverse events

The use of opioid treatment for the management of chronic non-malignant pain is associated with significant adverse events that affect multiple organ systems. These adverse events can occur with any use of opioids, but there is an increased risk in patients who use opioids long term.

Adverse effects of opioids include:[2]

- **Respiratory system** – respiratory depression, obstructive and central sleep apnoea, ataxic breathing, respiratory arrest and death
- **Central nervous system** – increased risk of falls, cognitive impairment, myoclonus, delirium, depression, somnolence and sleep disorders
- **Cardiovascular system** – orthostatic hypotension, bradycardia, vasodilation and an increased risk of cardiovascular events, e.g. myocardial infarction
- **Gastrointestinal system** – constipation, nausea and vomiting, gastric reflux, delayed gastric emptying, abdominal cramping and distension

Principles for managing patients with chronic non-malignant pain

- Communicate and listen to the patient and empower them to take a leading role in the management of their condition
- Focus on improving function and disability rather than just concentrating on pain outcomes
- Ensure that the patient has realistic expectations regarding treatment. Controlling or reducing pain rather than total elimination of pain is usually the goal.
- Treat any co-morbidities that are frequently associated with chronic pain, e.g. anxiety and depression. Non-pharmacological treatments, such as cognitive behavioural therapy and exercise, can play a major role in managing the psychological co-morbidities of pain. Short-term use of pharmacological treatments, e.g. selective serotonin reuptake inhibitors (SSRIs), can also be considered.
- Educate the patient that remaining active will be beneficial in managing their pain and encourage them to continue to do activities that bring enjoyment. A positive attitude or outlook can reduce the patient's perception of their pain. Focus on what the patient can do, as opposed to what they cannot do.
- If pharmacological treatment is used to manage pain, always have a plan to taper the dose (even if this is long term) and avoid increasing doses to "chase pain"

- **Immune system** – decreased wound healing, pruritus, altered cytokine production, increased histamine release, inhibition of macrophage, neutrophil and natural killer cell activity and recruitment, increased HIV replication and cancer progression
- **Endocrine system** – opioid-induced endocrinopathy (usually only with high opioid doses, long term), resulting in decreased libido, testicular atrophy, early menopause and sexual dysfunction

The sedative effects of opioid treatment can also add to psychological factors that patients with chronic non-malignant pain may be experiencing, and exacerbate feelings of helplessness and depression.

Opioids have high addiction rates

The rates of opioid misuse and addiction reported in the literature vary greatly for patients with chronic non-malignant pain. This is possibly due to different definitions and methods of measuring addiction and misuse. One systematic review reported that the rate of opioid addiction/misuse was relatively low (approximately 3%) but the rate of aberrant behaviour was much higher (approximately 12%) in patients with chronic non-malignant pain who received long-term opioid treatment.[7] However, other studies have reported much higher addiction/misuse rates. The retrospective TROUP study which investigated a number of factors associated with long-term opioid use, reported possible opioid misuse in 20% – 24% of patients with chronic non-malignant pain and probable misuse in 3% – 6%.[8] Another study reported even higher rates, with approximately 35% of patients with chronic non-malignant pain fitting the Diagnostic and Statistical Manual for Mental Disorders – fifth edition (DSM-V) criteria for a prescription opioid use disorder during a lifetime.[9]

Other treatment options are available

Clinicians may have a misconception that opioids are the only treatment option available for patients with chronic non-malignant pain. This can result in inappropriate prescribing of opioids, including switching patients from other treatments, e.g. NSAIDs, to opioids, which is generally not appropriate. Clinical judgement and individualised prescribing, which takes into consideration the risk and benefits of all treatments, are essential in managing patients with chronic non-malignant pain. Focusing solely on pharmacological treatments for these patients should be avoided.

For further information see **www.aci.health.nsw.gov.au**

Recognising the importance of the patient's emotional wellbeing

Psychological factors have been shown to play a major role in how patients experience and tolerate pain, but are often not considered when management plans for chronic non-malignant pain are implemented.[10] Recent research in patients with chronic pain has identified dysfunction and dysregulation in a several key brain structures.[11] This dysfunction is associated with changes in the patient's emotional and cognitive functioning, including increased activity, anxiety, depression, fear, addiction, altered attention and cognition (Figure 1).[11] These changes are also related to the phenomenon of "pain catastrophising", which can be defined as repetitive negative thoughts during actual or anticipated pain.[10] Pain catastrophising has been recognised as one of the major psychological determinants of the negative outcomes associated with chronic non-malignant pain.[10]

Clinical experience has shown that a "collaborative partnership" approach between patient and clinician is best when managing chronic non-malignant pain. For most patients, it is essential to them that the clinician believes that they are experiencing pain and recognises that their life has been significantly changed by this pain.[12]

Patients frequently report an "adversarial struggle" within themselves or with others when dealing with chronic non-malignant pain, which can result in:[12]

- A struggle with self-perception and self-worth – the patient may describe feeling alienated from their body and that they cannot meet other people's expectations and hide their pain in an attempt to appear normal
- Altered perceptions of the future – the day-to-day unpredictability of pain can mean that the patient changes their plans, expectations and dreams for the

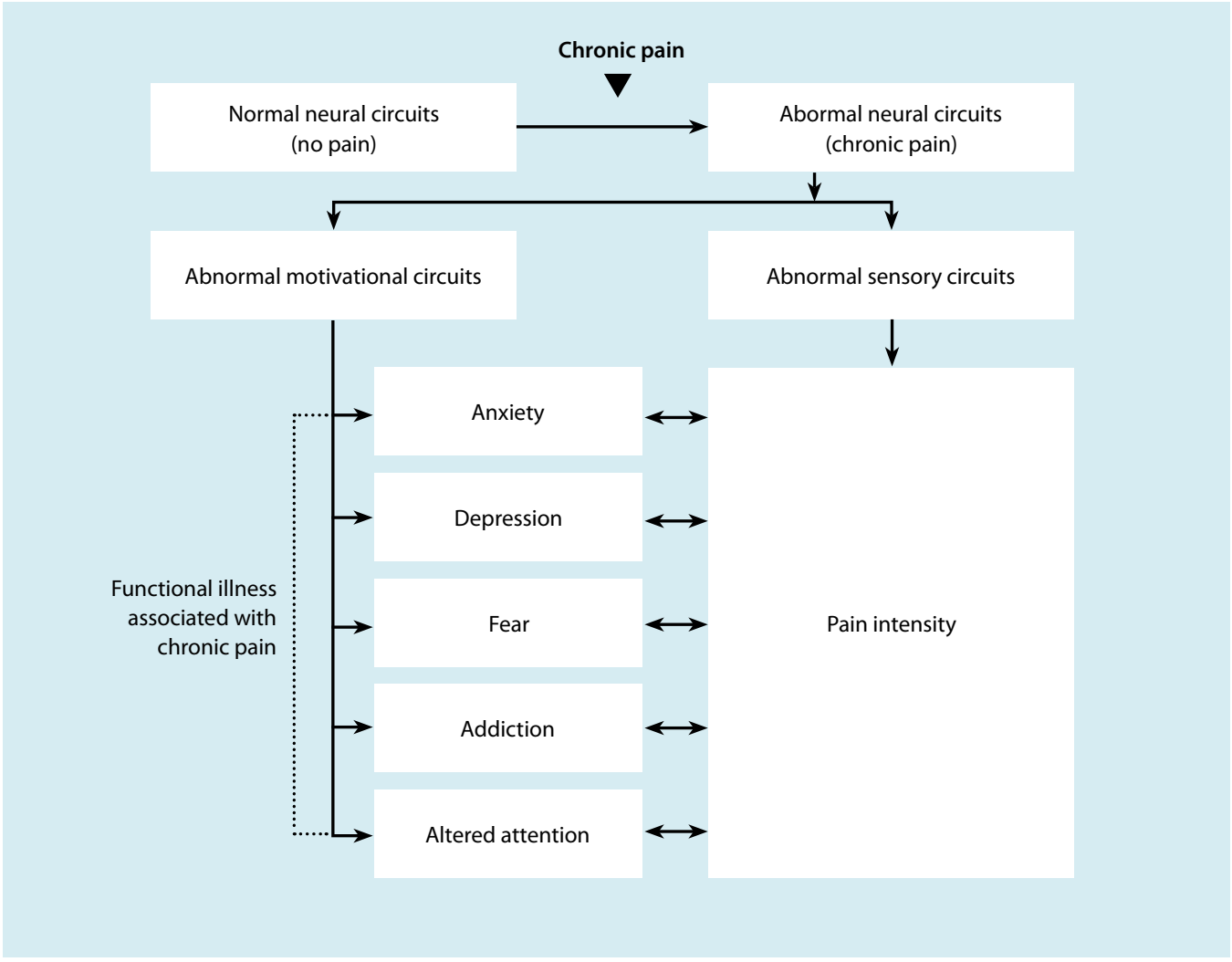


Figure 1: Chronic pain results in changes to emotional state, with resultant psychological symptoms. These effects are bi-directional, i.e. negative emotional states can augment the perceived intensity of pain. Adapted from Elman *et al*, 2011.[11]

future, resulting in an inwardly focused perspective on life

- A feeling that people do not understand or believe their pain – resulting in emotions of worthlessness, fear, guilt and doubt, which may influence the patient's work and relationships, as well as impacting on their likelihood of seeking help
- Problems with negotiating the healthcare system – the patient may feel as if they are being referred back and forth between clinicians and they are "trapped in the system"

Clinicians should aim to counsel the patient through these adversarial struggles and help them to move forward "alongside their pain".

Ways to achieve this include:[12]

1. Encourage the patient to recognise the type, intensity and duration of pain they are feeling and how this can vary throughout the day and between days. The aim is for the patient to feel increasingly more in control of their body and their pain.
2. Encourage the patient to redefine a "new normal" that does not focus on the losses which the pain has caused but reinforces positive self-images and plans for the present and the future
3. Encourage the patient to become part of a group and to share their pain experiences with others. This can help them realise that they are not the only person dealing with pain issues.
4. Reassure the patient that they do not have to hide their pain or seek the approval of others (i.e. convincing others that their pain is real). Patients should be encouraged to work with their pain to accomplish achievable and realistic goals and not to set goals based on other people's expectations.
5. Ensure that the patient understands that there may be no cure for their pain and that managing their pain and improving function are the goals of their management plan.
6. Help the patient to understand their pain condition and take a more active role in their health care. Patients should be given the confidence to experiment with different methods of managing their pain and the opportunity to make their own decisions about their treatment.

Pain is often complicated by a number of other factors, including anxiety, depression, substance use disorders and sleep difficulties.[13] Managing these co-morbidities is essential in gaining overall control of the patient's pain condition.

Further reading: Toye F, Seers K, Allcock N, et al. A meta-ethnography of patients' experience of chronic non-malignant musculoskeletal pain. Health Serv Deliv Res 2013;1(12). Available from: **www.journalslibrary.nihr.ac.uk/__data/assets/pdf_file/0010/94285/FullReport-hsdr01120.pdf**

Finding treatments for pain

When managing patients with chronic non-malignant pain, the aim is to maximise use of non-pharmacological treatments and non-opioid analgesics, and to avoid using opioid analgesics where possible. Most patients can be managed in primary care, but discussion with, or referral to, a specialist pain clinic may be required in some cases. This may include patients with pain that is difficult to treat or when multiple treatment failures have occurred.

A treatment approach that incorporates both pharmacological (non-opioid) and non-pharmacological interventions is recommended. This method has been found to be more effective in managing chronic pain than single treatment modalities. This is supported by a 2008 systematic review, that included 35 randomised studies (2407 patients), which investigated the use of multidisciplinary treatments* in patients with chronic musculoskeletal pain (mostly chronic back pain or fibromyalgia). The review reported that there was "moderate" evidence of better effectiveness of multidisciplinary treatments compared to single treatments in the treatment of this patient group.[14]

There are a wide range of social, psychological, non-pharmacological and non-opioid pharmacological treatment options available for patients with chronic non-malignant pain. The best combination of treatments will vary between patients depending on a number of factors. These include the underlying pain complaint, e.g. nociceptive versus neuropathic pain, the mind-set and demographics of the patient, e.g. older and younger patients may have different expectations and preferences for different treatments, the severity and duration of the pain, and the availability and affordability of different treatment options. It may be necessary to trial different combinations of treatments in order to find the best combination that suits the individual patient.

* Multidisciplinary treatments in the studies included cognitive behavioural therapy (CBT), psychotherapy, exercise programmes (including stretching and hydrotherapy), patient education, muscle relaxation, nutritional counselling, and vocational and occupational therapy.

Non-pharmacological treatment options for chronic pain

Exercise therapy

Physical activity is beneficial for people with pain as it can improve, or stop deterioration, in a number of parameters, including range of motion and flexibility, and the pain associated with these. The choice of exercise programme will vary depending on the patient's pain condition and physical capabilities. A patient may choose a structured exercise programme, or may prefer self-directed activities such as walking or swimming; these activities may be particularly beneficial in patients with osteoarthritis of the lower limbs or chronic back pain. Patients who are initially reluctant to begin exercise can be advised to gradually increase their level and duration of activity.

A Cochrane systematic review reported that exercise therapy was slightly effective in decreasing pain and improving function in adults with chronic low-back pain, and at least as effective as other conservative treatments, e.g. behavioural approaches.[15] The positive effects of exercise programmes were most pronounced in patients who presented to healthcare providers and received individually-designed programmes that commonly included strengthening or trunk-stabilising exercises.[15]

Pilates: A systematic review concluded that regular sessions of pilates (one to three times per week) resulted in greater improvements in pain and function than usual care and physical activity in the first 4 – 15 weeks in patients with chronic low-back pain.[16]

Yoga: A randomised trial that investigated the efficacy of the addition of yoga to usual care in patients with chronic low-back pain found that pain and function were both improved (at three, six and 12 months) in patients who underwent at least three yoga sessions.[17]

Tai Chi: A systematic review found that regular sessions of Tai Chi (on average one to two times per week for 6 – 15 weeks) had small positive short-term effects on pain and disability in patients with chronic musculoskeletal pain due to arthritis.[18] However, the studies included were generally of low quality.

Brisk walking and home-based quadriceps strengthening exercises have both been reported to significantly reduce pain and disability in patients with osteoarthritis of the knee.[19] Weight reduction in overweight patients with osteoarthritis of the knee has also been shown to improve pain and function scores.[20]

Massage

Massage therapy may have some benefits compared with placebo and relaxation in patients with chronic low-back pain in the short term, according to the results of a systematic review.[21] However, there were conflicting and contradictory findings regarding the effectiveness of massage therapy when compared to other manual therapies (such as mobilisation) and acupuncture.[21] The use of topical rubefacients during massage can also be recommended, e.g. heat rubs.

Acupuncture and nerve stimulation techniques

A systematic review and meta-analysis reported that acupuncture improved pain outcomes in patients with four chronic pain conditions – back and neck pain, osteoarthritis, chronic headache and shoulder pain.[22]

Transcutaneous electrical nerve stimulation (TENS) is a form of nerve stimulation for pain relief and involves delivery of low-voltage electrical current to the skin via surface electrodes. However, systematic reviews have found variable and inconclusive results for TENS in patients with chronic pain.[23]

Cognitive behavioural therapy (CBT)

CBT (individual or group) is one of the more commonly used behavioural approaches for treating patients with chronic pain. CBT focuses simultaneously on the environment, behaviour and cognition. The efficacy of CBT has been investigated in a number of chronic pain conditions including fibromyalgia and low back pain. A randomised study conducted in patients with chronic low-back pain in England reported that six sessions of group CBT resulted in significantly better pain and disability scores ($p < 0.001$ for both) compared with the control group (no CBT).[24] Another study reported that CBT improved the patient's ability to cope with pain, reduced depressive moods and reduced the number of follow-up appointments in patients with chronic pain due to fibromyalgia, but had no significant effects on the actual pain, fatigue, sleep and quality of life.[25]

Cognitive behavioural therapy for pain

The principle behind CBT is in examining the relationship between a person's thoughts, feelings and behaviours, and understanding that these factors are dependent on each other.

The patient may begin with:
"If I move, I will hurt more" (thoughts)
"This makes me feel anxious about doing anything" (feelings)
"I will avoid doing anything that might hurt" (behaviour)

This then progresses to:
"No one cares about my pain, and no one can fix me" (thoughts)
"I feel angry that no one cares, and fearful that I cannot be fixed" (feelings)
"This makes me tense and irritable" (behaviour)

The purpose of CBT is to help patients avoid feeling overwhelmed by the pain they are experiencing, and instead come to terms with their pain and feel that it is manageable. This means that the patient moves from a passive to an active role in their care, focusing on increasing their function and quality of life.

The goals of the clinician are to:

- Actively listen to the patient's experience of their pain
- Provide education about the cause of pain (if possible) and possible treatments
- Help patients find additional resources and support groups
- Set goals for the patient to achieve
- Solve problems that happen along the way
- Encourage engagement
- Positively reinforce any successes

For further information, see: Promoting mind-body approaches to pain self-management, by Debra Hughes. Available from: **www.empr.com**

The access to, and cost of, CBT in New Zealand varies throughout the country and can be a significant barrier to treatment. Some primary care clinicians may be trained in this technique, but referral to a Clinical Psychologist or Pain Specialist may be required.* When access to specialist CBT is not possible, there are some internet-based programmes available which have been shown to be effective in helping patients manage their pain (see below for details). A US-based study that examined the effectiveness of an internet-based CBT chronic pain management programme (mostly in patients with joint, back and osteoarthritic pain) reported positive results.[26] The study found that pain intensity was significantly reduced from baseline after both one and six months, and quality of life was also improved after six months.

An example of on online CBT programme that can be recommended for patients is available at: **www.getselfhelp.co.uk/chronicfp.htm**

Other treatment options and useful advice that can be given to patients

Other non-pharmacological treatment options for chronic non-malignant pain that can be considered include:

- Hot or cold compresses, depending on the pain condition and specific benefit, e.g. hot packs can be beneficial in patients with chronic back pain and cold packs can be beneficial in patients with pain due to osteoarthritis of the knee
- Biofeedback (the process of gaining greater awareness of many psychological functions, e.g. pain perception) and mind-body activities such as meditation, mindfulness and relaxation can also be considered, mostly in combination with other treatments
- Encourage the patient to engage in activities they enjoy or that make them laugh
- Referral to an Occupation Therapist who can assist with postural problems, e.g. in a patient with a repetitive strain injury due to work
- Referral to a Physiotherapist, Chiropractor or Osteopath who can perform massage, strapping, mobilisation and manipulation (where appropriate)

* The Aotearoa New Zealand Association for Cognitive Behavioural Therapy (AnzaCBT) offer courses and workshops on CBT, and more information is available at: **www.cbt.org.nz**

Pharmacological treatment options for chronic pain

Pharmacological treatment should not be the sole focus in managing patients with chronic non-malignant pain and should be used in combination with non-pharmacological interventions. As with non-pharmacological treatments, the most appropriate treatment (or combination of treatments) will vary between patients, and individual treatment trials should be undertaken. When undertaking a trial, use the pre-intervention level of pain and function to assess whether the medicine(s) is working.

Analgesic treatment options for chronic non-malignant pain may include*:[27]

- Paracetamol
- NSAIDs: naproxen (up to 1000 mg per day) or ibuprofen (up to 1200 mg per day) are the recommended first-line choices if NSAIDs are required for longer periods of time, due to the lower risk of cardiovascular events occurring when these medicines are taken at these doses, compared to other NSAIDs.[28] N.B. ibuprofen may be taken up to 2400 mg per day, but this is associated with increased cardiovascular risk.
- Tricyclic antidepressants, e.g. amitriptyline, nortriptyline (less sedating)
- Other neuromodulators, e.g. gabapentin, carbamazepine
- Topical analgesics, e.g. NSAIDS, capsaicin

Referral to secondary care to investigate surgical options, permanent nerve blocks, epidural steroid injections and spinal cord stimulation may be appropriate for some patients.

The use of opioids in chronic non-malignant pain

Opioids have a limited role in the treatment of chronic non-malignant pain and should only be used after other treatment options have failed. When considering using any opioid treatment it is recommended that there are strict protocols in place to minimise the associated risks. One method that has been proposed for the safe use of opioids for chronic non-malignant pain is the "10 universal precautions" approach (see: "The 10 Universal Precautions approach to pain management", over page).

When opioids must be used some considerations include:

- Use the weakest opioid possible, e.g. use codeine or tramadol before considering morphine
- Use opioids for the shortest possible time at the lowest possible dose
- Have a plan in place to decrease the opioid dose, e.g. ensure the patient knows that the dose will gradually be stepped down and then ceased
- Regularly review opioid treatment for efficacy, tolerability and signs of addiction. Re-evaluate opioid treatment at every consultation and only continue treatment if there is a very good reason for doing so.
- Have a system in place to identify and manage opioid misuse and addiction

Weaker/atypical opioid treatment options

Codeine, tramadol and dihydrocodeine can be considered as treatment options in combination with non-pharmacological and non-opioid analgesics in patients with chronic non-malignant pain.

Codeine is a pro-drug which is metabolised to morphine by the liver enzyme CPY2D6 to achieve its analgesic effect. Genetic differences mean that there is variation in how people metabolise codeine (either fast or slow metabolisers). Dihydrocodeine is similar to codeine in both its structure and analgesic effect. Tramadol is classed as an "atypical" opioid as it is both a relatively-weak mu opioid receptor agonist and a noradrenaline and serotonin reuptake inhibitor.[29]

Codeine, dihydrocodeine and tramadol are not recommended for use in patients with renal impairment. Use of all opioids is associated with constipation, but this can be particularly problematic with codeine. Co-prescription of a laxative is recommended. Tramadol may be more associated with nausea, vomiting, dizziness and sedation than codeine.

Strong opioids are ideally a "last resort"

When all other treatment options have failed, the clinician may decide that a strong opioid is the only treatment option available when the patient has moderate to severe chronic non-malignant pain. When a strong opioid is indicated, morphine is the first-line choice. Fentanyl patches are sometimes considered in patients with severe chronic pain. However, they are best reserved for patients with constant and stable opioid requirements.

* A number of these medicines are not subsidised or approved for use in pain management in New Zealand. For example, tricyclic antidepressants are not approved for neuropathic pain (but are frequently used for this indication) and capsaicin is subject to subsidy restrictions. Pregabalin and duloxetine are sometimes used for chronic non-malignant pain, but are not subsidised in New Zealand. Refer to the New Zealand Formulary for further information on approved indications and subsidies.

Take home messages

- Chronic non-malignant pain takes time to treat and the management plan needs to include not only physical treatments, but also acknowledgement of the patient's pain and emotional wellbeing, and support to help them self-manage their condition
- Use combinations of non-pharmacological interventions and non-opioid analgesics as the mainstay of treatment
- Only use opioid analgesics as a last resort
- If it is absolutely necessary to use opioids, consider weaker opioids such as codeine or tramadol before using strong opioids such as morphine, and use the opioid at the lowest possible dose, for the shortest possible time

Coming up: In the next edition we look at the growing problem with opioid addiction in New Zealand, and discuss strategies for withdrawing patients from opioids.

The 10 Universal Precautions approach to chronic pain management

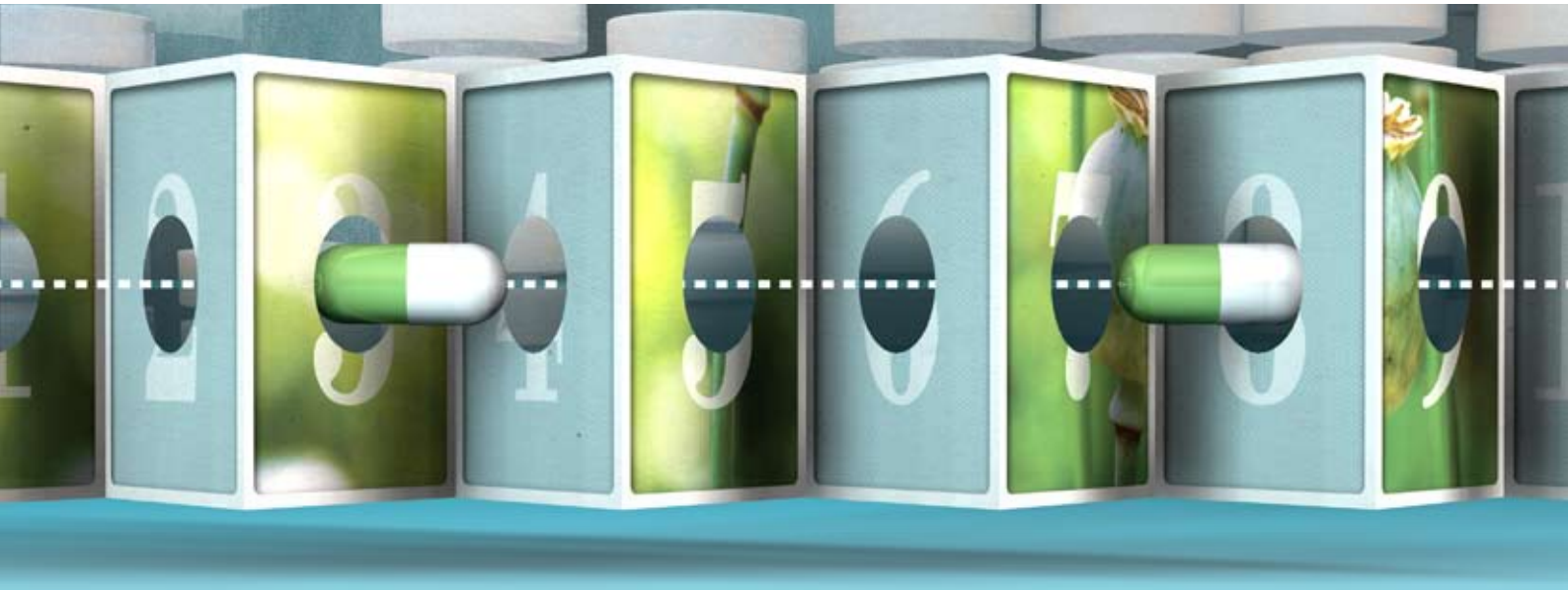
The "10 universal precautions" are a set of guiding principles which can be applied to the management of long-term pain. Opioids are not recommended for long-term use when treating chronic non-malignant pain. However, if there is no other treatment option and they must be used, these principles can help determine which patients may be at risk of opioid misuse, to guide opioid treatment and ensure appropriate review.[30]

The 10 Universal Precautions are:[30]

1. **Aim to diagnose the underlying cause of the pain**, considering differential diagnoses. If there is no clear diagnosis, and an absence of objective findings, treatment can be initially aimed at managing the patient's symptoms. If the pain persists the patient should be reassessed for a diagnosis, and their analgesic requirements reviewed, with the aim of stepping down from the use of a strong opioid, if appropriate.

2. **Conduct a comprehensive psychological assessment** including the risk of addiction. Question the patient about past or present alcohol or illicit drug use. In addition ask about any family history of substance misuse or addiction (including alcohol) as this increases the risk that the patient may misuse opioids. Other psychological factors, such as the patient's expectations and mood, and social aspects, e.g. sleep, work, family and social support should also be considered.

3. **Gain informed consent** from the patient. Discuss the proposed treatment with them, including the anticipated benefits and the possible adverse effects and risks of physical dependence, tolerance and addiction. Ensure the information has been delivered at an appropriate level and that the patient understands the information that has been discussed. Some patients may wish to include family members, a support person or caregivers in the decision making process.



4 **Obtain a treatment agreement**. The concept of universal precautions relies on clear communication between the clinician and the patient and is ideally based on mutual trust and respect. The expectations and obligations of both the patient and clinician need to be clearly understood and either agreed verbally or more formally in a written treatment agreement or opioid contract.*

5 **Record a measure of the pre- and post-intervention pain level and function**. In order to assess the success of a treatment trial, it is necessary to have a baseline measure of the patient's pain (e.g. pain score) and level of function. These aspects can then be monitored and documented periodically during treatment and at the conclusion of the trial treatment period, to determine whether functional goals have been met and pain has been reduced. This then forms the basis of a decision on continuation of treatment.

6 **Conduct an appropriate trial of opioid treatment**, ideally with adjunctive medicines. Prescribing an opioid should not be routinely thought of as the first step when choosing a pain treatment. Before opioids are considered, ensure there has been an adequate trial of both non-pharmacological and other pharmacological treatments that are appropriate for the patient's condition.

7 **Regularly reassess the patient's pain scores and level of function**. A regular reassessment of the patient to check how well their pain is being managed and their level of functioning will help the clinician to decide whether to continue or modify the current treatment. Ensure that the patient has realistic expectations of the treatment, i.e. that they may have an increase in their level of function and their ability to cope, but not a complete resolution of their pain.

8 **Regularly assess the "5 A's" of pain management:** analgesia (how much relief has the medicine provided?), activity (progress in functional goals), adverse effects (especially constipation, nausea and sedation), aberrant behaviours (signs or suspicion of medicine misuse) and affect (impact of pain on mood and psychological wellbeing)

9 **Periodically review the pain diagnosis,** co-morbidities and addictive disorders. The underlying illness can evolve during treatment and it is important to periodically re-assess the original condition for which analgesia is being used. In addition, a patient's co-morbidities can influence the success of pain management strategies, so where possible, other conditions need to be optimally managed.

10 **Carefully document** every step of the patient's treatment protocol.

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* There are a number of standard opioid contracts available online, e.g. www.hnehealth.nsw.gov.au/__data/assets/pdf_file/0017/108701/Opioid_treatment_agreement_Mar_2013.pdf

www.wps.ac.nz/Portals/9/Documents/Opioid%20Contract%20formv2%200-2012.pdf www.icsi.org/_asset/dyp5wm/Opioids.pdf (Appendix A)

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References

1. Reconsidering opioid therapy - A Hunter New England perspective. Available from: www.aci.health.nsw.gov.au/__data/assets/pdf_file/0015/212226/Reconsidering_Opioid_Therapy_1.pdf (Accessed Aug, 2014).
2. Provenzano DA, Viscusi ER. Rethinking the role of opioids in the outpatient management of chronic nonmalignant pain. Curr Med Res Opin 2014;1–12.
3. Furlan AD, Sandoval JA, Mailis-Gagnon A, et al. Opioids for chronic noncancer pain: a meta-analysis of effectiveness and side effects. CMAJ Can Med Assoc J 2006;174:1589–94.
4. Manchikanti L, Ailinani H, Koyyalagunta D, et al. A systematic review of randomized trials of long-term opioid management for chronic non-cancer pain. Pain Physician 2011;14:91–121.
5. Ballantyne JC, Shin NS. Efficacy of opioids for chronic pain: a review of the evidence. Clin J Pain 2008;24:469–78.
6. Noble M, Treadwell JR, Tregear SJ, et al. Long-term opioid management for chronic noncancer pain. Cochrane Database Syst Rev 2010:CD006605.
7. Fishbain DA, Cole B, Lewis J, et al. What percentage of chronic nonmalignant pain patients exposed to chronic opioid analgesic therapy develop abuse/addiction and/or aberrant drug-related behaviors? A structured evidence-based review. Pain Med 2008;9:444–59.
8. Sullivan MD, Edlund MJ, Fan M-Y, et al. Risks for possible and probable opioid misuse among recipients of chronic opioid therapy in commercial and medicaid insurance plans: The TROUP Study. Pain 2010;150:332–9.
9. Boscarino JA, Rukstalis MR, Hoffman SN, et al. Prevalence of prescription opioid-use disorder among chronic pain patients: Comparison of the DSM-5 vs. DSM-4 Diagnostic Criteria. J Addict Dis 2011;30:185–94.
10. Flink IL, Boersma K, Linton SJ. Pain catastrophizing as repetitive negative thinking: a development of the conceptualization. Cogn Behav Ther 2013;42:215–23.
11. Elman I, Zubieta J-K, Borsook D. The missing p in psychiatric training: why it is important to teach pain to psychiatrists. Arch Gen Psychiatry 2011;68:12–20.
12. Toye F, Seers K, Allcock N, et al. Patients' experiences of chronic non-malignant musculoskeletal pain: a qualitative systematic review. Br J Gen Pract J 2013;63:e829–41.
13. Chronic Pain Syndrome. Available from: emedicine.medscape.com/article/310834-overview (Accessed Aug, 2014).
14. Scascighini L, Toma V, Dober-Spielmann S, et al. Multidisciplinary treatment for chronic pain: a systematic review of interventions and outcomes. Rheumatology 2008;47:670–8.
15. Hayden JA, van Tulder MW, Malmivaara A, et al. Exercise therapy for treatment of non-specific low back pain. Cochrane Database Syst Rev 2005:CD000335.
16. Wells C, Kolt GS, Marshall P, et al. The effectiveness of pilates exercise in people with chronic low back pain: a systematic review. PloS One 2014;9:e100402.
17. Tilbrook HE, Cox H, Hewitt CE, et al. Yoga for chronic low back pain: a randomized trial. Ann Intern Med 2011;155:569–78.
18. Hall A, Maher C, Latimer J, et al. The effectiveness of Tai Chi for chronic musculoskeletal pain conditions: A systematic review and meta-analysis. Arthritis Rheum 2009;61:717–24.
19. Roddy E. Aerobic walking or strengthening exercise for osteoarthritis of the knee? A systematic review. Ann Rheum Dis 2005;64:544–8.
20. Christensen R, Bartels EM, Astrup A, et al. Effect of weight reduction in obese patients diagnosed with knee osteoarthritis: a systematic review and meta-analysis. Ann Rheum Dis 2006;66:433–9.
21. Kumar S, Beaton K, Hughes T. The effectiveness of massage therapy for the treatment of nonspecific low back pain: a systematic review of systematic reviews. Int J Gen Med 2013;733.
22. Vickers AJ, Cronin AM, Maschino AC, et al. Acupuncture for chronic pain: individual patient data meta-analysis. Arch Intern Med 2012;172:1444–53.
23. Nnoaham KE, Kumbang J. Transcutaneous electrical nerve stimulation (TENS) for chronic pain. Cochrane Database Syst Rev 2008;CD003222.
24. Lamb SE, Hansen Z, Lall R, et al. Group cognitive behavioural treatment for low-back pain in primary care: a randomised controlled trial and cost-effectiveness analysis. Lancet 2010;375:916–23.
25. Bernardy K, Füber N, Köllner V, et al. Efficacy of cognitive-behavioral therapies in fibromyalgia syndrome - a systematic review and metaanalysis of randomized controlled trials. J Rheumatol 2010;37:1991–2005.
26. Nevedal DC, Wang C, Oberleitner L, et al. Effects of an individually tailored Web-based chronic pain management program on pain severity, psychological health, and functioning. J Med Internet Res 2013;15:e201.
27. Liebschutz J, Beers D, Lange A. Managing Chronic Pain in Patients with Opioid Dependence. Curr Treat Options Psychiatry 2014;1:204–23.
28. National Institute for Health and Care Excellence (NICE). Non-steroidal anti-inflammatory drugs. NICE, 2013. Available from: www.nice.org.uk (Accessed Aug, 2014).
29. Kizilbash A, Ngô-Minh CT. Review of extended-release formulations of tramadol for the management of chronic non-cancer pain: focus on marketed formulations. J Pain Res 2014;7:149–61.
30. Gourlay DL, Heit HA, Almahrezi A. Universal precautions in pain medicine: a rational approach to the treatment of chronic pain. Pain Med 2005;6:107–12.

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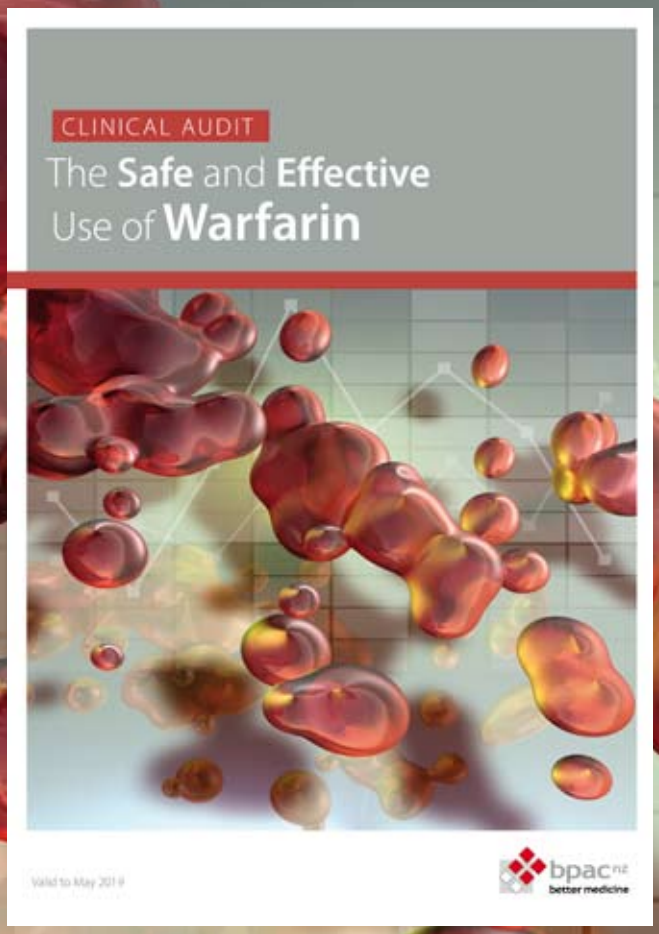

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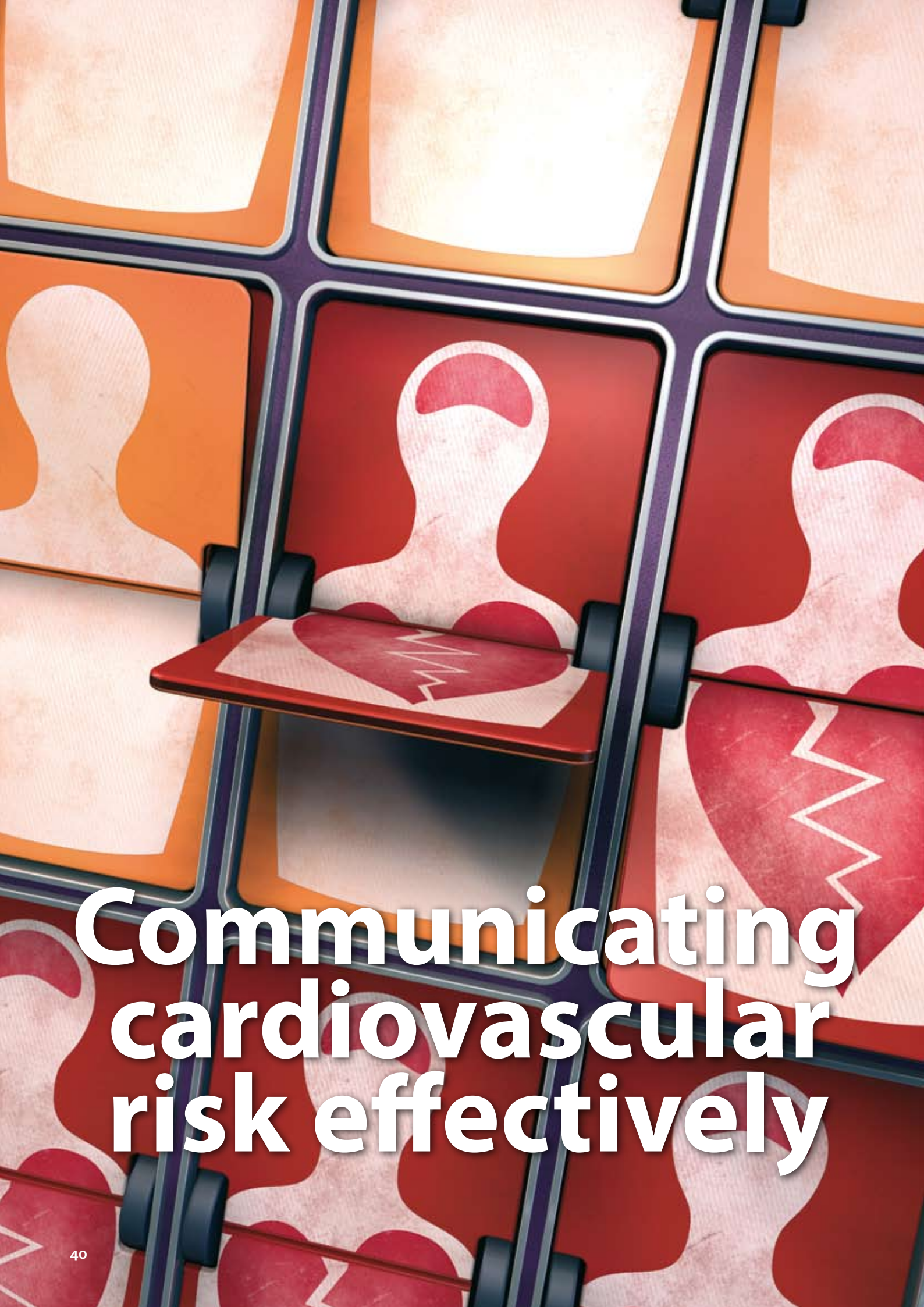

Communicating cardiovascular risk effectively

Calculating a patient's cardiovascular risk is relatively easy; communicating this to patients in a way that assists their decision making can be challenging. This is because patients and health professionals often think differently about cardiovascular risk. To empower decision making and self-efficacy among patients clinicians can choose to frame information in a variety of different ways.

The importance of calculating cardiovascular risk

Cardiovascular risk management is central to general practice. The current approach to this involves calculating the patient's cardiovascular risk, helping patients to understand what their risk means, and empowering patients, especially those with high cardiovascular risk, to make changes to reduce their risk. During these discussions if information is not delivered in a way that the patient understands then their ability to make informed decisions that are beneficial to their health is likely to be limited.

Calculating cardiovascular risk in New Zealand

In New Zealand, an equation based on Framingham data is used to calculate combined five-year cardiovascular risk. While this equation is not a perfect predictor of outcomes, it is very good at identifying those patients who are at a higher risk of experiencing a cardiovascular event.[1] The five-year cardiovascular risk combines key factors into an overall risk. This is intended to assist patient understanding, and allows clinicians to have an overview of the patient's health, rather than focusing on individual risk factors. The goal of the health professional is to help the patient to lower their overall cardiovascular risk and to place the patient's cardiovascular risk in the context of other co-morbidities, e.g. chronic obstructive pulmonary disease (COPD).

Many General Practitioners have validated electronic cardiovascular calculators embedded into their decision support tools, e.g. *bestpractice* or Predict, which enable rapid calculation of cardiovascular risk.* Furthermore, guidelines are relatively clear in the management of individual cardiovascular risk factors. However, increasing a patients understanding of cardiovascular risk and empowering them to use this information to make decisions is arguably the greatest challenge in the management of cardiovascular health. This is because many people base their assessment of risk on emotions, that will influence their decisions rather than data.[2] The task of communicating risk is further complicated because as many as one in five well-educated people incorrectly interpret basic statistical information.[3]

* Validated electronic cardiovascular calculators are not available in all regions. The New Zealand Cardiovascular Risk Charts can be used by clinicians in place of electronic calculators. These are available online at **www.health.govt.nz/system/files/documents/publications/090311_cvd_poster_final.pdf** or in the New Zealand Primary Care Handbook 2012.

Shared decision-making is central to risk management

Discussions with patients about treatment options for cardiovascular disease are best managed with a shared decision-making approach. This involves the clinician using their knowledge and skills to enable the patient to arrive at a decision which best fits the patient's values and priorities. This process takes many factors into account, including:

- The patient's age, ethnicity, co-morbidities and frailty
- The benefits versus harms of any interventions
- The patient's family/whānau
- Current evidence-based guidelines
- The patient's internal concerns, beliefs, expectations and values
- The health professional's clinical experience
- The patient's occupation, hobbies and commitments
- The patient's socioeconomic and occupational status which may limit their ability to meet the cost of appointments, travel to clinics or take time-off work to attend consultations
- The patient's willingness to consider change at this stage

Patients are more likely to take responsibility for managing their own health if they are actively engaged in treatment decisions and their family/whānau are encouraged to be involved.[4] Being actively involved in self-management means patients are also more likely to be satisfied with their treatment.[4] For shared decision-making to be meaningful patients need to understand the reasons why health professionals are making recommendations. Therefore discussions with patients about cardiovascular risk are crucial. These become even more important when the balance between the advantages and harms of a treatment are finely weighted, e.g. treating patients with statins when their cardiovascular risk is moderate; a strong patient preference for longevity of life or avoidance of adverse effects may be the difference between treating, or not treating.

Discussing cardiovascular risk with patients

Following a cardiovascular risk assessment all patients should be given the opportunity to discuss their result, regardless of their level of cardiovascular risk.[5] Some patients may appreciate being offered the opportunity to do this with whānau/family being present. The outcomes of this discussion will be influenced by the ability of the health professional to deliver information so that it is understandable to the patient. Health professionals who do this well will naturally adjust the complexity of the discussion and use different tools to explain concepts, as appropriate to the individual patient.

There are no clear recommendations about how risk should be communicated to patients, because each individual will interpret information differently and each representation of risk carries its own connotations and biases, e.g. absolute versus relative risk.[6] In psychological studies, how risk-related information is presented to patients has been repeatedly shown to influence how risk is perceived, and to a lesser extent this effect has also been demonstrated in clinical encounters.[7] Risk perception is vitally important because it, not clinical assessments, forms the basis for patient decision-making.[8]

Health professionals and patients may think differently about risk

Health professionals deal with the concept of cardiovascular risk daily. However, explaining this concept to people who are not familiar with it can be challenging because:

1. It is an abstract concept that does not apply to the present, but rather to an unspecified point in time at some stage in the next five years
2. Even those at high five-year risk of a cardiovascular event, e.g. 25%, are still unlikely to experience an event in the next five years, i.e. there is a 75% chance that they will not experience an event

Cardiovascular tools should therefore be used as a prompt for discussions about cardiovascular risk, with clinical expertise helping individual patients understand their risk.

First define what you mean by cardiovascular event

In New Zealand cardiovascular events are defined as a diagnosis of: myocardial infarction, new angina, ischaemic stroke,

The morbidity and mortality of cardiovascular disease

Cardiovascular events are the leading cause of mortality in New Zealand, accounting for almost one-third of deaths annually; every 90 minutes one New Zealander dies of coronary artery disease.[11] Stroke is the leading cause of disability among adult New Zealanders; seven out of ten patients that survive a stroke will be disabled long-term.[11]

transient ischaemic attack (TIA), peripheral vascular disease, congestive heart disease or cardiovascular-related death.[9] Depending on the patient's experience and level of health literacy, terms such as stroke, heart attack and cardiovascular disease can mean different things.[10] For example, if a patient has a relative who has had only a minor TIA then they may not be overly concerned about their likelihood of experiencing a similar event. Presenting a balanced and accurate picture of the morbidity and mortality associated with cardiovascular events will help some patients to make informed decisions (see: "The morbidity and mortality of cardiovascular disease", opposite).

Distinguishing between modifiable and non-modifiable risk factors

For patients who are at increased risk of cardiovascular disease due to non-modifiable factors, e.g. age or an early family history of ischaemic heart disease, it is important to acknowledge that these are outside the control of the patient and yet may contribute substantially to their risk.

Conversely, it is important that patients understand there are a number of modifiable risk factors that they can alter to improve their health. A series of interviews with 25 Māori patients in Northland with ischaemic heart disease, found that Māori patients were often aware of family histories of cardiovascular disease and that there was a genetic component to cardiovascular risk.[12] However, it was also found that these patients had less of an understanding of the impact of lifestyle factors on cardiovascular risk.[12] This small study shows the need to explore patients' awareness of the factors that contribute to their cardiovascular risk.

Present risk as statements rather than probabilities

Presenting risks as frequency statements, rather than single event probabilities, has been shown to reduce the likelihood of information being misunderstood.[13] For example, if the patient has a five-year cardiovascular risk of 15%, then it is more useful to tell them that 15 out of 100 patients like them will experience a cardiovascular event over the next five years. Doing this removes the potential for confusion over reference classes.[13] For example, some patients may think that a 15% risk of a cardiovascular event refers to a 15% heart attack, which they may perceive as a mild or small heart attack, while other patients may think there is a 15% chance they will have a heart attack every day, and become highly anxious.

Negotiating risk reduction with patients

The overarching principle of cardiovascular risk management is that those at higher risk have the greatest potential to gain from interventions. However, many patients struggle with this principle because some of the factors that contribute to cardiovascular mortality are insidious and asymptomatic, e.g. hypertension. From the patient's perspective it may seem logical to take a medicine to treat a symptom such as pain, however, they may be significantly more reluctant to take a pill everyday for dyslipidaemia if "I am feeling fine." Converting patients from a "how I feel" approach, to a more prognostic "how long will I live" view of their own health is frequently reported to be one of the greatest challenges in working alongside patients with long-term conditions.[14] This task is made more difficult by the fact that many interventions also involve the risk of symptomatic adverse effects, e.g. myalgia associated with statin use. However, for a trusting relationship to continue to evolve between patient and clinician the possibility of adverse effects of treatment should always be mentioned when discussing the pros and cons of treatment options. Explaining to patients that they are unlikely to feel an elevated blood pressure unless it is extreme is a simple way to begin conversations about risk factors that may be hidden to the patient. During consultations avoid descriptive terms, e.g. high-risk, which may have different meanings for different people and provide numeric examples with a consistent denominator where possible.[2]

Present a variety of treatment options to patients

When "making recommendations" it is easy for health professionals to narrowly suggest one course of action as outlined in a guideline, rather than helping patients see all of the options that are available. A wider perspective gives patients a number of options to consider, and for some, a sense of empowerment. When discussing the options available, the status quo, i.e. no change, is also a possibility. This "options approach" fits well with the process of informed consent, where patients are entitled to the risks and benefits of all reasonable options of care.

Patients may need time to consider their options

Many patients will be at the contemplative stage of change, and need time to think before deciding whether to begin a particular cardiovascular risk reduction treatment. This may involve discussion with family/whānau, or require more than one consultation with a health professional.

Remember that, for many patients, beginning long-term treatment for a chronic condition is a negative milestone in

The "Your Heart Forecast" tool

In 2008 the "Your Heart Forecast" tool was developed to support cardiovascular risk communication and this tool has now largely replaced the 2003 cardiovascular risk charts.[5] The "Your Heart Forecast" tool is designed to help health professionals explain to patients what their cardiovascular risk means. It provides a visual story for patients in four stages:[19]

1. First, the patient is provided with their current cardiovascular risk, i.e. you are here
2. The patient's cardiovascular risk is then compared to a peer with ideal modifiable risk factor control
3. The patient is then shown what will happen if they continue without making any changes in their life, i.e. their heart forecast
4. Finally, the patient is shown what would happen to their future risk if they made changes to their lifestyle

There is currently no patient outcome data available to assess the effectiveness of "Your Heart Forecast", although a questionnaire of 47 health professionals showed that the tool improved clinicians understanding of cardiovascular risk and increased their confidence in explaining cardiovascular risk to patients.[19]

The "Your Heart Forecast" online tool is available within New Zealand from: **www.heartfoundation.org.nz**

their life. Furthermore, the daily process of "taking pills" reminds them that they have a long-term medical condition. Denial and avoidance of "pill taking" can therefore be understood as natural human reactions in this context. Encouraging patients to think of pills as a positive step, helping them to live long enough to see grandchildren marry (or another goal that is important to them), may help to change this mindset.

Consider individual risk factors when deciding on the order of interventions

It is easier for a patient to achieve a clinically significant reduction in a risk factor that is very high than it is for one that is mildly abnormal.[5] Patients need to understand this when choosing between treatment options. For example, smoking cessation is likely to be of increased importance to a patient with elevated risk who has respiratory symptoms due to COPD. Lifestyle interventions can be presented as an alternative to medicines, e.g. "if you managed to lose a few kilograms by September I don't think there would be a need for you to start taking pills for hypertension." It will also often be necessary to treat multiple risk factors simultaneously.[5] Regardless of what level of cardiovascular risk a patient has they should be encouraged to exercise regularly, for example 30 minutes on most days.

Best Practice Tip: Visit our Facebook page (**www.facebook.com/bpacnz**) to comment on an excellent nine minute summary on the multiple benefits of exercise (also appropriate for patients to view): **www.youtube.com/watch?v=aUaInS6HIGo**

Graphical presentation improves understanding of risk

When presented with information about risk and probabilities people often pay more attention to the number of times an event happens (the numerator) and less attention to the number of opportunities it had to happen (the denominator).[15] This effect is referred to as denominator neglect.[15] A graphical representation is one way to overcome denominator neglect. A large systematic review of multiple studies found that the use of graphical presentations of information relating to health risks resulted in increased patient understanding and satisfaction.[16]

It is well established that graphical tools are more effective at conveying the benefits of cardiovascular interventions.[17] In a study of 100 patients in Auckland with a history of cardiovascular disease presented with information about a hypothetical medicine, the majority of patients who expressed a preference for how information was presented, preferred

to have it displayed graphically.[18] Relative risk was the next most preferred method of presentation, although presenting information in this form may be considered coercive (see opposite).[18] Interestingly, stating the number needed to treat (NNT) was the least preferred method of presentation. This suggests that while NNT may be a useful method of expression for clinicians, it may be less so for patients. NNT has also been found to decrease patient understanding of risk in other studies.[16]

The New Zealand Heart Foundation has provided two graphical online tools, one for health professionals (see: The "Your Heart Forecast" tool) and one for the general public. Both tools are designed to communicate, rather than calculate, cardiovascular risk.* The "Know Your Numbers" tool is available to all people and is intended for use without the support of a health professional. This tool does not include the physical effects of inactivity in its calculation, although it does recommend exercising on most days for 30 minutes.

* When calculating a patient's five-year cardiovascular risk it is important to use a validated decision-support tool, or the New Zealand Cardiovascular Risk Charts.

The "Know Your Numbers" tool is available from: **www.knowyournumbers.co.nz/**

Present absolute risk rather than relative risk

It has been shown that when patients are presented with information in the form of absolute risk they have an increased understanding, but are less likely to take action to reduce their risk based on this information.[16] Presenting the benefits of a cardiovascular intervention in terms of relative risk reduction is often more motivating for patients, but information presented solely in this manner can be easily misinterpreted,[2] raising the issue of informed consent.[7, 13, 18] Increasingly, it is being recommended that risk reduction should be presented in absolute terms, where possible.[2, 20] An example of the way that information about absolute risk reduction could be conveyed to a patient with a five-year cardiovascular risk of 20% who smokes would be: "If 100 people like you stopped smoking, then every five years there would be at least five fewer CVD events among these people*." However, clinical experience and patient knowledge will ultimately decide the preferred method of presenting information about cardiovascular risk to a patient. Checking with the patient that they have understood the intended meaning of the example is recommended whatever method of explanation is chosen.

* Presuming quitting smoking would reduce the patient's five-year cardiovascular risk by 5%

Figure 1: Graphic representation of a five-year cardiovascular risk of 20% and how the benefit of a reduction of risk to 15% may be explained to a patient, adapted from Paling, 2003,[2] available from **www.bmj.com/content/327/7417/745**

Framing affects perception

Framing refers to presenting logically equivalent information in different ways. This is an important concept when discussing cardiovascular risk with patients because although survival and mortality figures for a condition or procedure will be logically equivalent, e.g. 95% survival and 5% mortality, presenting only one or the other may result in markedly different results when patients are asked to make a treatment decision. A meta-analysis of four studies found that respondents were 1.5 times more likely to chose surgery over other treatments when surgery efficacy was framed in positive terms (percent survival), compared to negative framing (percent mortality).[21] It is recommended that when treatment effects are discussed with patients that health professionals express the information in more than one way, in order to present a balanced view and facilitate patients making informed decisions.[21] When discussing a possible lifestyle intervention with a patient a balanced framing of the benefits would be, "If you give up smoking you could live an extra five years and be much less likely to be disabled by a stroke like your uncle was." The benefits versus risks of starting a medicine could be presented by saying, "This medicine has a good chance of lowering your cholesterol, making you healthier, and helping you live several years longer. A small number of people may also experience side effects like the muscle aches we talked about before."

Present benefits from short to long-term

The degree to which people are motivated by short and long-term benefits varies, and for some patients short-term gains are more important than long-term benefits.[14] This may partially explain why some patients persist with behaviour that they know is doing them long-term harm. For example, the damage caused by smoking one cigarette may appear to be negligible to the smoker, however, the pleasure of smoking one cigarette may be perceived as being substantial. By framing the benefits of an intervention as both short-term and long-term, health professionals are likely to broaden the appeal of the message to patients. For example, "if you quit smoking today":

- Within two days food will smell and taste better
- Within three months your circulation will improve and that leg pain may go away
- After a year you will be able to afford to go on holiday
- Ten years from now you will be more likely to see your grandchildren

Set S.M.A.R.T targets

Cardiovascular risk reduction is dependent on patients understanding their risk and wanting to reduce it; a journey that is unique for every patient. A suggested format for interventions to be presented in is:

- **S**pecific – a specific target would be "I'm going to walk 30 minutes each day during lunch", rather than: "I'm going to exercise more"
- **M**easurable – this allows everyone to know if it has been achieved or not
- **A**chievable – unrealistic targets will cause patients to lose motivation. Modest targets, e.g. 500 g of weight loss a week, are achievable and are more likely to increase patient confidence
- **R**ecorded – patients are more likely to respond positively when they can measure their progress towards a future goal
- **T**ime bound – goals are more likely to be achieved if they are bound to an agreed time-frame, e.g. by their daughter's wedding

Writing down goals and sharing them with others is likely to make them more concrete and may mean the patient has a greater chance of achieving them.

Tailor interventions to the patient's lifestyle

Cardiovascular interventions should aim to improve aspects of life that are important to the patient and their family/whānau. Health professionals who have a longstanding and trusting relationship with patients and their family/whanau are likely to understand some of the personal motivators for engaging patients with cardiovascular interventions. Asking patients "what makes you smile?" is a good way to find out what they enjoy, and this answer can be used as a focus for interventions. For example, a patient with children may be motivated to exercise by playing with their children in the park or coaching one of their sport teams. This patient-centred model of care seeks to find common ground with clinical priorities and the individual patient's beliefs, goals and expectations (see: "Using the Te Whare Tapa Whā framework", opposite).

In situations where there is not a longstanding relationship, or where patients are unwilling to consider lifestyle change or treatment, i.e. they are at a pre-contemplative stage of change, the clinician still needs to respect the patient's decision-making autonomy, and work to maintain a trusting therapeutic relationship.

Check what the patient is taking away from the discussion

It is important not to overwhelm patients with information.[10] An "Ask, tell, ask" approach, or a "chunk and check" approach to

consultations means that information is presented to patients at a controlled rate, with pauses to confirm comprehension and agreement. A discussion about a cardiovascular risk factor can be started with a question like, "Why do you think your blood pressure might be up?" Asking a patient what they will tell their family/whānau is a simple way to check what message the patient is taking away at the end of the consultation. It also emphasises the benefit of including the patient's family in their management plan. Information that is written down and can be taken away or accessed via the internet means the patient is able to review the material on their own or with family/whanāu to improve understanding.

Before the end of the consultation, ask the patient to suggest a reasonable timeframe for the next consultation, e.g. "When would you like to catch up again?". Active management can be reserved for patients at higher risk, or who have difficulties attending consultations. More frequent consultations that are focused on specific issues may be more beneficial than longer appointments where multiple issues are addressed.

What matters to the patient – not what is the matter with the patient

Some patients may be reluctant to initiate a medicine, e.g. statins, once they have an increased understanding of the benefits versus risks of treatment. Sometimes it is necessary for health professionals to accept that most human decisions are made on an emotional basis.[2] Patients usually know that aspects of their lifestyle are unhealthy. By assessing patient readiness to change, the clinician may strengthen their relationship with the patient, so they are better placed to enable them to make healthier decisions about their lives at a later date. As one experienced diabetes educator said "I may know what is best for another person's health, but I am ignorant and arrogant if I think I know what is best for another person's life."[14]

Using the Te Whare Tapa Whā framework

Te Whare Tapa Whā is a conceptual framework developed as a way to view Māori well-being in a broad context. Over time Te Whare Tapa Whā has become a basis for developing health practice from national policy to models of service delivery. The four realms of this framework are centred on taha wairua (the spiritual side), taha hinengaro (thoughts and feelings), taha tinana (the physical side), and taha whānau (family).

Te Whare Tapa Whā encourages health professionals to consider not only the physical person and their conditions, but also the other elements of the framework when consulting with patients. It is seen as being a way to both enhance the relationship between the patient and the health professional, and to support health outcomes. By linking the benefits of an intervention to the ability of a patient to participate fully in all aspects of their life, from personal to community, the patient is encouraged not to think of medicines in isolation. An example of describing cardiovascular risk management using the four elements of Te Whare Tapa Whā is shown in Table 1.

Health professionals need to have a good understanding of a patient, including their whānau, beliefs, education and values in order to use the Te Whare Tapa Whā framework effectively. Asking patients what they see as being important in their life and talking about their priorities allows health professionals to gain a wider knowledge about patients they are consulting with.

Table 1: Engaging patients in cardiovascular risk management using the Te Whare Tapa Whā framework

Wairua (Spiritual)	**Hinengaro** (Psychological)	**Tinana** (Physical)	**Whānau** (Family)
Improved health provides a sense of well-being or happiness that is likely to be noticed by whānau	Improved cardiovascular management provides greater confidence in health and reduces anxiety	Improved cardiovascular health means participation in, and enjoyment from, a wider range of physical activities	Knowledge that family members are likely to live longer provides a sense of security for the whānau

The "how to" of cardiovascular risk assessment

The current recommendation for the age at which patients should first be offered a cardiovascular risk assessment depends on a variety of unmodifiable and modifiable risk factors (Table 2).

Calculations of cardiovascular risk using Framingham-based equations for patients outside the age range of 35 – 75* years are less accurate, but may still be useful for the purposes of shared decision-making.[5] This includes:[5]

- Patients with a HDL < 0.7 mmol/L - because there is a risk they have a genetic lipid disorder
- Patients with known familial dyslipidaemias or suspected genetic lipid disorders
- Patients with type 1 diabetes, type 2 diabetes with microalbuminuria or type 2 diabetes for longer than ten years

* In patients aged under 35 years their risk level should be calculated as if they are were aged 35 years.[5]

What should be recorded when performing a cardiovascular risk assessment?

The recommended information required to perform a five-year cardiovascular assessment is listed in Table 3.

If the patient's total cholesterol (TC) or total cholesterol:high-density cholesterol (TC:HDL-C) ratio is above 8 mmol/L then the test should be repeated, and a fasting lipid test considered if the patient has never had their lipid levels measured before.[5] It is acceptable to use blood pressure and non-fasting TC, HDL-C and HbA_{1c} that have been recorded in the previous five years for cardiovascular risk assessment, if the patient's circumstances have not significantly changed.[5] The clinical importance of a current cardiovascular risk assessment rises as the patient's cardiovascular risk increases.[5] Following an assessment the recommended risk factor monitoring period is

Table 2: The recommended age in years to begin cardiovascular risk assessments in patients without cardiovascular disease, adapted from Cardiovascular Disease Risk Assessment, 2013.[5]

Patient group	Males (years)	Females (years)
Patients without symptoms or known risk factors	45	55
Māori, Pacific or Indo-Asian patients*	35	45
Patients with known cardiovascular risk factors or at high risk of developing diabetes	35	45
Family history of: ■ Diabetes in a first-degree relative ■ Premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother < 55 years, mother or sister < 65 years)	35	45
Personal history: ■ Current smoker, or have quit smoking in the past 12 months ■ Gestational diabetes or polycystic ovary syndrome ■ Blood pressure ≥ 160/95 mmHg or TC:HDL ≥ 7 ■ HbA_{1c} 41 – 49 mmol/mol ■ BMI ≥ 30 or truncal obesity ≥ 100 cm in men or ≥ 90 cm in women ■ eGFR < 60 mL/min/1.73m^2	35	45
Patients with type 1 or type 2 diabetes	Annually from diagnosis	Annually from diagnosis

*Indo-Asian peoples include: Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani and Tibetan

determined by the individual patient's level of cardiovascular risk:[5]

- For patients with established cardiovascular disease initially at three months, then as clinically indicated
- For patients with a cardiovascular risk greater than 20%, annually, or as clinically indicated
- For patients with a cardiovascular risk from 10 – 20% as clinically indicated with a more intensive focus on patients with a higher combined risk. If the patient is not taking medicines to reduce their risk then offer reassessment at one year, for patients with a risk from 15 – 20%, and every two years for patients with a risk from 10 – 15%.
- For patients with a cardiovascular risk of less than 10% offer a further assessment in five to ten years

Cardiovascular risk is adjusted for some patient groups

The following patient groups will have 5% automatically added to their cardiovascular risk by the calculator as current Framingham-based estimates will tend to underestimate their cardiovascular risk:[5]

- Māori, Pacific or Indo-Asian peoples
- Patients with diabetes and microalbuminuria or persistent proteinuria, or diabetes for longer than ten years, or with HbA_{1c} consistently ≥ 64 mmol/L
- Family history of premature coronary heart disease or ischaemic stroke in a first-degree relative

A new cardiovascular disease risk assessment equation based on New Zealand data is anticipated to be available later in 2014.

Table 3: The recommended information to be recorded when performing a cardiovascular risk assessment, adapted from Cardiovascular Disease Risk Assessment, 2013.[5]

Category	Record
Patient characteristics	■ Age ■ Gender ■ Ethnicity ■ Smoking status
Family history	■ Type 2 diabetes ■ Premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother < 55 years, mother or sister < 65 years) ■ Genetic lipid disorder
Medical history	■ Diabetes ■ History of cardiovascular disease ■ Renal impairment ■ Atrial fibrillation ■ Genetic lipid disorder
Medical history	■ One sitting blood pressure for the purposes of risk assessment, if not above 160/95 mmHg, otherwise the average of two ■ BMI and waist circumference ■ HbA_{1c} ■ Non-fasting lipid profile

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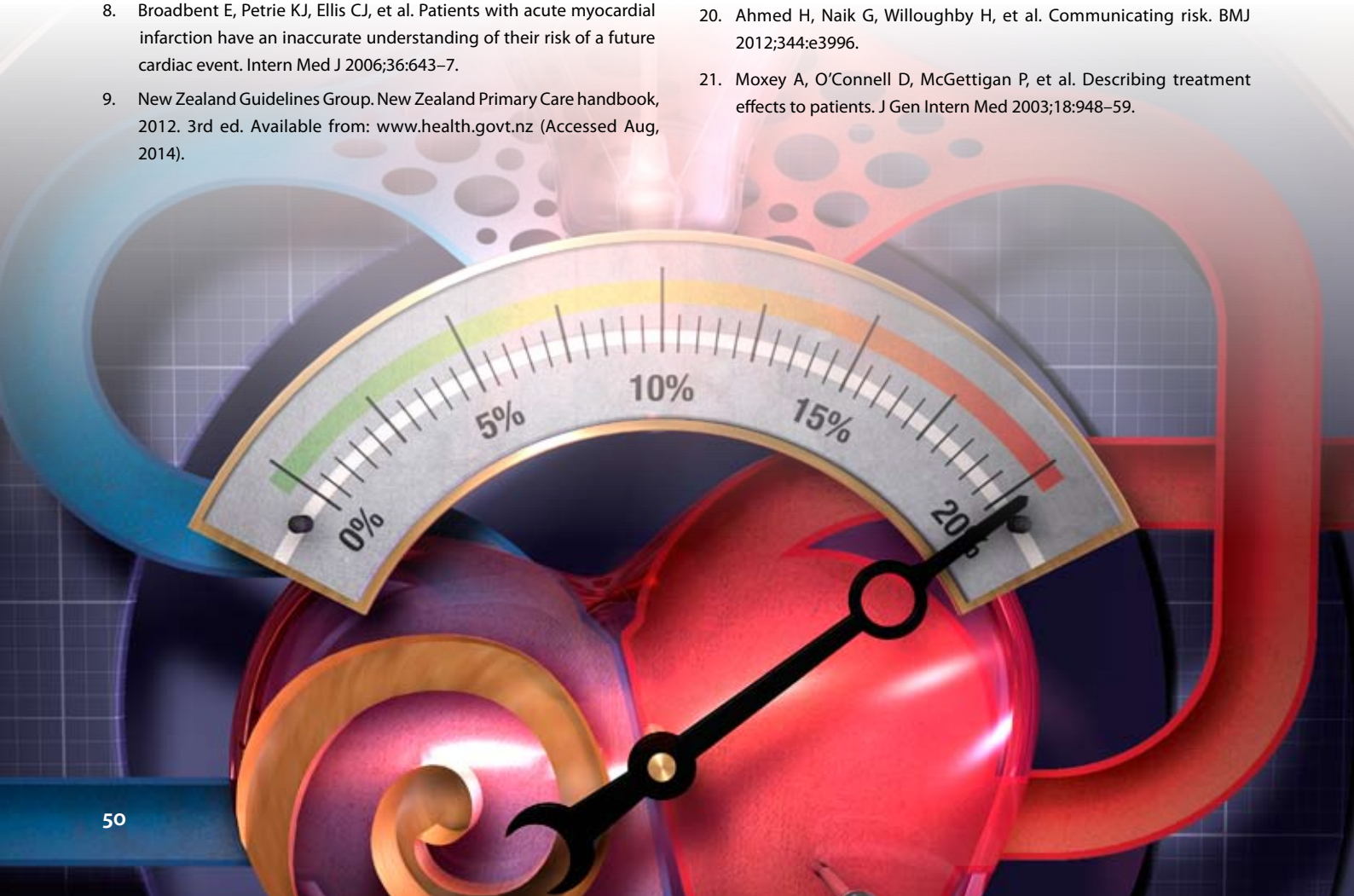
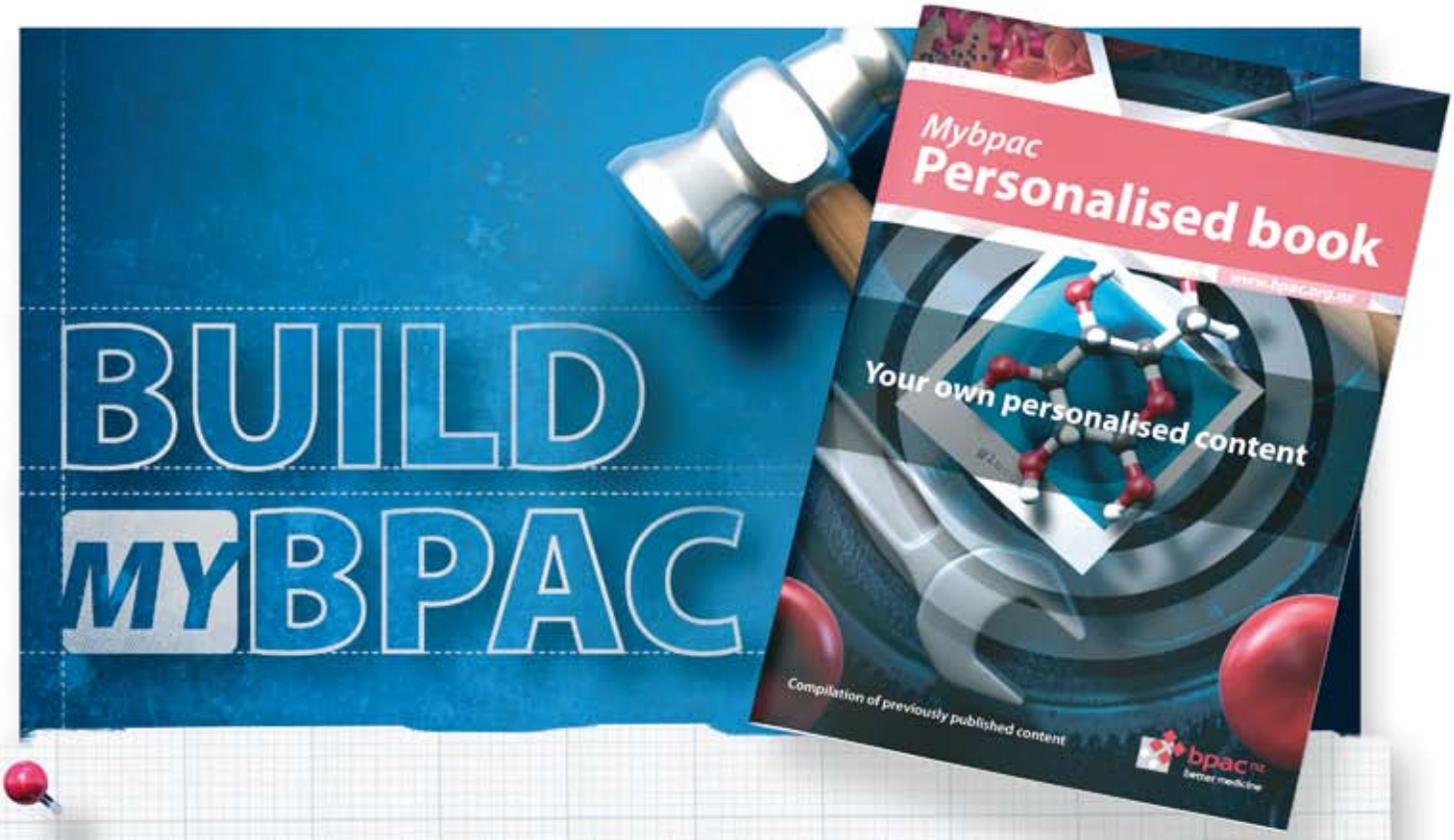
References

1. Lloyd-Jones DM. Cardiovascular risk prediction: basic concepts, current status, and future directions. Circulation 2010;121:1768–77.
2. Paling J. Strategies to help patients understand risks. BMJ 2003;327:745–8.
3. Lipkus IM, Samsa G, Rimer BK. General performance on a numeracy scale among highly educated samples. Med Decis Mak 2001;21:37–44.
4. Krones T, Keller H, Sönnichsen A, et al. Absolute cardiovascular disease risk and shared decision making in primary care: a randomized controlled trial. Ann Fam Med 2008;6:218–27.
5. Cardiovascular disease risk assessment: updated 2013 - New Zealand Primary Care Handbook. Available from: www.health.govt.nz/system/files/documents/publications/cardiovascular-disease-risk-assessment-updated-2013-dec13.pdf (Accessed Aug, 2014).
6. Spiegelhalter DJ. Understanding uncertainty. Ann Fam Med 2008;6:196–7.
7. Edwards A, Elwyn G, Covey J, et al. Presenting risk information--a review of the effects of 'framing' and other manipulations on patient outcomes. J Health Commun 2001;6:61–82.
8. Broadbent E, Petrie KJ, Ellis CJ, et al. Patients with acute myocardial infarction have an inaccurate understanding of their risk of a future cardiac event. Intern Med J 2006;36:643–7.
9. New Zealand Guidelines Group. New Zealand Primary Care handbook, 2012. 3rd ed. Available from: www.health.govt.nz (Accessed Aug, 2014).
10. Edwards A, Elwyn G, Mulley A. Explaining risks: turning numerical data into meaningful pictures. BMJ 2002;324:827–30.
11. New Zealand Heart Foundation. Know the facts. Available from: www.heartfoundation.org.nz/know-the-facts/statistics (Accessed Aug, 2014).
12. Kerr S, Penney L, Barnes HM, et al. Kaupapa Maori Action Research to improve heart disease services in Aotearoa, New Zealand. Ethn Health 2010;15:15–31.
13. Gigerenzer G, Edwards A. Simple tools for understanding risks: from innumeracy to insight. BMJ 2003;327:741–4.
14. Rodriguez KM. Intrinsic and extrinsic factors affecting patient engagement in diabetes self-management: perspectives of a certified diabetes educator. Clin Ther 2013;35:170–8.
15. Garcia-Retamero R, Okan Y, Cokely ET. Using visual aids to improve communication of risks about health: a review. Scientific World Journal 2012;2012:562637.
16. Zipkin DA, Umscheid CA, Keating NL, et al. Evidence-based risk communication: a systematic review. Ann Intern Med 2014;161:270–80.
17. Waldron C-A, van der Weijden T, Ludt S, et al. What are effective strategies to communicate cardiovascular risk information to patients? A systematic review. Patient Educ Couns 2011;82:169–81.
18. Goodyear-Smith F, Arroll B, Chan L, et al. Patients prefer pictures to numbers to express cardiovascular benefit from treatment. Ann Fam Med 2008;6:213–7.
19. Wells S, Kerr A, Broadbent E, et al. Does Your Heart Forecast help practitioner understanding and confidence with cardiovascular disease risk communication? J Prim Health Care 2011;3:4–9.
20. Ahmed H, Naik G, Willoughby H, et al. Communicating risk. BMJ 2012;344:e3996.
21. Moxey A, O'Connell D, McGettigan P, et al. Describing treatment effects to patients. J Gen Intern Med 2003;18:948–59.





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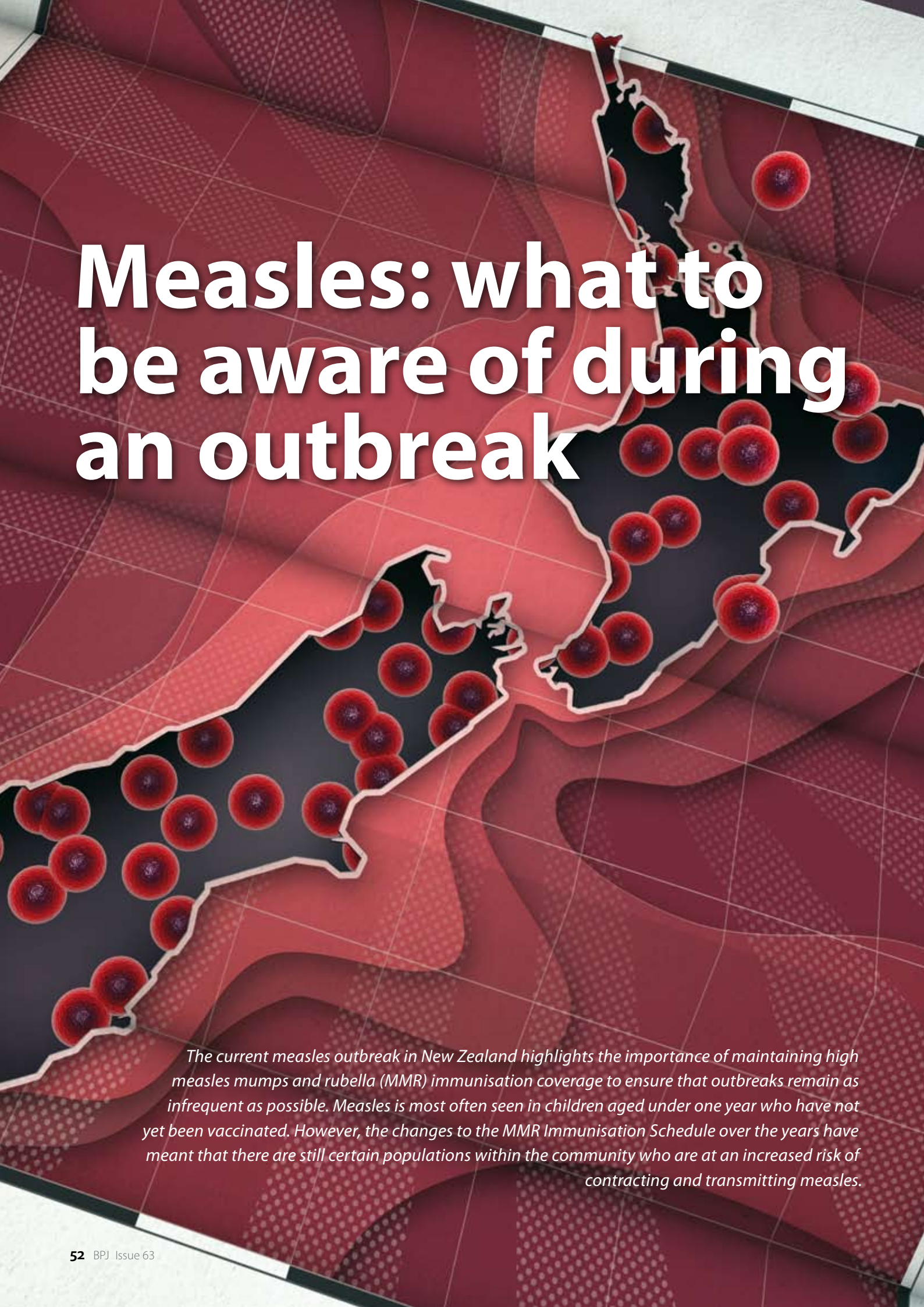
Measles: what to be aware of during an outbreak



The current measles outbreak in New Zealand highlights the importance of maintaining high measles mumps and rubella (MMR) immunisation coverage to ensure that outbreaks remain as infrequent as possible. Measles is most often seen in children aged under one year who have not yet been vaccinated. However, the changes to the MMR Immunisation Schedule over the years have meant that there are still certain populations within the community who are at an increased risk of contracting and transmitting measles.

New Zealand is currently in the midst of a measles outbreak, which began in December, 2013. As of 15 August 2014, 281 cases of measles have been reported in New Zealand, mostly linked to international travel (113 cases in Auckland and 125 in Waikato).[1]

Measles ("English measles", rubeola) is a highly contagious viral disease that is characterised by rash and fever and is associated with a number of serious complications. The best protection against measles is immunisation with the combined measles, mumps and rubella (MMR) vaccine. The MMR vaccine was added to the National Immunisation Schedule in January 1990, and is currently recommended for children at age 15 months with a second dose at age four years.[2] MMR replaced the separate measles and rubella vaccines that had previously been on the schedule since 1969 and 1970, respectively (for further details of the vaccination timeline see: "MMR vaccination", Page 54).[2]

To protect against measles, all people born on or after 1 January 1969 should receive two doses of a measles-containing vaccine.[2] Anyone who has only received a single measles vaccine prior to the introduction of the combined MMR vaccine should receive two doses of MMR to offer best protection. There is no safety concern in regards to giving an extra measles vaccine. People born prior to 1969 are considered to be immune to measles as it is presumed they would have been exposed to wild-type measles prior to the introduction of the measles vaccine.[2]

According to the World Health Organisation (WHO), 95% of the world's population born after the measles vaccination was introduced, need to be fully vaccinated against measles in order for the disease to be eliminated.[3] Between 1980 – 2012, 81% of children aged two years in New Zealand received a measles-containing vaccine.[3] In 2006 and 2007, 92 – 93% of children received the first dose of MMR, but only 89% received the second MMR dose.[3] These immunisation rates for MMR are not high enough to prevent outbreaks of measles. However, the most recent statistics for 2008 – 2011 are encouraging, with national coverage rates of 93% – 94% for the first dose of MMR in children aged two years.[3]

Who can contract measles?

Both adults and children can contract measles but the incidence rate decreases with increasing age. Children aged less than one year are still the most likely age group to be infected with measles as they have not yet received the MMR vaccine. However, there was an increase in the infection rates in adolescents aged 10 – 14 years during the 2009 and 2011 outbreaks.[3] This was likely due to changes in the MMR vaccination schedule and less than optimal vaccination rates for the birth cohort born between 1990 and 2000 (see: "MMR vaccination", Page 54). Measles infection results in lifelong immunity and people can only contract the disease once, but occasionally people who have received MMR can still contract measles (due to inadequate immune response or inadequate vaccination). Of the 68 cases of measles reported in 2012, 40 were in unvaccinated people (including 20 cases in children aged less than 15 months), 10 were in people who had received one dose of MMR and seven were in people who had received two doses of MMR. Vaccination status was unknown in the remaining 11 people.[2]

People most at risk of contracting measles include those who have not been vaccinated with MMR, those who are returning from international travel (due to higher risk of exposure in measles-endemic countries) and those born overseas in countries where appropriate vaccination is less likely.

What is measles?

Measles is caused by a paramyxovirus and transmission is by direct person-to-person contact via oropharyngeal and nasopharyngeal droplets, and less commonly via airborne spread or contact with infected surfaces, e.g. door handles and eating utensils, as the virus can survive on these surfaces for several hours. Measles is one of the most highly communicable of all infectious diseases. There is an incubation period of approximately 10 – 14 days after exposure before symptoms appear.[1]

The signs and symptoms are highly characteristic in most people and tend to appear in three stages:[7]

Prodromal stage, lasting three to four days, where symptoms can include fever of greater than 38°C, malaise, anorexia, diarrhoea, Koplik spots (tiny white spots like grains of salt on the buccal mucosa), the "3Cs" of cough, coryza (rhinitis) and conjunctivitis.

Exanthema (rash) stage, lasting four to five days, and characterised by a blotchy, bright red maculopapular rash that is generally not itchy. This rash typically starts behind the ears and then spreads to the face and neck and then the rest of the body.

MMR vaccination

MMR vaccination is fully subsidised on the National Immunisation Schedule. It is recommended that children are vaccinated at age 15 months with a second dose at age four years.[2] Anyone born in or after 1969 who has not received two documented doses of MMR needs two doses at least one month apart (Table 1).[2]

Because it is a live (attenuated) vaccine, MMR vaccination should not be given to pregnant women and pregnancy should be avoided for one month after vaccination.[4] The MMR vaccine is not contraindicated in women who are breastfeeding.[4]

Table 1: Recommended MMR vaccinations for protecting against measles in New Zealand[2]

Patient group	Recommended vaccination
Children born in New Zealand	Two doses of MMR; at age 15 months and at age four years
People born on or after 1 January, 1969 with no documented history of MMR vaccine (including those who have received a single measles-only vaccination)	Two doses of MMR; four weeks apart
People born before 1 January, 1969	MMR is not required

Vaccination with MMR is very effective. After one dose of the MMR vaccine, 90 – 95%, 95 – 96% and 90 – 97% of recipients aged greater than 12 months are protected from measles, mumps and rubella, respectively.[5] After the second dose, almost all recipients are immune to all three diseases.[5] The estimated duration of protection after administration of two doses is lifelong in more than 96% of recipients.[5] However, occasionally some people still contract measles despite having received two doses of MMR. This may be due to problems with the vaccine (inappropriate administration or storage), waning of immunity over time or maternal antibodies from breast-feeding blocking the vaccine, such as when doses are given in infants. If an immunised person does contract measles it is likely to be less severe.[6]

History of measles, rubella and MMR vaccination in New Zealand

The history of measles and rubella vaccination in New Zealand can provide useful information for clinicians as to which people are more likely to not be fully protected against measles and rubella.[2]

1969 Measles vaccine (one dose) introduced for children aged 10 months to five years (who will now be aged 40 – 45 years)

1970 Rubella vaccination (one dose) introduced for all children at age four years

1974 Age change for measles vaccination to age 12 months

1979 Due to low uptake of rubella vaccination at age four years (especially in boys) schedule changed to rubella vaccination in girls at age 11 years (the "schoolgirl" rubella vaccination programme)

1981 Age change for measles vaccination to age 12 – 15 months

1990 MMR vaccine is introduced for all infants at age 12 – 15 months, replacing the separate measles and rubella vaccines

1992 A second dose of MMR is added to the schedule at age 11 years

2001 The timing of the second dose of MMR is changed from age 11 years to age four years and a school-based, catch-up vaccination programme is offered to all children aged 5 – 10 years

For further information see MMR – frequently asked questions, available from: **www.immune.org.nz**

Convalescent stage where the rash fades leaving a temporary brownish stain on the skin.

Differential diagnoses for measles include other causes of rash, fever and conjunctivitis, e.g. drug sensitivities, roseola, enterovirus, adenovirus and infectious mononucleosis (EBV) infections, scarlet fever, Kawasaki disease and rubella.[8]

Laboratory testing and notification

Confirming clinically suspected cases of measles with laboratory testing is recommended. The virus is more likely to be present within the first week of onset of rash, so ideally sampling should occur within this time period.[9] Measles is a notifiable disease and the Medical Officer of Health should be notified as soon as measles is suspected and prior to laboratory confirmation.

A nasopharyngeal swab in a vial of universal transport medium is the preferred method of testing.[9] However, if unavailable, throat swabs may be used in young children. A blood sample (for serology) can also be used as long as the rash has been present for at least three days.[9] Local protocols on testing vary, i.e. whether testing is done locally or sent to another laboratory – clinicians should check with their local laboratory to see where testing is performed. Once measles has been isolated, samples are sent to the New Zealand National Measles Laboratory (at Canterbury Health Laboratories).[9]

N.B. Swabs are best taken in the general practice (or initial site of contact with the patient), so other patients at laboratory collection centres are not exposed to the virus.

For further information, see: **www.measles.co.nz/specimen-guidelines**

Prevention of transmission

People with measles are infectious for five days before and after the rash appears.[1] This can make prevention and control measures difficult to implement as people are infectious well before symptoms become apparent. However, some measures can be implemented in health clinics during outbreaks and when dealing with a patient with suspected measles. This can include having suitable triage and isolation areas for patients, allowing only immune staff to have contact with the patient, and having hand gels and surgical masks at reception areas and at practice entrances/exits. Children should be kept away from day care or school for at least five days after the rash appears.[7] Transmission between family/household members is common and people with suspected or confirmed measles should avoid contact with non-immune infants and pregnant women.

In the event of a measles outbreak, The Immunisation Advisory Centre advises that non-immunised children (who have not had measles and whose parents do not want them to receive MMR) who have contact with people with measles should not attend school or early childhood services (or be in public places) until notified. This may be for a period of 14 days (incubation time).

MMR and immunoglobulins as prophylaxis for measles

There are only a few management options available that may help prevent infection after people have potentially been exposed to measles. These include either active (MMR) or passive (human immunoglobulin) immunisation.

MMR: During outbreaks, or other circumstances when protection against measles is urgently required, MMR can be given to unvaccinated people, who are not immunocompromised, within 72 hours of exposure and this may prevent infection.[5] If measles infections are being reported in very young infants, MMR can given to children aged six to 12 months, i.e. infants are fast tracked for vaccination before the recommended age of 15 months.[2] However, as the immune response may not be as effective, the child will still require a further two doses of MMR at age 15 months and four years.[2]

Human normal immunoglobulin (HNIG): This is a preparation of concentrated antibodies (immunoglobulins) that are not specific to a particular infection, but can boost immunity in people who are immunocompromised. An Infectious Diseases Specialist or Paediatrician may recommend intramuscular (IM) administration of HNIG in the following scenarios:[4]

- In people with contraindications to MMR
- In immunocompromised children and adults
- In pregnant women
- In children aged less than 15 months who present more than 72 hours after exposure to measles
- In people who present more than 72 hours after exposure and have no history of measles OR have not received MMR

HNIG prophylaxis should be given as soon as possible after exposure to measles, but can be administered for up to six days. It should not be administered within three weeks of a live virus vaccine and live virus vaccines should not be given for 11 months after a patient has received HNIG.[2]

For further information on HNIG see: **www.nzblood.co.nz/assets/Transfusion-Medicine/PDFs/POST-EXPOSURE-PROPHYLAXIS-FOR-MEASLES-111G001.pdf**

Intravenous (IV) Immunoglobulin (Intragram P): is a similar preparation as HNIG but is administered via the IV and not the IM route. Intragram P can be considered in immunocompromised people who have had contact with a person with confirmed measles, people who require large doses of immunoglobulin prophylaxis, and people with a reduced muscle bulk for whom IM administration would be difficult.[2]

Treatment of measles

There are no specific antiviral treatments for measles and patients usually improve within seven to ten days without treatment. However, patients/parents can be advised about some measures to help control the symptoms. These can include:

- Using analgesia, e.g. paracetamol or ibuprofen, to reduce pain and discomfort
- Ensuring adequate fluid intake to avoid dehydration, especially if febrile
- Treating sore eyes. This can include wiping the crustiness from the eyelids and lashes using cotton wool and water (use a separate piece of cotton wool for each eye) and avoiding bright light

Complications of measles and follow up

Complications from measles are common and up to 30% of people experience at least one complication.[5] The most common complications (with approximate rates) are otitis media (7 – 9%), pneumonia (6%) and diarrhoea (8%).[5] The risks of complications are highest in children aged less than five years, in adults aged over 20 years and in people who have chronic co-morbidities (especially those who are immunocompromised).[5]

Approximately one in 1000 people who contract measles will die; pneumonia accounts for approximately two-thirds of these deaths.[5] Death rates are much higher than this in less developed countries. Some of the more serious complications of measles include acute encephalitis (approximately 0.1% of people) and subacute sclerosing panencephalitis, a rare degenerative central nervous system disease, which is always fatal, and occurs in approximately one in 100 000 people.[5] Contracting measles during pregnancy can increase the risk of premature labour and miscarriage.[5]

Patients should be informed of the possible complications of measles and advised to contact a health professional at any time when they are concerned about their symptoms, if their condition worsens, or if their symptoms are not resolving.

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References

1. Ministry of Health (MOH). Measles information for health professionals. MOH, 2014. Available from: www.health.govt.nz/our-work/diseases-and-conditions/measles-information-health-professionals (Accessed Aug, 2014).
2. Ministry of Health (MOH). Immunisation Handbook 2014. MOH, 2014. Available from: www.health.govt.nz/publication/immunisation-handbook-2014 (Accessed Aug, 2014).
3. Ministry of Health (MOH). Measles immunisation coverage. MOH, 2014. Available from: www.health.govt.nz/our-work/diseases-and-conditions/measles-information-health-professionals/measles-immunisation-coverage (Accessed Aug, 2014).
4. New Zealand Formulary (NZF). NZF v25. 2014. Available from: www.nzf.org.nz (Accessed Aug, 2014).
5. Immunisation Advisory Centre. Measles. Available from: www.immune.org.nz (Accessed Sep, 2014).
6. Mitchell P, Turner N, Jennings L, et al. Previous vaccination modifies both the clinical disease and immunological features in children with measles. J Prim Health Care 2013;5:93–8.
7. Murtagh J, Rosenblatt J. Common childhood infectious diseases (including skin eruptions). In: Murtagh's General Practice. NSW, Australia: McGraw-Hill Australia Pty Ltd, 2011. pp. 899–912.
8. Longo DL, Fauci AS, Kasper DL, et al. Chapter 192: Measles (Rubeola). In: Harrison's principles of internal medicine. New York, USA: McGraw Hill Medical, 2012. pp. 1600–4.
9. Measles Specimen Collection and Transportation, Measles Protocol. Available from: www.measles.co.nz/specimen-guidelines (Accessed Aug, 2014).
10. Ministry of Health (MOH). Rubella. MOH, 2014. Available from: www.health.govt.nz/your-health/conditions-and-treatments/diseases-and-illnesses/rubella (Accessed Sep, 2014).

Rubella infections are relatively rare in New Zealand

Rubella ("German measles") is caused by a togavirus and is therefore a different disease to measles (rubeola). Transmission is via contact with infected nasopharyngeal and oropharyngeal secretions with an incubation period of approximately 16 – 18 days.[10] The infectious period is approximately seven days before the rash appears until at least four days after, but rubella is not as infectious as measles.[11] Rubella infections are rarely reported in New Zealand – only five cases of rubella were reported in 2011 – 12 and no cases of congenital rubella syndrome have been reported since 1998.[2] However, the exact prevalence of rubella is hard to establish as the illness can be mild and non-specific in childhood and many cases are not reported.

The clinical presentation of rubella is characterised by a discrete, pale-pink maculopapular rash starting on the face and neck and spreading to the rest of the body. The rash is not as confluent as that observed in measles (more discrete and does not tend to merge together) and usually fades on the third day. Other symptoms can include lymphadenopathy, fever (in children) and arthralgia (in adolescents and adults).

Approximately one-third of people with rubella are asymptomatic (subclinical infection) and all infections are considered relatively benign.[7] Rubella infections are only considered significant if there is a possibility that a pregnant woman has been in contact with a person with rubella due to the risk of congenital rubella syndrome (see below).[2] Rubella is a notifiable disease and the Medical Officer of Health should be notified as soon as rubella is suspected, followed by laboratory testing to confirm (serology).

Rubella in pregnancy

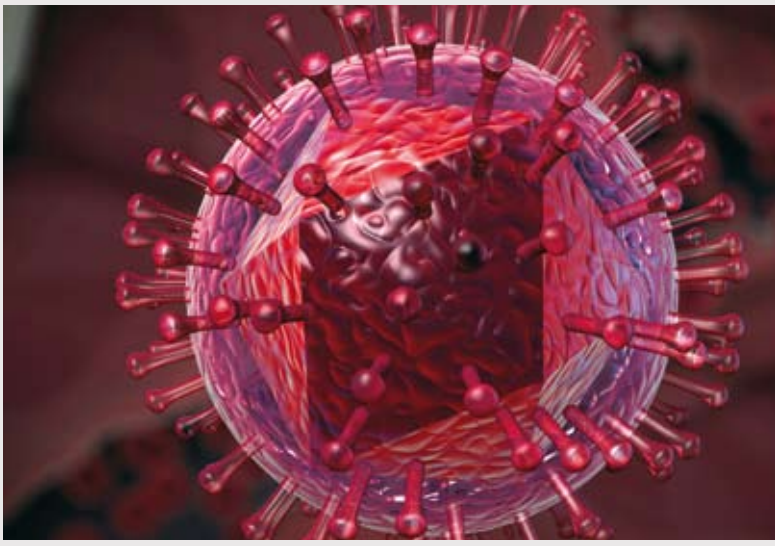
Ideally all women of child-bearing age should be aware of their rubella immune status. It is recommended that women are tested for rubella immunoglobulin G (IgG) antibodies (not subsidised) when pregnancy is planned.[2] Rubella IgG testing is part of routine antenatal care (subsidised) once a women is pregnant, and should be requested by the Lead Maternity Carer or General Practitioner.[2] Certain groups of women are more likely to be seronegative for rubella, including women aged over 35 years and women born overseas (especially in the Pacific Islands, Asia, sub-Saharan Africa and South America) and entering New Zealand after the age when routine childhood vaccinations are administered.[2] Women who have no evidence of rubella immunity can be given two doses of MMR, four weeks apart (but not during pregnancy).

The most serious complication of rubella is congenital rubella syndrome as a result of rubella infection during pregnancy (especially during the first trimester). The syndrome is characterised by miscarriage or stillbirth and foetal malformations, e.g. deafness and blindness, growth abnormalities and congenital heart disease.

Although MMR is contraindicated during pregnancy, the vaccine is not contraindicated in women who are breastfeeding. MMR may be given to protect against rubella in previously unimmunised and seronegative (no antibodies against rubella) post-partum women.[4] Human normal immunoglobulin is not recommended as prophylaxis for rubella in pregnant women exposed to the infection as it has not been shown to be effective.[5]

Pregnant women with low rubella IgG levels (<10 IU/mL) should be advised to avoid situations where contact is more likely (especially in the first trimester), e.g. international travel to countries with endemic disease or known outbreaks. MMR can be given after delivery of the infant (subsidised).[2]

For further information see: Routine laboratory testing during pregnancy" Best Tests (July, 2011).



Safer prescribing of high-risk medicines

Colchicine – extremely toxic in overdose

Colchicine is a plant-based alkaloid, extracted from *Colchicum autumnale* (autumn crocus, meadow saffron) and *Gloriosa superba* (glory lily) used to treat gout and some other inflammatory conditions.[1] It is considered a high-risk medicine because it is associated with significant toxicity when not used correctly.

Colchicine has long been used to treat acute flares of gout, due to its anti-inflammatory properties. Although not an approved indication, colchicine is also used for prophylaxis of gout flares, particularly during the first few months of urate-lowering treatment (usually allopurinol). Colchicine inhibits neutrophil migration, chemotaxis, adhesion and phagocytosis in the area of inflammation. It reduces the inflammatory reaction to urate crystals, but has no effect on uric acid production or excretion.[2]

Non-steroidal anti-inflammatory drugs (NSAIDs), e.g. naproxen, and low-dose corticosteroids are also used for acute management of gout flares and prophylaxis of flares during the initiation of urate-lowering treatment. For many patients, NSAIDs are associated with less adverse effects and risk of toxicity than colchicine,[3] and may be the preferred treatment. However, colchicine is still an important treatment option as it is particularly useful for patients with co-morbidities, such as diabetes, renal impairment and peptic ulcer disease, in whom NSAIDs and prednisone may cause significant adverse effects.[4]

Colchicine can cause significant toxicity and death

Colchicine has a narrow therapeutic index, which means that the range between therapeutic and toxic doses is small, and in some cases they overlap.[1] Acute overdose exceeding 0.5 mg/kg is usually fatal.[1] Fatalities have been associated with doses as low as 7 mg. In contrast, patients have survived doses up to 60 mg.[5] In a case series of nine patients presenting with colchicine overdose in the Auckland region over a 15 year period, eight died.[6] Four of the patients had taken an accidental overdose of colchicine (ranging from 18 – 24 mg) due to lack of knowledge about the medicine.[6] Colchicine is particularly toxic to children and even one or two tablets can cause serious toxicity.[5]

Gastrointestinal disturbance is usually the first sign of toxicity

Abdominal pain, diarrhoea, nausea and vomiting are usually the first symptoms of colchicine toxicity. A burning sensation in the throat, abdomen or on the skin has also been reported. These symptoms, particularly diarrhoea, can also occur with doses within the therapeutic range. Later features of toxicity (24 hours to seven days after ingestion) include tachypnoea, electrolyte disorders (e.g. hypocalcaemia, hypophosphataemia), hypovolaemia, haematological effects (e.g. leukopaenia, thrombocytopaenia), cardiac dysrhythmias, renal failure and liver damage.[1,5,6] The cause of death is usually progressive multiple organ failure and sepsis.[1]

Adverse effects can occur even at "safe" doses

Prior to 2005, colchicine dose instructions included the advice to continue dosing until the pain settled or gastrointestinal adverse effects occured. The standard dose instructions have now been changed to improve safety. Patients are advised to stop taking colchicine immediately if they experience abdominal pain, diarrhoea, nausea or vomiting, or a burning feeling in their throat, stomach or on their skin.[5]

Table 1 shows the current New Zealand dosing recommendations for colchicine used in patients with gout.[7,8] Internationally, specifically in Australia and the United States, even lower doses are recommended. A study comparing low-dose colchicine (1.2 mg followed by 0.6 mg in 1 hour; 1.8 mg total) with high-dose colchicine (1.2 mg followed by 0.6 mg every hour for 6 hours; 4.8 mg total) found that efficacy of the low-dose regimen was comparable to the high-dose regimen, however, there was a significant reduction in the rate of adverse effects with the low dose regimen.[9] The lower dosing regimen is now recommended in colchicine guidelines in Australia and the United States.

Interactions increase the risk of colchicine toxicity

The risk of colchicine toxicity is increased when inhibitors of cytochrome P450 3A4 (CYP3A4) or P-glycoprotein (P-gp) are taken concurrently, e.g. some azole antifungals (e.g. fluconazole), calcium channel blockers (e.g. diltiazem, verapamil) and macrolide antibiotics (e.g. erythromycin) (see New Zealand Formulary for full list).[8]

If these medicines are required at the same time as colchicine, the dose of colchicine should be reduced and the patient monitored for symptoms and signs of colchicine toxicity. These combinations are contraindicated in patients with renal or hepatic impairment, as this increases the risk of toxicity.[10]

Managing colchicine toxicity

All patients with known or suspected overdose of colchicine, or displaying symptoms of colchicine toxicity, should be immediately referred to hospital. There is no specific antidote for colchicine when taken in overdose and treatment options are limited. Haemodialysis and haemoperfusion are not effective because colchicine has a large volume of distribution, binds significantly to plasma proteins and has rapid distribution.[6] If a patient presents soon after ingestion, repeated doses of activated charcoal can be given to remove colchicine from the gastrointestinal tract. Although colchicine is rapidly absorbed from the gastrointestinal tract, removal of even a small amount can improve the patient's prognosis.[6] Patients who do not present soon after ingestion, and those with pre-existing renal or hepatic impairment, have a less favourable prognosis.[6] Patients with colchicine toxicity are managed with supportive care.

Avoiding adverse effects

Manage gout more effectively

Patients who frequently use colchicine for acute gout flares should be encouraged to take long-term urate-lowering treatment, e.g. allopurinol. Preventative treatment will reduce the frequency of flares, and therefore reduce the need for acute treatment with colchicine, and the risk of toxicity.[11] Urate-lowering treatment is indicated for patients with gout who: experience recurrent flares, e.g. two or more in one year, have tophi, concomitant renal impairment or changes characteristic

Table 1: Recommended colchicine dosing regimen[7,8]

Indication	Dose
Treatment of acute gout	1 mg, followed by 500 micrograms every six hours until relief of pain, up to 2.5 mg (five tablets of 500 micrograms) on the first day; maximum 1.5 mg (three tablets) on subsequent days; total maximum 6 mg (12 tablets) over four days; course not to be repeated within three days NB: In elderly patients, patients with renal or hepatic impairment, or patients weighing < 50 kg, if it is necessary to use colchicine the initial dose should not exceed 1 mg (two tablets of 500 micrograms) in the first 24 hours; total maximum 3 mg (six tablets) over four days; course not to be repeated within three days
Prophylaxis during initiation of urate-lowering treatment	500 micrograms, once or twice daily, during the first three to six months treatment with a urate-lowering medicine, such as allopurinol

of gout on x-ray. Ideally, urate-lowering treatment should be initiated early before there has been any erosive damage to joints and before tophi have appeared.

For further information, see: "An update on the management of gout", BPJ 51 (Mar, 2013).

Provide patients with clear instructions

Patients are at risk of overdose if they have a poor understanding of how to take colchicine and its potential adverse effects. Appropriate patient education includes:

- Providing clear advice about how to take colchicine, especially the maximum dose
- Advising patients to stop taking colchicine and see their doctor if they develop nausea, vomiting or diarrhoea; unusual bleeding or bruising; muscle pain or weakness; or numbness or tingling in their fingers or toes
- Ensuring patients are aware that colchicine is not an analgesic for general use and should not be used to manage pain not due to gout
- Advise patients to tell a doctor or pharmacist about all the medicines they take and to check before taking new medicines

Advice should be tailored to the patient's level of health literacy. This is particularly important for patients for whom English is not their first language. Of the four accidental overdose cases reported in Auckland, three of those patients were of Pacific Island descent. It is possible that language barriers, cultural differences and health literacy may have been contributing factors to these accidental overdoses.[6]

A patient information handout on colchicine is available from: **www.saferx.co.nz/colchicine-patient-guide.pdf**

What can General Practitioners do?

- Provide patients with clear instructions on how to take colchicine, both verbal and written, and check for understanding. Advise patients about the dangers of overdose, overuse and the importance of safe storage.
- Limit prescriptions to 12 tablets for acute attacks of gout (6 tablets for older people)
- Prescribe monthly for prophylactic use and ensure colchicine is stopped after three to six months
- Be aware of significant medicine interactions with colchicine

ACKNOWLEDGEMENT: Thank you to **Associate Professor Matt Doogue**, Clinical Pharmacologist and Endocrinologist, University of Otago – Christchurch and Canterbury District Health Board for expert review of this article.

References:

1. Finkelstein Y, Aks SE, Hutson JR, et al. Colchicine poisoning: the dark side of an ancient drug. Clin Toxicol 2010;48:407–14.
2. Australian Medicines Handbook. Adelaide: Australian Medicines Handbook Pty Ltd, 2011.
3. Nuki G. Colchicine: its mechanism of action and efficacy in crystal-induced inflammation. Curr Rheumatol Rep 2008;10:218–27.
4. Dalbeth N, Gow P, New Zealand Rheumatology Association. Colchicine prescribing in patients with gout. N Z Med J 2011;124:107–8.
5. Waitemata District Health Board. Colchicine - safe prescribing - toe the line!! Saferx. 2011. Available from: www.saferx.co.nz/full/colchicine.pdf (Accessed Aug, 2014).
6. Jayaprakash V, Ansell G, Galler D. Colchicine overdose: the devil is in the detail. N Z Med J 2007;120:U2402.
7. New Zealand Rheumatology Association (NZRA). NZRA consensus statement on the use of colchicine in the treatment of gout. NZRA, 2005.Available from: www.rheumatology.org.nz/position_statement.cfm (Accessed Aug, 2014).
8. New Zealand Formulary (NZF). NZF v25. 2014. Available from: www.nzf.org.nz (Accessed Aug, 2014).
9. Terkeltaub R, Furst D, Bennett K, et al. High versus low dosing of oral colchicine for early acute gout flare: Twenty-four-hour outcome of the first multicenter, randomized, double-blind, placebo-controlled, parallel-group, dose-comparison colchicine study. Arthritis Rheum 2010;62:1060–8.
10. Medsafe. Colchicine: Beware of toxicity and interactions. Prescr Update 2011;32:2.
11. Dalbeth N, Gow P. Prevention of colchicine toxicity in patients with gout. N Z Med J 2007;120:U2503.

Does aspirin protect against cancer? More high-quality research is needed

There is a growing body of evidence and opinion that aspirin can play a role in the prevention of some cancers, particularly colorectal cancer.[1] This mechanism is thought to be related to the anti-platelet effects of aspirin due to the inhibition of the cyclo-oxygenase (COX) -1 and -2 pathways.[1]

A recent 2014 review that investigated the protective effect of aspirin for the primary prevention of cancer and cardiovascular events has been widely reported in the media. The review found that long-term aspirin prophylaxis in people aged 50 – 65 years had a net health overall benefit due to reductions in the rates of cancer.[2] However, the reliability of these results was questionable due to major inconsistencies in the research methodology.

At this stage, there is not enough evidence to recommend any change in practice.

What did the review report?

The authors of the review calculated that people aged 50 – 65 years who took aspirin for the primary prevention of cancer and cardiovascular events for at least five years would have lower rates of some cancers and some cardiovascular events (Table 1). The optimum dose for preventing cancer was unclear – indirect comparisons showed little difference between low (75 – 100 mg/day) and standard dose (300 – 325 mg/day) aspirin.[2]

The authors balanced the benefits of aspirin treatment against the increased risk of adverse events. It was calculated that depending on age and sex, major bleeding events would increase by between 0.16% and 0.81% over a 15 year period.[2] The net absolute reduction in mortality due to taking aspirin was almost entirely due to a reduction in deaths from cancer and varied depending on the sex and age of the patient; due to differences in baseline incidence rates of various cancers.[2] The authors estimated that the benefits of treatment with aspirin for 20 years would range from a 0.47% decrease in deaths (from all causes) for women starting treatment aged 50 years, to a 2.18% reduction in deaths for men starting treatment at age 65 years.[2] This equated to a number needed to treat (NNT) to save one life of 46 to 213.[2]

It was reported that the effects of aspirin on the risk of cancer were not apparent until after at least three years of treatment, but that some of these benefits were sustained for several years after cessation of treatment in long-term users.[2]

What were the problems with the research?

There were a number of issues regarding the compilation and interpretation of the evidence and whether the methods used were systematic. Some of the inconsistencies with the review included:

- It was not clear whether it was a systematic review, and therefore whether the included evidence was rigorously assessed for quality and risk of bias
- A meta-analysis was not performed. The authors performed their own estimates of risk
- The cardiovascular risk data was obtained from one meta-analysis[3]
- The methods stated that the evidence for cancer risk were obtained from the most recent systematic reviews (published between 2009 – 12) and some individual studies on specific cancers. However, not all recent systematic reviews were included in the review and there was no information provided as to how the included studies were selected
- Most of the evidence that was included in the review was obtained from observational studies with few randomised trials included
- An unpublished analysis was used to estimate the rates of bleeding and peptic ulcer
- Several assumptions were made regarding the effects of aspirin on the rates of cancer and cardiovascular events
- Several of the authors are consultants to, or have affiliations with, pharmaceutical companies who are conducting research into anti-platelet medicines

The authors arrived at a more positive conclusion regarding the beneficial effects of aspirin for cancer prevention than a thorough and well-reported systematic review published in 2013.[4] This 2013 review was not discussed in the current review.

The authors of the 2013 systematic review concluded that the uncertainty around their cancer estimates remained high.[4] The long-term all-cause mortality data did not provide compelling evidence for the use of aspirin for protection against cancer and cardiovascular mortality.[4] It was reported that the absolute benefits and harms of aspirin for the primary prevention of cancer and cardiovascular disease were low, with only 34 – 36 colorectal cancer deaths and 60 – 84 major cardiovascular events averted per 10 000 people over ten years.[4]

Conclusions and implications for clinical practice

Although the 2014 review reported promising results for aspirin as prophylaxis for certain types of cancer it is unclear how reliable the results are. More high-quality research is needed, including long-term randomised trials, before any definite conclusions can be drawn regarding the benefits and harms of aspirin for the primary prevention of cancer. It is important that clinicians weigh up the benefits and adverse events associated with aspirin before prescribing it, especially in older patients, as gastrointestinal bleeding and peptic ulcer are common in this group.

References

1. Thun MJ, Jacobs EJ, Patrono C. The role of aspirin in cancer prevention. Nat Rev Clin Oncol 2012;9:259–67.
2. Cuzick J, Thorat MA, Bosetti C, et al. Estimates of benefits and harms of prophylactic use of aspirin in the general population. Ann Oncol 2014;[Epub ahead of print].
3. Antithrombotic Trialists' (ATT) Collaboration, Baigent C, Blackwell L, et al. Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data from randomised trials. Lancet 2009;373:1849–60.
4. Sutcliffe P, Connock M, Gurung T, et al. Aspirin for prophylactic use in the primary prevention of cardiovascular disease and cancer: a systematic review and overview of reviews. Health Technol Assess Winch Engl 2013;17:1–253.

CORRESPONDENCE

We have received many letters in regards to our recent and ongoing series on the use of oxycodone in New Zealand. We have dedicated the correspondence section in this edition to these letters. To respond to any of these letters and express your views, you can add to the discussion online at: **www.bpac.org.nz/BPJ/2014/September/correspondence.aspx**

What are the real reasons behind the use of oxycodone?

Dear Editor,
Re: Oxycodone – How did we get here and how do we fix it?

I read with interest the prescribing rates for oxycodone in your latest update sent to all GPs. I would like to make a few points.

The first is that the article is unlikely to be convincing to those doctors who prescribe oxycodone, and is more likely to be a pleasant message to those who do not. If this is true the article is pointless as nothing will change!

Thinking on that a little deeper the question is "why do we (GPs and hospital doctors) prescribe oxycodone?

I think the answer is the widely held belief that this is a medication with some reduction in side effects. I think perhaps that needs to be directly and thoroughly addressed, quoting serious research, if there is to be a sea change in prescribing habits. Having said that there are countless times that GPs have claimed certain drugs are better or worse despite so called evidence and in the end we have often been found to be correct. So the research has to be very, very good, i.e. double blind crossover, etc.

The suspicion is that the main reason for publishing articles on various opioids is that funding for bpac and other organisations like yours, e.g. Pegasus, comes with strings attached asking you to help save money – perhaps this should be stated? To me the money is irrelevant as my clients would be incensed to think it is relevant.

The third point is if you add up all opioid scripts apart from codeine, then the overall trend for the total is going up strongly. Is this true? The drivers for this could be widespread abuse of non-cancer pain but it also reflects a growing trend for total failure of the health system to keep up with demands for orthopaedic procedures. This should be highlighted.

***Dr Hammond Williamson**, General Practitioner*
Christchurch

We thank our correspondent for his candid comments which add to the debate on the challenges of prescribing opioids for use in the community.

In the article "Oxycodone – How did we get here and how do we fix it?" (BPJ 62, Jul, 2014), we discussed the available evidence on the efficacy and adverse effects of oxycodone. The problem is that there are very few high quality, head-to-head trials comparing oxycodone with other strong opioids. Perhaps it is better to view the evidence in terms of what is not proven, which would lead to the conclusion that we cannot say for certain that oxycodone is superior to morphine in terms of adverse effects. There is some evidence that oxycodone may be less associated with nausea and vomiting than morphine, but there is also some evidence that it is more associated with constipation. If in balance, these two medicines are considered similar in efficacy and adverse effect profile, it then comes down to a decision based on other risks and benefits. The emerging misuse and addiction problems with oxycodone in New Zealand, coupled with the lessons learnt from other countries that have been dealing with these problems, swings the balance in favour of morphine, if a strong opioid is required at all. This is actually the bigger message in the "oxycodone story" – with the exception for use in malignant pain, why are we prescribing strong opioids in the community at all?

Although the prompt to explore and write about medicines is often directed at those medicines with significant cost to the health system, in order to reap the health benefit of this expenditure the medicines need to be used in accordance with best available evidence and practical application of this evidence, which is the thrust of many of our articles. This is true of medicines that are relatively inexpensive and used frequently in medicine (often general practice), as it is true of more expensive treatments that are used in a very few patients. Cost is relevant to many patients, who make cost-benefit decisions daily in all aspects of their lives. Patients are sometimes flabbergasted when they find out the actual cost of their medicine, as opposed to the $5 prescription fee they pay at the pharmacy.

In the case of oxycodone, we have collaborated with both PHARMAC and Medsafe to highlight the general safety issues with this medicine, and cost has not been a significant factor in these discussions.

As for the final point - the amount of strong opioids being used in New Zealand is increasing, as evidenced by national pharmaceutical dispensing data. It is difficult to say with certainty what is driving this increased use, but it is likely to be a combination of many factors, including widespread use of strong opioids for both malignant and non-malignant pain, misuse and misappropriation of prescriptions and pressures on the health care system to meet demands for definitive treatment of painful conditions.

We hope that the article in this edition, "Helping patients cope with chronic non-malignant pain: it's not about the opioids" may at least give clinicians some tools to help stem the tide of opioids.

Were problems with morphine formulations a factor in the uptake of oxycodone?

Dear Editor,
Re: Oxycodone – How did we get here and how do we fix it?
I have read the above article in your July edition of Best Practice. I have taken on board a number of the relevant points that you have made.

I would like to remind you and your readers that the introduction of Oxycontin to the New Zealand market was prompted by a group of concerned providers in the palliative care sector. This

followed PHARMACs decision to stop funding MST branded slow release morphine in favour of M Eslon. M Eslon appeared to have only an eight hour duration of action rather than the 12 hours previously experienced by MST. This led to widespread concern amongst palliative care patients. PHARMAC as usual totally ignored their concerns and it was a great relief when a new product that worked well did become available.

Oxycontin and Oxynorm have been my first-line choice where an opiate is medically indicated because of this previous experience.

***Dr Ross Ogle,** General Practitioner*
Tauranga

It is pleasing that points made in the article "Oxycodone – How did we get here and how do we fix it?" (BPJ 62, July, 2014) have been found to be relevant to our correspondent and useful in his clinical practice.

It is also interesting to review some of the history of the introduction of m-Eslon and oxycodone in New Zealand. When prescribing a new product, be it a new medicine (in the case of oxycodone) or a new product formulation (in the case of m-Eslon) there will be a period of gaining experience with prescribing it, understanding its effect and effectiveness for patients, and learning where it fits into your own, and wider, clinical practice.

As substantiated by our correspondent, when m-Eslon became funded on the Pharmaceutical Schedule there were anecdotal reports that its analgesic effect did not always last the expected 12-hour duration; this may have been more prevalent at lower daily doses of m-Eslon. However, there have also been reports of the MS Contin formulation not controlling cancer pain using a 12-hourly dosing regimen: in an review of early studies of patients with moderate to severe cancer-related pain who were transferred from a 4-hourly, immediate-release morphine dosing regimen to 12-hourly, sustained release MS Contin, 93% of patients were successfully treated with 12-hourly MS Contin, but 7% required 8-hourly MS Contin dosing.*

Similarly, we have learned more about oxycodone and its formulations as experience has been gained. So is it the medicine, the new formulation, the patient or the change?

* Kaiko R, Grandy R, Oshlack B, et al. The United States experience with oral controlled-release morphine (MS Contin tablets). Cancer 1989;63:2348-54.

Managing patients expectations when prescribing oxycodone

Dear Editor,
Re: A Disaster in the making
I enjoyed reading Dr McMinn's impassioned discussion in your June 2014 edition [BPJ 61] warning of an opioid tsunami heading towards New Zealand. As a New Zealand GP on a locum adventure to a single doctor outback town in NSW a few years ago, I remember my bewilderment on discovering a staggering 15% of consultations related to the renewal of prescriptions of Australia's favourite opioid, oxycodone. Patients seemed evenly divided between those purportedly suffering from chronic back pain and those professing to be hopelessly addicted to strong analgesics. All pleaded their case that an increase in dose was surely required and hoped that I had a better understanding of their suffering than their regular doctor. It was hard to know what to believe and even harder to know what approach to take. Should I adopt the path of least resistance and risk escalation of the community drug problem or put my foot down, refuse to play ball and risk a riot and the hard-working GP returning and wishing he had not left me in charge? I chose the middle ground; refusing to escalate analgesia and documenting a sufficient degree of concern in the medical records hoping the returning GP might be able to use this as leverage.

I would be most interested in Dr McMinn's comments on the use of combined preparations of oxycodone and naloxone available in Australia and other countries. Evwwidence seems to suggest that constipation is lessened without reducing analgesic effect. It is promoted as having less potential for abuse but I am uncertain if there is evidence of this. Is this a product we should be making available in New Zealand?

***Dr Kerr Wright,** General Practitioner*
Bay of Plenty (currently working overseas)

Response from Dr Jeremy McMinn:
Dr Wright's experiences are common – "inheriting" patients in whatever capacity, e.g. as a locum, present compelling opportunities to re-consider diagnosis and the consequences of earlier treatment. The patients on long-term opioids seeking escalating doses demonstrate the need to review the original treatment contract in the light of incomplete recovery or, worse, emerging iatrogenic deficits.

How to address this depends on which prescriber(s) will be able to see a revised treatment approach through. The duration of the cover will determine how much can be tackled – often setting the scene for change (but not actually making changes) is a powerful tool to hand over to the returning doctor, who may be grateful for the opportunity to use a colleague's second opinion to good effect. But if you are taking over the prescribing for the longer haul, this needs to fit with your prescribing integrity – after six months, every treatment contract with the patient is yours, not the earlier doctor's!

The combination of oxycodone and naloxone has much false promise, in my view. The key concern is that this maintains a perspective that chronic opioids are a normal, valid, readily used intervention – we should be questioning this with wise and grave doubt.

As for the alleged effects on constipation and abuse potential, it is important to distinguish between evidence and marketing. There is little good evidence for oxycodone, but one can extrapolate from other combination attempts. Naloxone, when combined with buprenorphine (in the form of Suboxone), has no clinically useful effect on constipation. Research by Larance et al*[*] *demonstrated that even buprenorphine with naloxone in a buccal film preparation was still subject to significant rates of abuse. That having been noted, buprenorphine alone may have a greater abuse potential: the same may be true for oxycodone alone.*

The analogy of low tar cigarettes springs to mind – the gains are so little, but the product has a seductive sense of being safer.

* Larance B, Lintzeris N, Ali R, et al. The diversion and injection of a buprenorphine-naloxone soluble film formulation. Drug Alcohol Depend 2014;136:21-7.

ACKNOWLEDGEMENT: Thank you to **Dr Jeremy McMinn**, Consultant Psychiatrist and Addiction Specialist, Wellington for providing this response.

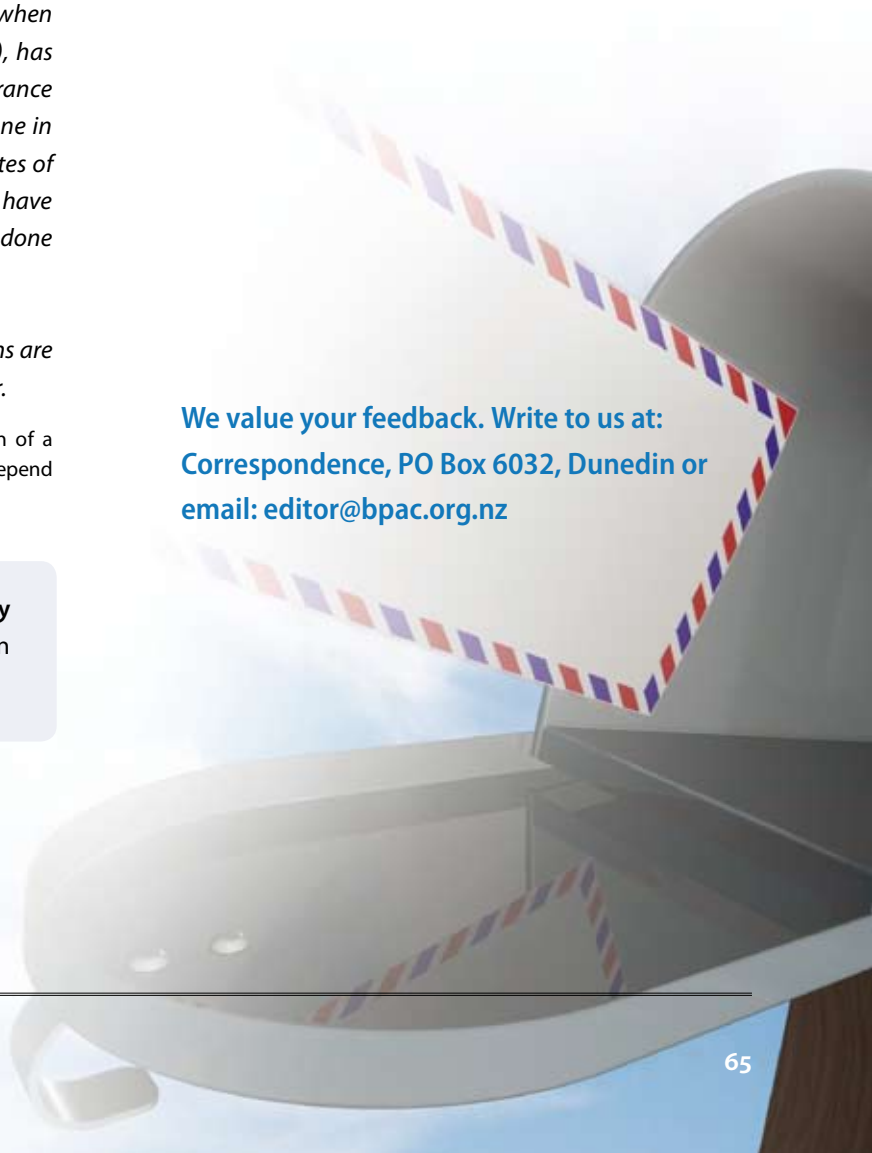
Incorrect graphic in oxycodone article

Dear Editor,

Page 24 of Issue 62 (July, 2014) of Best Practice Journal carries a graphic of a tablet bottle with an oxycodone hydrochloride label on it. I wonder if it was intentional that the label also had "Pharmacy Only Medicine" displayed? I'm sure you're aware that oxycodone is classified as a Controlled Drug and not a Pharmacy-Only Medicine and as such, it is inappropriate, and illegal for a real tablet bottle for oxycodone to bear the words "Pharmacy Only Medicine". I understand that this is just a graphic, but a reasonable level of accuracy in your publication is expected.

***Andrew Orange**, Pharmacy Advisor*
MidCentral DHB, Palmerston North

We apologise for this oversight. You are correct, this was just a graphic and is not a real dispensing container. We have corrected this graphic in our online version of this article.



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