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Managing medicines in older people

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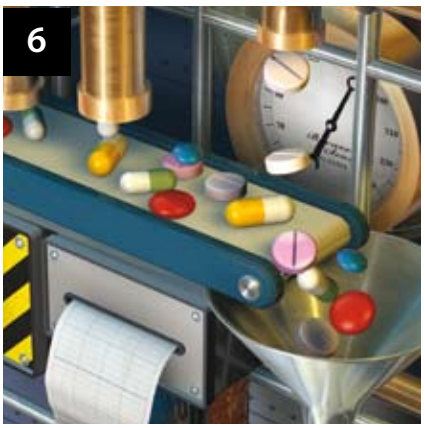

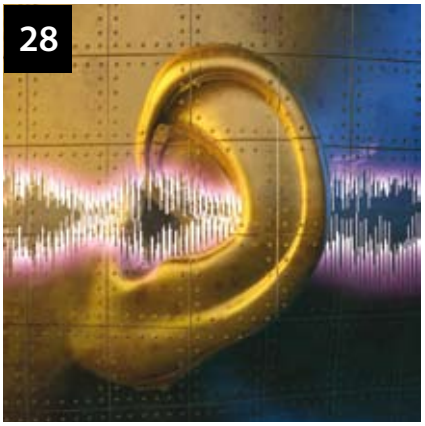

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New national genetic service launched

A new national genetic service has been established to improve access to genetics clinics and genetic testing in New Zealand. Genetic Health Service New Zealand (GHSNZ), launched in May 2012, provides genetic diagnosis, genetic counselling, assistance in managing genetic conditions and expert advice and education on genetic diseases.

GHSNZ encompasses the former Northern, Central and Southern Regional Genetics Services. The service is funded by the National Health Board, and provided by Auckland and Capital and Coast DHBs, and is part of the public health system. Genetic services are delivered from three main centres: Christchurch (South Island Hub), Wellington (Central Hub) and Auckland (Northern Hub), and outreach clinics throughout the country.

The Northern Hub covers the upper half of the North Island. It is based at Auckland Hospital and provides outreach clinics in Whangarei, Hamilton, Tauranga, Rotorua and Gisborne.

The Central Hub covers the lower half of the North Island and Nelson/Marlborough. It is based at Wellington Hospital and provides outreach clinics in New Plymouth, Whanganui, Hastings, Palmerston North, Porirua, Lower Hutt and Nelson-Marlborough.

The South Island Hub covers all of the South Island except Nelson/Marlborough. It is based at Christchurch Hospital and provides outreach clinics in Greymouth, Timaru, Dunedin, Queenstown and Invercargill.

Who provides the services?

GHSNZ clinical staff includes clinical geneticists and genetic associates.

Clinical Geneticists are doctors with specialist training in medical genetics. Patients are referred to them for diagnosis of a genetic condition.

Genetic Associates are health professionals with post-graduate qualifications in medical genetics and genetic counselling. Patients are likely to see them after their condition has been diagnosed.

What services are provided?

GHSNZ services include:

- Assistance in the diagnosis, clinical management of genetic disease and identification of preventable complications by early surveillance
- Advice about inheritance of genetic conditions, further information and genetic testing for those affected by or perceived to be at risk of genetic disorders in extended families
- Telephone enquiry service for doctors, midwives and other health professionals concerning genetic diseases
- Genetics education for professional and lay groups

Conditions frequently seen at genetic clinics in New Zealand

Patients most often seen in New Zealand genetics clinics include those with:

- Abnormal pre-natal scans or screening tests, e.g. amniocentesis with abnormal karyotype
- Dysmorphic features and developmental delay
- Neurological disorders with features suggestive of an inherited condition
- Fragile X syndrome
- Connective tissue disorders, e.g. Marfan syndrome, Ehlers-Danlos, osteogenesis imperfecta
- Familial cancer syndromes, e.g. hereditary breast and ovarian cancer, Lynch syndrome
- Metabolic disorders
- Inherited cardiac conditions, e.g. Long QT syndrome, hypertrophic cardiomyopathy
- Cystic fibrosis
- Muscular dystrophies (Duchenne, Becker, myotonic)
- Chromosome alterations
- Adult onset disorders of many systems, e.g. renal, ophthalmology, respiratory

Genetic Testing

Genetic testing should only be requested by GHSNZ clinical staff (or by a doctor in their specific scope of practice). Written consent is required by most reference laboratories. The majority of genetic testing is done overseas and is expensive.

All ages are covered, including:

Pre-conception/prenatal:

- Diagnostic, pre-conceptional, pre-natal or pre-symptomatic tests for a genetic condition and reproductive options including pre-implantation genetic diagnosis (PGD)
- Diagnostic assessment if there are concerns about foetal abnormalities during pregnancy
- Information and genetic counselling concerning prenatal diagnosis

Childhood:

- Diagnostic assessment of infants or children with dysmorphic features or developmental delay
- Genetic assessment for a child and their family/whānau with a likely genetic disorder
- Post-mortem review where a genetic disorder is suspected

Adulthood:

- Genetic risk assessment and testing for familial cancer syndromes
- Diagnostic assessment for adult-onset genetic disorders
- Explanation and further information about a genetic diagnosis
- Discussion of the implications of a family history of a genetic condition
- Recommendations for surveillance and management of genetic conditions

Patients seen through GHSNZ receive a detailed explanatory letter after their assessment, with copies sent to their General Practitioner and referring doctor (if different).

Who should be referred to the genetic service?

Referrals should be considered for a patient with an identified family history of a genetic disorder, or where a patient has been diagnosed with a genetic disorder or is at risk of a genetic disorder.

A common reason for referral to genetics services is for familial cancer syndromes. Approximately 5% of people with breast cancer and 2% of people with colon cancers have an inherited genetic susceptibility. There are more than 50 familial cancer syndromes, which increase susceptibility for specific cancers. Genetic testing is available for most familial cancer syndromes, but screening for a mutation usually has to start in an affected family member.

Referral is recommended for people with:

- A blood relative who is a known carrier of a familial cancer gene mutation, e.g. BRCA1, BRCA2, MLH1, MSH2, APC
- Ashkenazi Jewish ethnicity (increased occurrence of familial cancer syndromes)
- A personal or family history of breast cancer under age 40 years
- Male breast cancer
- High-grade serous ovarian cancer (any age)
- Colon cancer under age 40 years
- Multiple primary tumours (excluding lung, skin, cervix) under age 70 years, e.g. breast and ovarian, endometrial and colorectal cancer
- Rare tumour under age 45 years or any age if there is a close relative with a similar tumour, e.g. phaeochromocytoma, paraganglioma, sarcoma, glioblastoma, choroid plexus carcinoma, retinoblastoma
- Two or more first or second degree relatives on the same side of the family with colon or endometrial cancer with one diagnosed under age 60 years
- Two or more first or second degree relatives on the same side of the family with breast cancer under age 60 years and/or ovarian cancer at any age

How do you refer a patient to the genetic service?

Referrals to clinics can be arranged by faxing or posting a request to Auckland, Wellington or Christchurch centres. Detailed medical information is usually required.

Contact addresses:

Genetic Health Service NZ – Northern Hub
Auckland Hospital
Private Bag 92 024
Auckland Mail Centre
Auckland 1142
Ph: (09) 307 4949 Ext. 25870
Toll Free: 0800 476 123
Fax: (09) 307 4978
Email: GenSec@adhb.govt.nz

Genetic Health Service NZ – Central Hub
Wellington Hospital
Private Bag 7902
Wellington South
Ph: (04) 385 5310
Toll free: 0508 364 436
Fax: (04) 385 5822
Email: genetic.services@ccdhb.org.nz

Genetic Health Service NZ – South Island Hub
Christchurch Hospital
Private Bag 4710
Christchurch 8140
Ph: (03) 378 6574
Toll free: 0508 364 436
Fax: (03) 379 1343
Email: genetic.servicenz@cdhb.health.nz

For more information see:
www.geneticshealthservice.org.nz

GENETIC HEALTH SERVICE
NEW ZEALAND

ACKNOWLEDGEMENT Thank you to **Dr Caroline Lintott**, Senior Genetic Associate/Team Leader, Genetic Health Service New Zealand, South Island Hub, for contributing this article.

Managing medicines in older people

Primary care oversight of prescribing to older people can reduce both inappropriate prescribing and patient risk. By asking specific questions in consultations and using validated tools to eliminate unnecessary medicines, clinicians can further improve prescribing to older people with complex co-morbidities.

Prescriber responsibility and polypharmacy

It is increasingly recognised that health care for older people is improved when one prescriber takes responsibility for all of a patient's medicines.[1] An important reason for this is that multiple prescribers are associated with increased polypharmacy (Figure 1). This exposes patients to increased risk, as the probability of an adverse drug reaction occurring increases as the patient takes more medicines.[2] Multiple prescribers are also an independent risk factor for adverse drug reactions in older populations.[3]

Polypharmacy among older populations is also significantly associated with hypoglycaemia, malnutrition, pneumonia, fractures, hospitalisation and death.[5] An analysis of national dispensing data showed that 44% of enrolled patients aged over 75 years in New Zealand used five or more medicines continuously over a nine month period in 2011/2012.[4]

A single prescriber provides consistency

Oversight of medicines by a single prescriber allows for consistent advice and decision making. This is important when treating elderly people, as prescribing recommendations are often based on studies of younger populations where co-morbidities have been excluded. In older patients with complex co-morbidities, strict adherence to such guidelines can result in confusing regimens with a high probability of adverse drug reactions.[6] Prescribers need to tailor interventions to an individual's overall risk, as the benefits gained by participants in clinical trials may not be generalisable to an older person with multiple co-morbidities. In some cases, it may be jointly decided by patient and clinician to stop, or not to initiate, treatment.

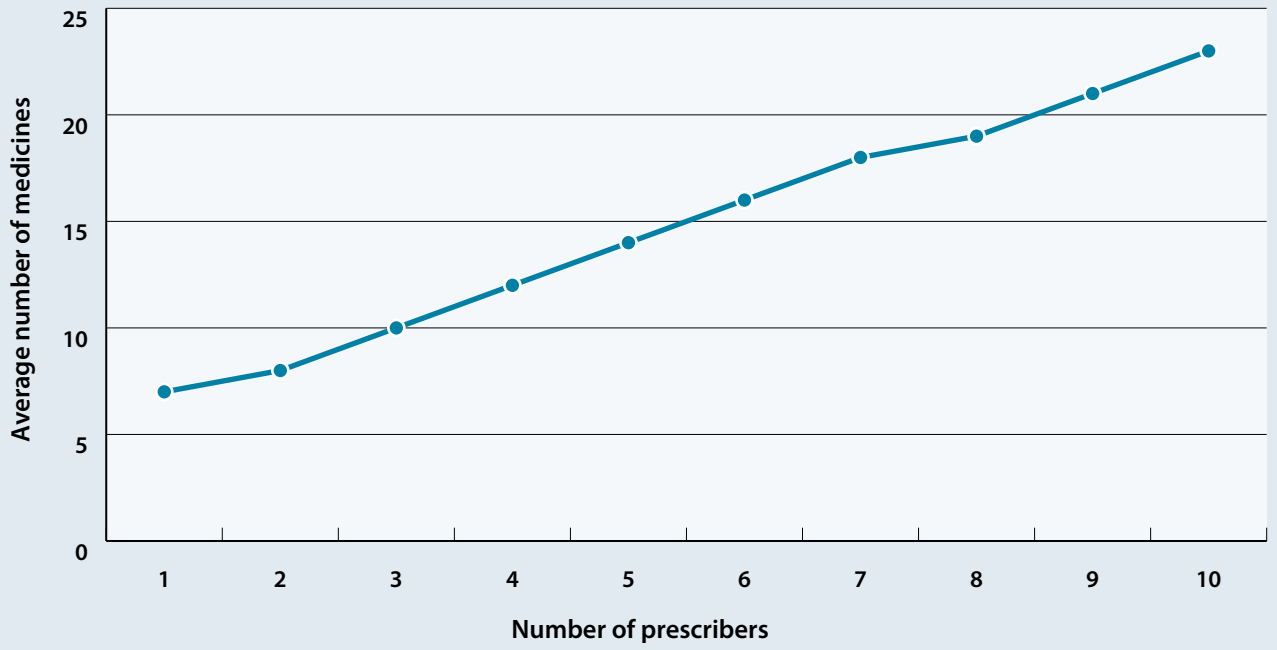

Figure 1: Average number of medicines prescribed per patient (Jul 2011 – Mar 2012) for all patients aged over 75 years in New Zealand, by number of prescribers[4]

Adopting a standardised approach to prescribing for older people

Three essential elements of prescribing to older people in primary care are:

1. The introduction of a standardised format when assessing medicine use
2. Avoiding inappropriate prescribing
3. The use of validated tools to withdraw unnecessary medicines

Standardising consultations with routine questions

What medicines are you currently taking?

Older people frequently have multiple co-morbidities and reduced renal and hepatic function. They are therefore more likely to be affected by adverse drug reactions than younger people.

It is also important to find out if the patient is adhering to their prescribed medicines. Medicine non-adherence in older people may occur due to forgetfulness, adverse effects, limited organisational skills or belief that the medicine is unnecessary, ineffective or too costly.[7] Reports of non-adherence in older people varies from one-quarter to over one-half, depending on the definition and the population studied.[7] Simple questions about what, why and how patients take their medicines can reveal gaps in understanding and compliance that otherwise may not be volunteered. Medicine organisers or blister packs may be introduced for patients who experience confusion or forget to take their medicines regularly.

What medicines do you buy yourself and do you take medicines prescribed for anybody else? Some patients may not consider products that they have purchased from the supermarket or health store as medicines. Specifically asking about medicine purchasing habits may reveal relevant information. For example, an older person with chronic kidney disease taking angiotensin converting enzyme inhibitors (ACEIs) and diuretics will be at increased risk of developing acute kidney injury if they also take ibuprofen purchased from a supermarket.

Sharing of medicines is also common among older people. Survey results indicate that 13 – 20% of older people have shared their medicine with another person.[8]

Over-the-counter (OTC) products and complementary and alternative medicines can also have significant interactions with prescribed medicines, e.g. warfarin in combination with significant quantities of garlic, ginger, ginseng or ginkgo may result in an increased risk of bleeding.[9] One study of over 1000 people, with a mean age of 76 years, found that approximately one-third of participants had complementary medicines in their home, with almost all of these in active use.[10] The most common potential adverse effects due to interactions between prescribed medicines and alternative products were: increased risk of bleeding due to non-steroidal anti-inflammatory drugs (NSAIDs) interacting with vitamin E, fish oils, ginkgo and garlic, or hyperkalaemia caused by ACEIs and concurrent use of potassium or arginine supplements.[10]

Have you visited another doctor or been admitted to hospital?

Primary care management of medicines requires timely information about prescribing changes initiated in secondary care. Asking patients if they have visited another health professional is a simple way of checking if their medicine regimen has been altered. If the patient has had a consultation with another clinician, ask if they were prescribed any new medicines, or if they were told that they could stop taking medicines that had been previously prescribed.

Hospital admission in older people is associated with an increase in the number of long-term medicines taken. A New Zealand study of over 400 older people found that following a stay in hospital, the average number of medicines prescribed to patients increased from 6.6 to 7.7.[11] If secondary care prescribers are unaware of all the medicines a patient is taking, the risk of prescription duplication and/or adverse drug reactions are increased. Furthermore, some medicines initiated in secondary care may not be intended for long-term use, e.g. omeprazole taken for prophylaxis against stress ulceration before surgery. Secondary care initiation of uncommon medicines can also present challenges to primary care prescribers assessing the risk and benefits of continuing these medicines. Similarly, if secondary care prescribers are unaware of the indications for continuing some medicines, e.g. antidepressants, appropriate treatment may be inadvertently withdrawn.

Best Practice tip: Practices may consider implementing a system where older patients who have been recently discharged from hospital for non-routine events, are phoned and offered a follow-up consultation to discuss any possible changes to their treatment.

Do you have any concerns about any medicines you are taking?

Regularly assessing medicine effectiveness (including asking patients if they feel their medicines help) and monitoring for adverse effects allows for appropriate dose adjustment and the risk of long-term adverse drug reactions to be minimised. This question also provides a prompt for some older patients who may just "put up" with adverse effects without mentioning them. Discussing concerns may also increase patients' health literacy and improve treatment adherence.

Actions and questions for prescribers

Medicines information gathered from consultations can be combined with information taken from patient records (including hospital discharge notes and dispensing record) to reconcile the list of medicines a patient is taking.

Performing medicine reconciliations and medicine reviews

Medicine reconciliation is the systematic process of obtaining a complete list of all of a patient's medicines. It should be performed whenever a patient is seen for the first time, or discharged from hospital. All medicines, including OTC medicines, supplements and alternative products should be recorded in the PMS. This list should include doses, regimen, administration routes and last dose taken.[12] All drug allergies, intolerances and previous treatment terminations, and the reasons for discontinuation, should also be recorded.

Many older patients take their medicines from blister packs and have limited knowledge about some medicines prescribed to them. Suggesting that patients bring their medications to the consultation and performing a "brown-bag review" can assist the reconciliation process.

Medicine reviews aim to eliminate medicines that are no longer required or have a high risk of toxicity. These should be conducted annually for all older patients taking medicines and every six months for those taking multiple medicines, e.g. five or more medicines taken simultaneously.[1] However, thought should be given to the appropriateness of medicines every time a prescription is renewed.

Pharmacist and/or nurse participation in the reconciliation process has been shown to be beneficial (see: "The Pharmacy Services Agreement", over page). A Canadian study found that in 120 patients aged over 50 years, following review by a pharmacist and a nurse practitioner, the number of inappropriate medicines decreased from 27% to 9% and the

Patient Safety Incident Reporting

An example of multiple prescribers contributing to an adverse drug interaction occurred when a patient who had been taking simvastatin 40 mg for several years was prescribed cyclosporin for psoriasis by his dermatologist. Cyclosporin is known to increase the risk of myotoxicity when taken with simvastatin. The prescription was dispensed at the patient's community pharmacy where the dispensing pharmacist also failed to notice the potential interaction. After several weeks the patient developed rhabdomyolysis and was admitted to hospital with acute kidney injury.

More examples are available from:
www.bpac.org.nz/safety

The Pharmacy Services Agreement – The changing role of the community pharmacist

Under the new Long Term Conditions (LTC) rules of the Pharmacy Services Agreement, community pharmacists will now be identifying patients with complex co-morbidities and/or difficulties with medicine adherence. To assist pharmacists, an objective assessment tool has been developed. Patients can be referred to be assessed by indicating "Refer for LTC assessment" on a prescription or by contacting the pharmacist directly. Family members and patients themselves can also request an LTC assessment.

Pharmacists will be funded to assist in the management of patients referred for LTC assessment. Part of the assessment will be to determine dispensing frequency. Patients who are currently dispensed their medicines under Close Control can remain on this dispensing frequency until they are assessed for LTC eligibility. Pharmacists have until 31 January 2013 to complete these assessments.

In addition to the LTC service, some DHBs are still funding medicine use reviews by accredited pharmacists to improve patient education and adherence. To find out if this service is available in your area, contact your local DHB.

For further information see: "New service model for community pharmacy", BPJ 45 (Aug, 2012) or **www.pharmac.govt.nz/ccc**

number of patients taking at least one inappropriate medicine decreased from 78% to 39%.[13]

Specifically trained clinical pharmacists can provide another level of expertise by contributing to medicine management through clinical recommendation.

Before prescribing a medicine, consider the following:

Am I treating the condition or a symptom? It is important to establish a diagnosis so that treatment will target the underlying process rather than an isolated symptom. If a patient is taking multiple medicines, clinicians should exclude the possibility that any new symptoms may be due to adverse drug reactions.

Be aware of the possibility of the prescribing cascade concept, which can begin when an adverse drug reaction is misinterpreted as a new medical condition. This can result in a new medicine being unnecessarily prescribed to treat the adverse reaction which in turn increases the risk of the patient experiencing another adverse drug reaction. An example of this is the use of prochlorperazine for the treatment of dizziness in patients taking medicines such as antihypertensives.[14] Prochlorperazine is associated with postural hypotension and may exacerbate the original symptom and can also cause drug-induced parkinsonism. These adverse effects may explain the resulting 50% increase in the observed rates of hip fracture in older people following initiation of prochlorperazine.[14]

Have I considered non-pharmacological treatment? Non-pharmacological interventions, in some cases, can provide both physical and psychological health benefits to older people, e.g. physiotherapy for chronic back pain. Improving nutrition and physical activity can also reduce people's risk of chronic disease and increase their independence. The introduction of mobility aids, e.g. a walking frame, may also improve quality of life.

The Ministry of Health provide nutrition guidelines for healthy older people, available from: **www.health.govt.nz/search** (key words = nutrition older)

Is the medicine appropriate to the patient's condition and stage of life? When considering prescribing medicines to older people it can be useful to group medicines into those that improve quality of life, e.g. analgesics, and those used for disease prevention. The benefit of medicines that improve quality of life is clearly evident. However, preventative interventions require (to some degree) the clinician convincing the patient, and themselves, that treatment will provide benefit.

For example, the Prospective Study of Pravastatin in the Elderly at Risk of vascular disease (PROSPER) trial showed that while pravastatin given to over 5000 participants aged 70 – 82 years did reduce cardiovascular morbidity and mortality, it was not associated with a reduction in all-cause mortality.[17] This was partially explained by an increased incidence of new cancer diagnosis, suggesting that for some patients, preventing one cause of death simply revealed another. Some older patients, who had elected to take a statin to reduce cardiovascular risk for primary prevention, may not have made the same decision if they had been told that treatment was unlikely to prolong their life. However, aggressive statin treatment may be appropriate for a patient who has previously had a stroke and wishes to reduce the risk of a second, potentially more disabling event from occurring.[18] Clearly, discussions about medicines need to be focused on the individual preferences of the patient and their specific circumstances and life stage.

Assessing prescription appropriateness

Factors which should be considered when prescribing medicines to older people include:[19]

- Remaining life expectancy (Table 1)
- Time until benefit of treatment
- Treatment target
- Goals of care

Consider using prescribing tools

Prescribing tools provide specific examples of medicines that may be inappropriate for older people and are an effective way of reducing adverse drugs reactions.

The STOPP/START criteria were developed to improve prescribing to patients aged over 65 years. The criteria consist of two components; the first to halt inappropriate or unnecessary medicines in older patients, the second is used to consider medicine appropriateness when initiating treatment. An advantage of the STOPP/START criteria is that it accounts for patient co-morbidities. The STOPP/START criteria was considered by an expert panel to be the most appropriate tool for measuring treatment adherence and appropriateness in patients with multiple co-morbidities.[12]

The STOPP/START toolkit can be accessed from: **www.cumbria.nhs.uk** (key words = STOPP/START).

The Beers criteria consists of three lists of medicines ranked according to their potential for causing adverse reactions in older people. It was recently updated by the American Geriatrics Society (AGS) in 2012.[21] However, the criteria's usefulness is limited as it does not take into account individual patient needs and co-morbidities.

The first grouping is medicines that are known to cause adverse reactions in older people. The second classification is medicines that may be inappropriate for older people with specific diseases or risks factors. The third list is medicines that should be prescribed to older people with caution.

For further information see the American Geriatrics Society. Available from: **www.americangeriatrics.org** (keyword search = Beers).

Table 1: Life expectancy by age for older New Zealand male and female populations, 2009 – 2011[20]

Male		Female	
Age years	Expected number of years of life remaining	Age years	Expected number of years of life remaining
65	18.8	65	21.2
70	15.0	70	17.1
75	11.5	75	13.3
80	8.6	80	9.8
85	6.2	85	6.9
90	4.5	90	4.8

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

1. Stockley's Interaction Alerts
2. British National Formulary (BNF) interaction summaries

For further information see: Medicine interactions: using the New Zealand Formulary", BPJ 46 (Sep, 2012).

When medicines are prescribed

Consider if there are any medicines that can be altered or stopped if a new medicine is started, e.g. if the patient is switched from an NSAID to an opioid, a gastro-protective medicine may no longer be necessary.

If repeating a prescription, confirm that the original is still applicable, e.g. if a loading dose of colecalciferol has been prescribed, check that subsequent prescriptions do not repeat the loading dose.

Double check the prescription is correct both before printing it out and handing it to the patient.

Ensure the patient knows what medicine they have been prescribed and how it should be taken. Where appropriate, encourage people to take educational material home about both the medicine prescribed and the condition being treated. This can increase patient understanding and family/ whānau involvement in treatment.

When finishing a consultation a good approach to see if the patient has understood a message is to ask them what they will say to their partner or family when they return home.

The Ministry of Health website provides information and links to patient resources for diseases that are common in older people, e.g. cardiovascular disease, cancer and diabetes. See: **www.health.govt.nz**

Regular monitoring for adverse effects and treatment adherence

It has been suggested that suboptimal monitoring of older people taking medicines may be a more significant problem than inappropriate prescribing.[8] Effective monitoring requires regular recording of parameters such as body weight and blood pressure, scheduling of relevant laboratory tests, e.g. serum creatinine and HbA_{1c}, regular communication between patient and clinician and a good knowledge of the patient's cognitive function and general wellbeing.

The danger of under-prescribing

Under-prescribing is the omission of medicines which are generally recommended by clinical guidelines for the treatment or prevention of a disease or condition.[15] A study of 123 patients aged over 60 years found that approximately one-third of instances of under-prescribing were not clearly justified.[15]

Commonly under-prescribed treatments for older people include medicines for the secondary prevention of coronary and cerebrovascular diseases and osteoporotic fractures.[8] In particular, there is good evidence of short-term benefits gained from continuing treatment of hypertension in people aged over 80 years.[16]

Although inappropriate under-prescribing should be avoided, it is complicated by the fact that clinical guidelines are generally for single conditions and do not take into account co-morbidities. The use of several different guidelines for one patient with multiple conditions may lead to inappropriate prescribing, if all advice is followed concurrently.[6]

If prescribers are unsure whether a medicine is appropriate for an older patient then discussion with a geriatrician, general physician or clinical pharmacist is recommended.

When to decide to withdraw a medicine

Medicine withdrawal should be considered for older people when:[22]

- There are a large number of medicines in use
- Adverse drug reactions are suspected or falls have occurred
- Treatment is ineffective
- The goals of care change due to declining organ function

A study investigating inappropriate medicine use in 70 community dwelling older people recommended discontinuation of 311 medications in 64 people.[23] Of the medicines that were discontinued, only 2% were restarted due to the recurrence of the original indication.[23]

Medicine groupings that may be appropriate for withdrawal in older people include; anticholinergics, antihistamines, antiplatelet medicines, centrally acting medicines (e.g. antipsychotics, benzodiazepines), diuretics, NSAIDs, proton pump inhibitors and vasodilators.

 For further information see: "A practical guide to stopping medicines in older people", BPJ 27 (Apr, 2010).

If a medicine is withdrawn due to a suspected adverse drug reaction, symptom resolution can be expected once the medicine has been cleared from the patient's blood (within three to five half-lives) – if the reaction is dose dependent.[22] Symptoms such as delirium and skin reactions and those caused by renal or hepatotoxicity may take considerably longer to resolve.

ACKNOWLEDGEMENT Thank you to **Dr Carl Hanger**, Geriatrician, Older Persons Health Specialist Service, The Princess Margaret Hospital, Canterbury DHB for expert guidance in developing this article.

The "Pill Pruner"

A medication review guide for frail, older patients, the Pill Pruner, has been developed in New Zealand. The guide condenses the STOPP/START criteria into the 13 most potentially inappropriate medicines relevant to New Zealand prescribing practice. These are printed on a pocket-sized card.

The Pill Pruner was tested at Christchurch Hospital on two groups of 500 people aged over 75 years, consecutively admitted to hospital. On admission, over 70% of patients were taking five or more medicines. Before the Pill Pruner was introduced, the mean number of medicines on discharge increased from 6.8 to 7.7. When the Pill Pruner was introduced, no statistically significant increase in discharge medicines occurred (medicines increased from 6.3 to 6.5). Over the course of the study over 1000 medicines were stopped including: loop diuretics, antiplatelet medicine, statins, ACEIs, β-blockers and benzodiazepines. There appeared to be no harmful effects of medicine withdrawal. Medicine changes were communicated to the patient's General Practitioner and community pharmacist. Of the General Practitioners who responded to follow-up questionnaires, less than 10% reported a need to restart medicines in patients.

The Pill Pruner has yet to be trialled in primary care. As hospital admission is frequently a period of acute illness and increased need, use of the Pill Pruner in general practice may be even more effective. Publication of the results of the Pill Pruner study is underway.

 For further information see: "Polypharmacy in the elderly – evaluating the Pill Pruner project." Available from: **www.otago.ac.nz/christchurch/research/publichealth/studentships/otago013401.html**

How to "de-prescribe" and issues to be aware of

Before "de-prescribing" an unnecessary medicine, a discussion should be held with the patient as to why the change has been suggested and what can be expected. This is important, as many medicines have a placebo component to their effectiveness. Explain that the process will be monitored.

If there is more than one medicine to be de-prescribed, these should be prioritised and withdrawn one at a time. The dose should be slowly reduced over a period of weeks to months and the patient warned and monitored for signs of:[22]

- Withdrawal – common in medicines acting on the central nervous system
- Rebound symptoms – e.g. rebound tachycardia and hypertension following beta blocker withdrawal, or rebound gastric acid secretion following withdrawal of a proton pump inhibitor
- Unmasked drug interactions – e.g. the INR of a normally stable patient taking warfarin may decrease over several weeks following cessation of amiodarone

Clinicians should consider if any new symptoms are due to withdrawal or a re-emergence of the indicated condition. If the patient experiences significant withdrawal, the medicine can be reintroduced, possibly at a reduced dose. Assessments should also focus on the beneficial aspects of de-prescribing, which can reinforce patient adherence and the prescriber's confidence to reduce medicines.

References

1. Milton JC, Hill-Smith I, Jackson SHD. Prescribing for older people. BMJ 2008;336(7644):606–9.
2. Goldberg RM, Mabee J, Chan L, Wong S. Drug-drug and drug-disease interactions in the ED: analysis of a high-risk population. Am J Emerg Med 1996;14(5):447–50.
3. Green JL, Hawley JN, Rask KJ. Is the number of prescribing physicians an independent risk factor for adverse drug events in an elderly outpatient population? Am J Geriatr Pharmacother 2007;5(1):31–9.
4. bpac^{nz}. Polypharmacy in people aged over 75 years. Oct, 2012. Available from: www.bpac.org.nz (Accessed Oct, 2012).
5. Frazier SC. Health outcomes and polypharmacy in elderly individuals: an integrated literature review. J Gerontol Nurs 2005;31(9):4–11.
6. Boyd CM, Darer J, Boult C, et al. Clinical practice guidelines and quality of care for older patients with multiple comorbid diseases: implications for pay for performance. JAMA 2005;294(6):716–24.
7. Bilotta C, Lucini A, Nicolini P, Vergani C. An easy intervention to improve short-term adherence to medications in community-dwelling older outpatients. A pilot non-randomised controlled trial. BMC Health Serv Res 2011;11:158.
8. Elliott R. Problems with medication use in the elderly: An Australian perspective. J Pharm Pract 2006;36(1):58–66.
9. Ramsay NA, Kenny MW, Davies G, Patel JP. Complimentary and alternative medicine use among patients starting warfarin. Br J Haematol 2005;130(5):777–80.
10. Ientile C, Sorensen L, Lemanski L, Roberts M. Complementary medicines use by Australian veterans. J Pharm Pract Res 2005;35(2):110–1.
11. Betteridge TM, Frampton CM, Jardine DL. Polypharmacy - we make it worse! A cross-sectional study from an acute admissions unit. Intern Med J 2012;42(2):208–11.
12. Alfaro Lara ER, Vega Coca MD, Galván Banqueri M, et al. Selection of tools for reconciliation, compliance and appropriateness of treatment in patients with multiple chronic conditions. Eur J Intern Med 2012;23(6):506–12.
13. Fletcher J, Hogg W, Farrell B, et al. Effect of nurse practitioner and pharmacist counseling on inappropriate medication use in family practice. Can Fam Physician 2012;58(8):862–8.
14. Caughey GE, Roughead EE, Pratt N, et al. Increased risk of hip fracture in the elderly associated with prochlorperazine: is a prescribing cascade contributing? Pharmacoepidemiol Drug Saf 2010;19(9):977–82.
15. van den Heuvel PML, Los M, van Marum RJ, Jansen PAF. Polypharmacy and underprescribing in older adults: rational underprescribing by general practitioners. J Am Geriatr Soc 2011;59(9):1750–2.
16. Beckett N, Peters R, Tuomilehto J, et al. Immediate and late benefits of treating very elderly people with hypertension: results from active treatment extension to Hypertension in the Very Elderly randomised controlled trial. BMJ 2012;344:d7541.
17. Mangin D, Sweeney K, Heath I. Preventive health care in elderly people needs rethinking. BMJ 2007;335(7614):285–7.
18. Amarenco P, Bogousslavsky J, Callahan A III, et al. High-dose atorvastatin after stroke or transient ischemic attack. N Engl J Med 2006;355(6):549–59.

19. Holmes HM, Hayley DC, Alexander GC, Sachs GA. Reconsidering medication appropriateness for patients late in life. Arch Intern Med 2006;166(6):605–9.

20. Statistics New Zealand. New Zealand abridged life table, 2009-2011. 2012. Available from: www.stats.govt.nz (Accessed Oct, 2012).

21. American Geriatrics Society. American Geriatrics Society: Updated Beers Criteria for potentially inappropriate medication use in older adults. J Am Geriatr Soc 2012;60(4):616–31.

22. Le Couteur D, Banks E, Gnjidic D, McLachlan A. Deprescribing. Aust Prescr 34(6):182–6.

23. Garfinkel D, Mangin D. Feasibility study of a systematic approach for discontinuation of multiple medications in older adults: addressing polypharmacy. Arch Intern Med 2010;170(18):1648–54.

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Appropriate use of tetracyclines

What are tetracyclines and how do they work?

Tetracyclines are broad spectrum antibiotics which are active against a number of microbes including; chlamydiae, mycoplasmas, rickettsiae, mycobacteria, spirochetes, many aerobic and anaerobic Gram-positive and Gram-negative pathogenic bacteria and some protozoa.[1]

Tetracyclines were first discovered in the 1950s (first generation), and newer agents were introduced in the late 1960s with a longer half-life (second generation). Recently third generation agents have been developed, such as tigecycline which is used intravenously for difficult to treat infections in the hospital setting.

There are two second generation tetracycline antibiotics available on the Pharmaceutical Schedule in New Zealand – doxycycline (fully subsidised) and minocycline (partially subsidised). Other tetracyclines include lymecycline (not subsidised) and demeclocycline (only available under Section 29).[2]

Tetracyclines are taken up into bacterial cells by an active transport process. Once within the cell they bind reversibly to ribosomes and inhibit protein synthesis, and therefore impair cell growth.[1]

Which infections should tetracyclines be used for?

Tetracyclines are used to treat a variety of infections including chest, urethral, pelvic and skin infections. They are often used in combination with other antibiotics.

First-line indications for tetracyclines

Community acquired pneumonia – suspected atypical infection

Amoxicillin 500 mg – 1000 mg, three times daily, for seven days **plus** erythromycin 400 mg, four times daily (or 800 mg, twice daily), for seven days **or** roxithromycin 150 mg, twice daily (or 300 mg once daily), for seven days **or** doxycycline 100 mg, twice daily, for the first day, then 100 mg, once daily, for six days.

Table 1: Common first-line indications for tetracyclines

Infection	First-line treatment
Suspected atypical pneumonia (adult)	Amoxicillin 500 mg – 1000 mg, three times daily, for seven days plus erythromycin, roxithromycin or **doxycycline**.
Pelvic inflammatory disease	Ceftriaxone 250 mg IM stat and **Doxycycline** 100 mg, twice daily, for two weeks and Metronidazole 400 mg, twice daily, for two weeks
Epididiymo-orchitis (if STI pathogens suspected, usually males < 35 years)	Ceftriaxone 250 mg, IM stat and **Doxycycline** 100 mg, twice daily, for at least two weeks
Acne (moderate severity)	**Doxycycline** 100 mg, daily (or alternate days), for four to six months
Rosacea (moderate severity)	**Doxycycline** 50 mg, daily, for six to twelve weeks.
Specific infections including **Q fever, Lyme disease and anthrax exposure**.	These infections occur rarely in New Zealand. Consider in people who have recently travelled from an endemic area and in those who have handled animals such as sheep and goats or been bitten by ticks.

For further information see "Antibiotic choices for common infections", bpac^{nz} (Apr, 2011).

Amoxicillin is the first-line treatment for community-acquired pneumonia. Erythromycin, roxithromycin or doxycycline should be added to the treatment regimen for suspected atypical infections, or if the patient has not improved within 24 – 48 hours. This is to cover atypical respiratory pathogens including *C. pneumoniae*, *M. pneumoniae*, *C. psittaci*, and *Legionella pneumophila*.

Due to the high rate of tetracycline resistance among *S. pneumoniae* and *Haemophilus influenzae*, doxycycline is not used first-line as monotherapy for community acquired pneumonia unless the patient is allergic to penicillin (erythromycin or roxithromycin are also alternatives to amoxicillin).[3]

For further information see: "The management of community-acquired pneumonia", BPJ 45 (Aug, 2012)

Pelvic inflammatory disease

Ceftriaxone 250 mg IM stat, **plus** doxycycline 100 mg twice daily, for two weeks, **plus** metronidazole 400 mg twice daily, for two weeks

Pelvic inflammatory disease is a polymicrobial infection, which, if left untreated, can result in serious consequences such as ectopic pregnancy and infertility. Testing should be carried out for chlamydia, gonorrhoea and trichomonas and consider a pregnancy test, full blood count and CRP. A broad-spectrum treatment regimen is indicated to cover *Neisseria gonorrhoea*, *Chlamydia trachomatis* and anaerobes.

Azithromycin 1 g stat, repeated in seven days (i.e. two doses in total) may be used instead of doxycycline in this treatment regimen, if chlamydia is present and compliance is an issue.[4]

N.B. Ceftriaxone is subsidised if prescribed for the treatment of confirmed ciprofloxacin-resistant gonorrhoea, and the prescription or MPSO is endorsed accordingly

Epididiymo-orchitis

Ceftriaxone 250 mg IM stat **plus** doxycycline 100 mg, twice daily, for at least two weeks

Epididymo-orchitis is an inflammation of the epididymis and/or testis. It is usually due to infection, most commonly from a urine tract or sexually transmitted infection. Doxycycline is included in the antibiotic regimen when sexually transmitted pathogens are suspected. Most guidelines recommend this regimen in males aged less than 35 years.[5] Tests for *Chlamydia trachomatis* and *Neisseria gonorrhoea* should also be requested.

If urinary tract pathogens are suspected (e.g. in males aged over 35 years), an appropriate treatment regimen is amoxicillin + clavulanic acid 500/125 mg, three times daily, for two to three weeks or ciprofloxacin 500 mg, twice daily, for 10 – 14 days.[5]

Acne vulgaris – moderate severity

Doxycycline 100 mg, daily, for four to six months

Oral antibiotics may be trialled in people aged over 12 years with moderate acne and when acne has not improved with topical treatment alone. Tetracycline antibiotics such as doxycycline are usually the first-line choice. They inhibit the growth of *Propionibacterium acnes* and also have a direct anti-inflammatory effect.

It is recommended that doxycycline is used in combination with a topical retinoid or benzoyl peroxide. It can be prescribed for four to six months and tapered, e.g. used alternate days, and discontinued once acne improves.

Minocycline (partly subsidised) may be considered second-line, but it is associated with rare adverse effects such as blue-gray pigmentation of the skin, hepatic dysfunction and, in very rare cases, systemic lupus erythematosus (Page 20).[2] If minocycline is used for longer than six months, liver function tests should be requested every three months.

Tetracyclines should not be used with oral retinoids, e.g. isotretinoin, as this may increase the risk of idiopathic intracranial hypertension (Page 20).[2]

For further information see: "How to treat acne", BPJ 20 (Apr, 2009)

Rosacea – moderate severity

Doxycycline 50 mg, once or twice daily, for six to 12 weeks

N.B. Doxycycline 50 mg tablets are subject to a part-charge

Rosacea is a chronic facial rash, which most commonly affects people with fair skin, aged 30 – 60 years. It may be transient, recurrent or persistent, and may be aggravated by facial creams or oils, topical steroids and by alcohol.[6]

For mild cases of rosacea, metronidazole cream or gel is first-line treatment, used intermittently or long-term. For more severe cases, topical metronidazole may be used in combination with oral antibiotics.[6] Azelaic acid cream or lotion is an alternative to topical metronidazole.[6]

Tetracycline antibiotics reduce inflammation, papules, pustules and eye symptoms of rosacea, therefore doxycycline is the first-line oral antibiotic choice. The dose and duration of treatment is dependent on the severity of the symptoms. Repeat courses are often required as antibiotics suppress the symptoms, rather than cure them.[6] Low dose, oral isotretinoin is an alternative to doxycycline if it is ineffective or not tolerated.[6] N.B. Oral isotretinoin and doxycycline should not be used concurrently (Page 21).[2]

Second-line indications for tetracyclines

Tetracyclines may be used as a second-line alternative in the treatment of many infections (Table 2). However, emergence of bacterial resistance and the development of other antibacterials have limited their use in recent years.

Malaria prophylaxis

Doxycycline is commonly prescribed to travellers for malaria prophylaxis, depending on susceptibility in the region(s) visited. Doxycycline 100 mg daily (2 mg/kg per day in children aged over 12 years, weighing less than 50 kg) should be commenced two days before entering and continued until four weeks after leaving the malarial area.

WHO publishes annually updated information on malaria, its geographical distribution and recommended preventive measures: **www.who.int/malaria/travellers/en**

For further information see: "Providing medical advice to travellers", BPJ 41 (Dec, 2011)

Table 2: Common second-line indications for tetracyclines

	First Line treatment	Second line treatment
Chlamydia	Azithromycin 1 g stat	**Doxycycline** 100 mg, twice daily, for seven days
Acute non-specific urethritis	Azithromycin 1 g stat If purulent discharge, treat as for gonorrhoea, i.e. ceftriaxone 250 mg IM stat and azithromycin 1g stat	**Doxycycline** 100 mg, twice daily, for seven days
Acute exacerbation of chronic bronchitis or COPD	Amoxicillin 500 mg, three times daily, for five days	**Doxycycline** 100 mg, twice daily, for five days
Acute sinusitis (only if antibiotic indicated)	Amoxicillin 500 mg, three times daily, for seven days	**Doxycycline** , co-trimoxazole or cefaclor
Bites and clenched fist infections	Amoxicillin clavulanate 500/125 mg, three times daily, for five to ten days	Metronidazole plus either **doxycycline** or co-trimoxazole

For further information see:

"Antibiotic choices for common infections", bpac^{nz} (Apr, 2011).

"Treatment of sexually transmitted and other genital infections", BPJ 20 (Apr, 2009).

Prescribing notes for tetracyclines

Advice to patients taking doxycycline

- Take with a full glass of water and avoid lying down for at least one hour to reduce risk of oesophageal irritation
- Take with food to reduce gastrointestinal adverse effects
- Avoid sun exposure, wear protective clothing and use sunscreen due to the risk of photosensitivity
- Do not take antacids, iron, calcium or zinc supplements within two hours of taking doxycycline as they may decrease effectiveness

Adverse effects associated with tetracyclines

Tetracyclines are generally safe and their most common adverse effects relate to gastrointestinal symptoms.

Gastrointestinal adverse effects are common, especially with high doses, and are mostly attributed to irritation of the mucosa. They include nausea, vomiting and diarrhoea. Oesophageal ulceration has been reported, particularly after a dose is taken with insufficient water or at bedtime.[1]

Photosensitivity may occur, depending on the tetracycline dose and degree of sun exposure. Symptoms include exaggerated sunburn and itching within minutes to hours of sun exposure. In most cases, symptoms resolve within days (provided no further sun exposure occurs). In severe cases, vesicles and bullae may develop, resulting in hyperpigmentation of the skin, which may take weeks to months to resolve. Studies have demonstrated phototoxic reaction rates of 3%, 20% and 42%, for doses of 100, 150 and 200 mg of doxycycline respectively.[7] The use of broad-spectrum sunscreen (UV-A + UV-B) can minimise or prevent the potential effects of a phototoxic reaction.

Autoimmune adverse effects are very rarely associated with minocycline, such as systemic lupus erythematosus, autoimmune hepatitis, serum sickness and vasculitis, with or without the development of antinuclear antibodies or other autoantibodies. Symptoms may be expressed as fever, malaise, loss of appetite, rash, arthralgia or myalgia. Most cases occur in young females being treated for acne (i.e. long-term use).[1] Minocycline is also very rarely associated with blue-grey pigmentation of the skin, which may be irreversible.

Idiopathic intracranial hypertension with headache, dizziness, visual disturbances and papilloedema has been reported in people using tetracyclines long-term. Symptoms can develop from within two weeks to one year or more of starting a tetracycline.[1]

Renal and hepatic impairment

Both doxycycline and minocycline can be used with caution in people with renal impairment, however, high doses should be avoided.[2] All tetracyclines should be avoided or used with caution in patients with hepatic impairment.[2]

Avoid tetracyclines in children

Tetracyclines are contraindicated in children aged under 12 years, as they are associated with impaired bone growth and permanent discoloration of teeth and enamel hypoplasia.[2] This is because tetracyclines bind to calcium molecules and are deposited in calcifying areas in bone, nails and teeth.[1]

Avoid tetracyclines during pregnancy and breast feeding

Tetracyclines are contraindicated in women who are pregnant or breast feeding.[2]

Doxycycline and minocycline are classified as pregnancy category D.* Effects on skeletal development of the embryo during the first-trimester have been documented in animal studies. Administration during the second or third trimester may cause discoloration of the child's teeth.[2] Large parenteral doses of tetracyclines have been associated with acute fatty necrosis of the liver in pregnant women, especially those with pyelonephritis.[2, 8]

Chelation with calcium in breast milk is likely to reduce the adverse effects (i.e. tooth discoloration) of tetracyclines in an infant who is breast feeding, however, they still should not be used in women who are breast feeding.[2]

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* **Category D**: Drugs that have caused, are suspected to have caused or may be expected to cause, an increased incidence of human foetal malformations or irreversible damage. These drugs may also have adverse pharmacological effects. Specialised texts should be consulted for further details.

Common medicine interactions with tetracyclines[2]

Interacts with:	Notes:
Isotretinoin	**Avoid:** Increases risk of intracranial hypertension
Acitretin	**Avoid:** Increases risk of intracranial hypertension
Antacids containing aluminum or magnesium	**Adjust:** reduces efficacy of tetracyclines; separate antacid and tetracycline doses by two to three hours (or use an H2-receptor antagonist)
Calcium	**Adjust:** reduces efficacy of tetracyclines; separate calcium and tetracycline doses by two to three hours
Iron	**Adjust:** reduces absorption of tetracyclines and may reduce absorption of iron; give iron three hours before or two to three hours after tetracycline
Zinc (oral)	**Adjust:** reduces absorption of tetracyclines; give zinc three hours before or two to three hours after tetracycline
Quinapril	**Adjust:** reduces absorption of tetracyclines (effect may be less with doxycyline); separate quinapril and tetracycline doses by two to three hours
Carbamazepine	**Adjust:** reduced serum levels of doxycycline in patients using carbemazepine long-term; consider doubling dose of doxycycline
Phenytoin	**Adjust:** reduced serum levels of doxycycline in patients using phenytoin long-term; consider doubling dose of doxycycline
Warfarin	**Monitor:** may increase effect of warfarin, monitor coagulation within three days of commencing tetracycline

For further information see New Zealand Formulary. Available from: **www.nzf.org.nz**

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References

1. Sweetman S, editor. The complete drug reference. London: Pharmaceutical Press, 2011.
2. New Zealand Formulary (NZF). NZF v4. NZF, 2012. Available from: www.nzf.org.nz (Accessed Oct, 2012).
3. Grayson M. Kucers' the use of antibiotics. 6th ed. Credo; 2010. Available from: www.medicinescomplete.com (Accessed Sep, 2012).
4. New Zealand Sexual Health Society (NZSHS). Pelvic inflammatory disease (PID). Treatment guidelines. NZSHS, 2012. Available from: www.nzshs.org (Accessed Sep, 2012).
5. New Zealand Sexual Health Society (NZSHS). Epidymo-orchitis. Treatment guidelines. NZSHS, 2009. Available from: www.nzshs.org (Accessed Sep, 2012).
6. DermNet NZ. Rosacea. New Zealand Dermatological Society Inc, 2012. Available from: www.dermnetnz.org (Accessed Sep, 2012).
7. Drucker AM, Rosen CF. Drug-Induced Photosensitivity: Culprit Drugs, Management and Prevention. Drug Saf 2011;34(10):821-37.
8. Mylan New Zealand Ltd. Doxine. Medicine datasheet. 2012. Available from: www.medsafe.govt.nz (Accessed Sep, 2012).

Managing breathlessness in palliative care

Breathlessness is a complex and subjective symptom that is common among people with a terminal illness. Once reversible causes have been exhausted, management of breathlessness requires a flexible approach, however, low-dose morphine is the first-line pharmacological treatment. Patient anxiety, preferences and individual circumstances are also likely to affect management decisions.

Breathlessness – complex for clinicians, frightening for patients

Breathlessness (dyspnoea), is one of the most common symptoms experienced by people who are nearing the end of life.[1] It is reported as being the most debilitating symptom by 95% of people with Chronic Obstructive Pulmonary Disease (COPD), and is also common in patients with lung fibrosis, heart failure and terminal cancer.[2]

Breathlessness is caused by a complex interaction of signals from the chest walls, lungs, upper airways and central nervous system. Activity, anxiety level and previous experiences all influence a patient's perception of breathlessness. While there is often an obvious reason, for some people no cause is found despite thorough assessment. Breathlessness is described by patients as "hard work", "painful", "frightening" or a "continuous fight".[3]

The initial aims of management are to reduce the level of breathlessness and treat any underlying conditions that may be aggravating the breathlessness. For example, furosemide can be given by several routes, including subcutaneously, to reduce fluid overload occurring secondary to congestive heart failure or dexamethasone can be given for partial obstruction of the airways. Referral for drainage of pleural effusions or abdominal ascites may also be considered where appropriate.[8] However, once all reversible causes have been addressed and pharmacological treatments are maximal, breathlessness, if still present, is considered to be refractory.

Goals of care when treating refractory breathlessness

Once breathlessness is refractory, the goal of treatment becomes symptom relief. Treatment should begin with non-pharmacological interventions, then consider low-dose morphine, and if appropriate, benzodiazepines (if anxiety is a factor) or oxygen treatment (if hypoxaemic). However, clinicians should individualise their approach, as treatment effectiveness varies between patients, and other interventions may be trialled on an individual basis.

Non-pharmacological treatments of breathlessness

Non-pharmacological treatments should be considered first-line for treating breathlessness in people with a terminal condition. The acceptability and effectiveness of treatments will depend on individual patient circumstances and preference.

A Cochrane review found evidence supporting the use of physiotherapy in the treatment of breathlessness, e.g. pursed-lip breathing training where the respiratory rate is decreased and vital capacity increased, as well as evidence for walking aids for patients who are mobile.[4] However, the number of studies was insufficient to conduct a meta-analysis. Electrical muscle stimulation was also shown to be effective, although this is not widely available.[4] A small study of 50 patients found that a handheld fan directed at the face reduced the sensation of breathlessness.[5]

Cognitive behavioural approaches, where available, can provide some relief from breathlessness.[6] This includes relaxation and modification of negative thoughts.[6]

There is no strong evidence for recommending other non-pharmacological treatments for breathlessness, however, any intervention that improves the patient's psychological state may provide some degree of symptom relief, e.g. acupuncture or music therapy. Encouraging the patient to maintain a small level of activity (if able), may also be helpful.

Strategies aimed at improving patient comfort include:[7]

- Positioning the patient to maximise their comfort, e.g. elevating the head and torso, or lying with the affected lung downwards if only one lung is dysfunctional
- Using a humidifier
- Reducing the room temperature
- Eliminating irritants such as smoke or allergens
- Opening a window to create a draft
- Providing a window view if possible
- Providing reassurance

 The Asthma Foundation's "Breathe easier with COPD" booklets provide a list of techniques and practical ways of dealing with the stress and limitations of breathlessness.

Morphine for breathlessness

Morphine is the most widely studied and extensively used medicine for the treatment of breathlessness in patients with a terminal condition.[1]

How does morphine reduce breathlessness?

The exact mechanism by which morphine alleviates breathlessness is unknown, however, opioid-induced vasodilation in pulmonary vasculature has been demonstrated in animal studies.[1] Endogenous circulatory opioids (e.g. endorphins) have been shown to improve breathlessness during exercise in people with COPD.[1]

Overcoming potential barriers to morphine use

Health professionals in primary care may be reluctant to use morphine in the management of breathlessness because of concerns about respiratory depression.[9] Opioids can relieve breathlessness by decreasing the respiratory rate without causing hypercapnia or hypoxia.[1] A study comparing the effects of morphine or hydromorphone on breathlessness in 15 opioid-naive and 12 opioid pre-treated patients, found no higher risk of respiratory depression or hypercapnia in the opioid-naive group.[10] As of 2009, there were no reported cases of respiratory depression from the use of oral, low-dose, strong opioids in patients with breathlessness.[3]

Lack of guidelines may make clinicians feel that treatment of breathlessness with morphine is more suited to a hospital or hospice setting.[9] In response, the American College of Chest Physicians released a consensus statement in 2010 recommending that opioids should be used and titrated for the relief of breathlessness in patients with progressive lung or heart disease and a limited prognosis.[11]

Patients and their family/ whānau may be concerned about their ability to administer morphine, or about the presence of narcotics in the home. Clear written and explained instructions regarding dose titration and regular phone follow-up in the first weeks of treatment may provide reassurance. Safety concerns can be addressed by discussing the maximum ten-day dispensing requirement and by suggesting patients request pharmacists use child resistant closures on tablet and liquid containers.

Initiating opioids for breathlessness

First-line pharmacological treatment is oral morphine. Initially low doses may be given on an as-needed basis, e.g. 2 mg of an oral solution pre-measured in a syringe or 2.5 mg immediate release tablet (one-quarter of a 10 mg tablet). An example of a more structured regimen is: 2 – 2.5 mg, given four to six hourly.[12] This dose can be increased in steps of 30% if tolerated.[12]

Where patients require regular dosing of morphine for breathlessness, consider switching to a sustained-release formulation. A study of 83 opioid-naive patients with breathlessness, found 10 mg of sustained-release oral morphine once daily was safe and effective for 70% of patients who responded to morphine for the treatment of breathlessness.[13] The sustained-release dose can be calculated by summing all the morphine doses given over a 24 hour period. Oral morphine solution can also be used for acute relief, as required.

If oral medication is unsuitable for the patient, then subcutaneous morphine is an alternative at a dose of 1 – 2 mg, given four to six hourly.[12] A syringe driver may also be considered, depending on the required dosing frequency. Patients who are concurrently taking regular opioids for analgesia can be given additional small doses of morphine for breathlessness, as required, e.g. using one quarter of the patient's current four-hourly breakthrough pain dose.[12]

In practice, it is unlikely that patients will receive significantly more benefit by increasing the total morphine dose for breathlessness over 20 mg in a 24 hour period.

Switching to another opioid. If morphine is unable to be used due to allergy or intolerable adverse effects, a different strong opioid can be trialled. Oxycodone can be initiated at a dose equipotent to morphine, however, there is no evidence that oxycodone is as effective as morphine for relief of breathlessness (although a major study is underway). Oxycodone is between 1.5 and two times more potent than morphine due to its increased bioavailability. For example,

if the patient was taking 10 mg oral morphine, they would require approximately 5 mg of oxycodone.

Nebulised opioids are not recommended for the treatment of breathlessness in a community setting, due to a lack of evidence supporting their efficacy.[2]

Benzodiazepines for breathlessness

There is no evidence that benzodiazepines reduce breathlessness, however, they can be very effective at reducing the anxiety associated with breathlessness,[12] and may be considered if non-pharmacological treatments and morphine have not been effective.

Lorazepam 0.5 mg (half a 1 mg tablet), every four to six hours, as required, is an appropriate starting dose.[12] Oral diazepam, 2 – 5 mg given at night is appropriate where there is continuous anxiety.[12] In some cases clonazepam, e.g. 0.5 mg at night or as required, may also be considered. Clonazepam has a relatively long half-life which can result in a cumulative effect occurring over time.

Intranasal midazolam (unsubsidised), a short-acting benzodiazepine, is rapidly absorbed and a useful alternative if administration via other routes is not appropriate for treating breathlessness that is caused by anxiety, or for calming a highly anxious patient.[14] Peak plasma concentrations occur within ten minutes.[14] The short-acting nature of this medicine means that it is less likely to accumulate in the body and the rapid speed of onset reduces the likelihood that multiple doses will be administered. The preparation is made in the pharmacy by pouring midazolam injection (15 mg/3 mL) into a nasal spray bottle.[14] It can be prescribed at one to two sprays in each nostril, per hour as required. Each spray delivers 0.5 mg of midazolam.[14]

Oxygen treatment for breathlessness

Oxygen treatment is not beneficial to most patients with breathlessness, and should only be considered for patients with established hypoxaemia ($PaO_2 \leq 55$ mmHg).[16]

Oxygen treatment in a hypoxic patient aims to avoid hypercapnia and may reduce polycythaemia, improve sleep quality and prevent right heart failure.[16] Oxygen treatment in a community setting requires authorisation from a respiratory physician.

In patients who are not significantly hypoxaemic, there is currently no evidence that oxygen reduces breathlessness.[17]

Bronchial secretions – The "death rattle"

In the last days and hours of a person's life, bronchial secretions can accumulate in the throat and cause a choking sound referred to as the "death rattle". This can cause discomfort for the patient and distress for their family and friends. First-line treatment for bronchial secretions in this circumstance is hyoscine butylbromide (buscopan), 20 mg subcutaneously given hourly as required.[12] This can be delivered via a syringe driver and is compatible with most other medicines commonly administered via this route in palliative care. Subcutaneous hyoscine hydrobromide 400 mcg up to every two hours or glycopyrronium (not subsidised) 200 mcg up to every four hours are other treatment options.[12, 15]

N.B. The reason these medicines are used are because of their anticholinergic properties, which dry secretions. However, this also causes the discomfort of a dry mouth therefore the benefits need to be weighed with the adverse effects.

An article on using syringe drivers in palliative care will appear in the next edition of Best Practice Journal.

A blinded study of 239 patients randomly assigned to either domiciliary oxygen or room air via a nasal cannulae for 16 hours per day, found small improvements in breathlessness for both treatments, but no difference between the two.[18] This suggests that the movement of air across the face alone may provide symptom relief. A Cochrane review found no overall improvement in breathlessness among patients with cancer using oxygen, however, some individuals felt better breathing oxygen.[19]

Patient attitudes may vary towards long-term oxygen treatment, as it is restrictive and impacts on patient and carer routines. Some patients may also be concerned about the unhealthy appearance that cylinders and masks present to family, or dislike the technical focus on the machines and oxygen readings.[3] Other patients may find reassurance in the potential 'life-line' of oxygen supply.

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Advanced Care Planning – communicating with the patient before hospital admission

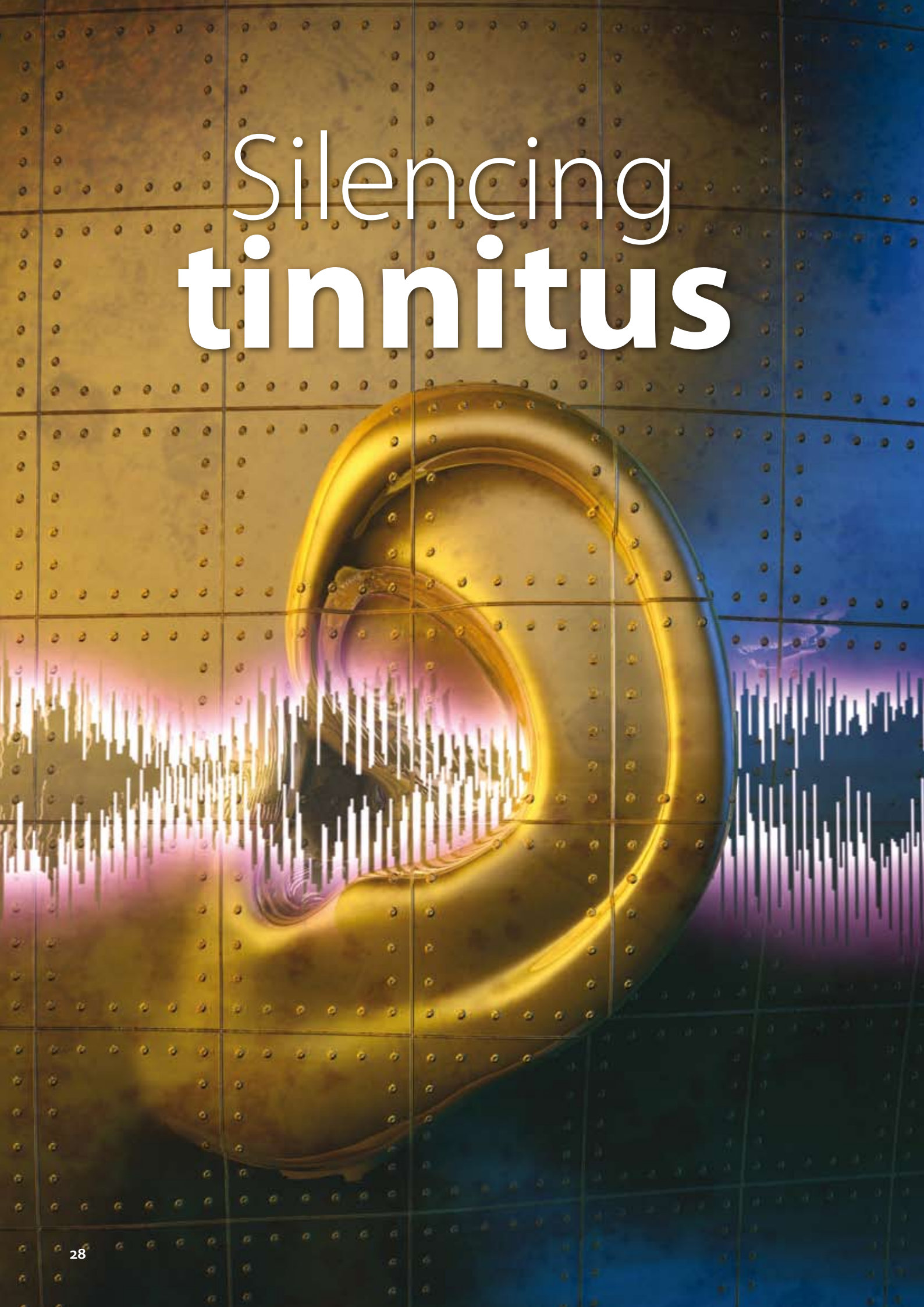
Despite the best efforts of carers and clinicians in the community, patients with terminal conditions often require hospital or hospice admission in the final days of their life. Admitting staff may have had no previous contact with the patient and may be unaware of the patient's wishes. Health professionals in primary care, who have a long-standing relationship with the patient, can enable continuity of care by initiating Advanced Care Planning. This involves proactive discussions about the expected course of the patient's disease and its prognosis, including the patient's preferences for care at the end of life – both medical and spiritual. A folder containing information about the patient's medicines, contacts, decisions and wishes can then be left with the patient. This allows admitting staff to ensure that the treatment and care they provide are consistent with the patient's values and goals.

"A Good Death" is a thirty minute documentary, produced by Professor Robin Taylor and Dr Paul Trotman, following a patient with COPD, and his family, during the last months of his life. The film focuses on end of life care and Advanced Care Planning, and is available at: **www.agooddeath.co.nz**

References

1. Kamal AH, Maguire JM, Wheeler JL, et al. Dyspnea review for the palliative care professional: treatment goals and therapeutic options. J Palliat Med 2012;15(1):106–14.
2. Uronis HE, Currow DC, Abernethy AP. Palliative management of refractory dyspnea in COPD. Int J Chron Obstruct Pulmon Dis 2006;1(3):289–304.
3. Rocker G, Horton R, Currow D, et al. Palliation of dyspnoea in advanced COPD: revisiting a role for opioids. Thorax 2009;64(10):910–5.
4. Bausewein C, Booth S, Gysels M, Higginson I. Non-pharmacological interventions for breathlessness in advanced stages of malignant and non-malignant diseases. Cochrane Database Syst Rev 2008;(2):CD005623.
5. Galbraith S, Fagan P, Perkins P, et al. Does the use of a handheld fan improve chronic dyspnea? A randomized, controlled, crossover trial. J Pain Symptom Manag 2010;39(5):831–8.
6. Howard C, Dupont S, Haselden B, et al. The effectiveness of a group cognitive-behavioural breathlessness intervention on health status, mood and hospital admissions in elderly patients with chronic obstructive pulmonary disease. Psychol Health Med 2010;15(4):371–85.
7. Tyler L. Dyspnea in palliative care patients. J Pharmaceut Care Pain Symptom Contr 2000;7(4):109–27.
8. Macleod A, MacLeod R, Vella-Brincat J. The palliative care handbook. 5th ed. NZIJ Hospice Charitable Trust; 2011.
9. Young J, Donahue M, Farquhar M, et al. Using opioids to treat dyspnea in advanced COPD: attitudes and experiences of family physicians and respiratory therapists. Can Fam Physician 2012;58(7):e401–7.
10. Clemens KE, Quednau I, Klaschik E. Is there a higher risk of respiratory depression in opioid-naïve palliative care patients during symptomatic therapy of dyspnea with strong opioids? J Palliat Med 2008;11(2):204–16.
11. Mahler DA, Selecky PA, Harrod CG, et al. American College of Chest Physicians consensus statement on the management of dyspnea in patients with advanced lung or heart disease. Chest 2010;137(3):674–91.
12. National Health Service (NHS) Lothian. Breathlessness in palliative care. NHS, UK 2010. Available from: http://gp-palliativecare.co.uk/files/lothia_guideline_breathlessness.pdf (Accessed Oct, 2012).
13. Currow DC, McDonald C, Oaten S, et al. Once-daily opioids for chronic dyspnea: a dose increment and pharmacovigilance study. J Pain Symptom Manag 2011;42(3):388–99.
14. Canterbury DHB (CDHB). Intranasal midazolam in palliative care patients. CDHB, 2011. Available from: http://palcare.streamliners.co.nz/ (Accessed Oct, 2012).
15. New Zealand Formulary (NZF). NZF v4. NZF; 2012. Available from: www.nzf.org.nz (Accessed Oct, 2012).
16. McDonald CF, Crockett AJ, Young IH. Adult domiciliary oxygen therapy. Position statement of the Thoracic Society of Australia and New Zealand. Med J Aust 2005;182(12):621–6.
17. Clark AL, Johnson MJ, Squire I. Does home oxygen benefit people with chronic heart failure? BMJ 2011;342:d234.
18. Abernethy AP, McDonald CF, Frith PA, et al. Effect of palliative oxygen versus room air in relief of breathlessness in patients with refractory dyspnoea: a double-blind, randomised controlled trial. Lancet 2010;376(9743):784–93.
19. Cranston JM, Crockett A, Currow D. Oxygen therapy for dyspnoea in adults. Cochrane Database Syst Rev 2008;(3):CD004769.

Silencing **tinnitus**



Tinnitus is a common, but frustrating problem for both patients and clinicians. In the majority of people tinnitus is a subjective, neurophysiological problem. However, it is important to identify people who are hearing genuine sounds generated within the body (objective tinnitus) or have some other underlying treatable condition. Subjective tinnitus arises within the brain, but may be triggered by and/or predisposed to, changes in the ear, in any part of the auditory pathway, by other related structures and sensory pathways, by emotional factors and by conditions that have an indirect effect such as metabolic disorders. There is no cure for subjective tinnitus and no one effective treatment. Most treatments aim to help patients habituate to tinnitus.

What is tinnitus?

Tinnitus is the perception of sound within the ear or head, without a corresponding external stimulus for that sound. Tinnitus may be described as a buzzing, ringing, roaring, clicking, booming, hissing, whistling or cicada-like noise. It can be heard as a single sound or as a complex mix of different sounds. It can be unilateral or bilateral, of sudden or gradual onset, constant or intermittent, and with fluctuating pitch and intensity. Its characteristics tend to reflect associated pathology and hearing loss. Approximately 40% of people with tinnitus are also intolerant to the loudness of everyday sounds.[1] When this intolerance is severe it is referred to as hyperacusis.

Tinnitus can be subjective (approximately 95% of cases) or, less commonly, objective (approximately 5% of cases).[2] Subjective tinnitus can arise because of problems in any part of the ear and auditory pathways. It may also arise or be modulated because of problems in the skin and muscles of the head and neck and their sensory pathways.

Objective tinnitus is generated by musculoskeletal and vascular structures that are in close proximity to the cochlea. The "somata-sounds" (body noises) produced by these structures are often vibratory or pulsatile and perceived by patients as originating within the ear – in some people they are (see "Objective vs. subjective tinnitus". page 31).

Clinically significant tinnitus can be defined as noises in the ear or head lasting for more than five minutes and occurring more than once per week.[3] An alternative definition is: "tinnitus that bothers people, affects their life and causes them to frequently seek professional help".[4]

Transient tinnitus (i.e. lasting for less than five minutes) is a common occurrence after exposure to loud sounds such as an explosion or after a music concert. It may also be associated with transient hearing loss and is significant because of the risk of permanent damage.

The incidence of tinnitus

It has been estimated that at least 15% of adults will experience tinnitus during their lifetime.[3, 6] For the majority of people, symptoms are mild and brief. However, in approximately 1 – 2% of people (some studies report as much as 5%), tinnitus is severe, on-going and causes significant distress.[5, 6]

Risk factors for tinnitus include:

- Increasing age – peak incidence is in people in their 60's, after which the incidence of distressing tinnitus declines again, although tinnitus can occur at any age[7]
- Hearing loss – approximately 80% of people with tinnitus have some degree of hearing loss, although the prevalence varies with age (67% of those aged 16 – 48 years, and up to 86% of those aged 64 – 95 years).[6] There is little correlation between the degree of hearing loss and the severity of the tinnitus.

- Exposure to loud noise – occupational and recreational noise exposure is associated with the development of tinnitus and is often accompanied by noise-induced hearing loss.[7] Exposure to loud noise causes injury to the cochlea, in particular the outer hair cells which may be permanently damaged.[8]
- Smoking and hypertension – there is some evidence that vascular disease may be a factor in the development of tinnitus[7]

The majority of studies report that more **males** are affected by tinnitus than females.[5, 7]

There are well established associations between tinnitus and **mental health disorders**, such as depression and anxiety.[2] People with tinnitus frequently report sleep disturbance, decreased productivity at work, increased stress levels, inability to concentrate and feelings of annoyance or frustration, and that these factors contribute further to their psychological distress.[2, 7, 9] There is on-going debate in the literature as to the extent to which tinnitus causes anxiety and depression or if it is more common in people with these conditions.[5]

It has been proposed that the tendency for an individual to be aware of tinnitus and for it to be persistent may be influenced by their **personality type**. People who experience tinnitus may tend to be less social, less self-controlled and more negatively emotional than people without tinnitus.[10] Parallels are often drawn between people with tinnitus and those with chronic pain.[11, 12] There are similarities in the physiological and psychological factors that initiate and perpetuate these complex conditions and also similarities in the impact that the conditions have on the life of people with them.[12]

Management of tinnitus begins at the first consultation

Tinnitus is a symptom, not a specific diagnosis. In the majority of people, no specific pathological cause is found and tinnitus is therefore considered to be subjective and neurophysiological (idiopathic).[3, 5] However, there are multiple conditions which contribute to the presence of tinnitus (Table 1) and it is important to identify them.

The aim of the history and clinical examination is not only to determine if there is an identifiable cause for the tinnitus, but also to "set the scene" for ongoing management. Reassurance and support are important to avoid inadvertently increasing anxiety, fear or anger.[11, 15] Making negative statements to patients such as "there is nothing that can be done" or "you have to learn to live with it" are untrue and may result in an increased focus on tinnitus and therefore an exacerbation of the distress that it causes.[3,11] Tinnitus can interfere with a person's ability to perform at work and cause significant difficulties with family and social interactions. The consequences of tinnitus can therefore be very distressing for patients and may include feelings of frustration, a sense of isolation, low mood or depression and in severe situations suicidal thoughts.[11]

Table 1: Possible causes of tinnitus[13, 14]

Conditions that cause tinnitus by affecting the middle or inner ear and its function	Conditions that cause tinnitus by a direct or indirect effect on the auditory pathway	Other causes of objective tinnitus
■ Wax build-up in the ear canal ■ Chronic otitis externa ■ Chronic otitis media ■ Perforation of the tympanic membrane ■ Otosclerosis ■ Ménière's disease ■ Noise ■ Trauma to the ear ■ Medicines such as aminoglycoside antibiotics, loop diuretics and aspirin in excess	■ Multiple sclerosis ■ Cerebellopontine angle tumour (acoustic neuroma) ■ Meningioma ■ Intracranial pathology, e.g. stroke or tumour ■ Trauma to the head or neck ■ Temporomandibular joint disorders ■ Hypertension ■ Hypo- or hyperthyroid disease ■ Hyperlipidaemia ■ Hyperinsulinaemia and diabetes ■ Vitamin B12 deficiency	■ Vascular – an arterial bruit, arteriovenous malformation, vascular tumour ■ Neurological – spasm of the stapedial muscle, palatomyoclonus ■ A patulous (patent) eustachian tube

When taking a history:

Ask the patient to describe the **characteristics of the tinnitus**. Is it pulsatile? Unilateral or bilateral? Constant or intermittent? And if intermittent, how often and for how long does it persist? Pulsatile tinnitus is usually indicative of objective tinnitus. If tinnitus is unilateral it is more likely to be caused by underlying pathology, and conversely, if it is bilateral it is more likely to be benign in origin. If the tinnitus is of shorter duration (months rather than years) there is a higher likelihood that it will improve over time. It should also be distinguished from an auditory hallucination, secondary to a psychotic disorder.

Ask if there are any **associated symptoms** including deafness, dizziness, vertigo, hyperacusis (intolerance of loud noises), a blocked sensation in the ear, otalgia or otorrhoea. Associated symptoms may point towards an underlying cause such as otalgia due to otitis media, episodic vertigo and deafness due to Ménière's disease, or unilateral sensorineural hearing loss and tinnitus in a patient with a cerebellopontine angle tumour.

Ask the patient if they are aware of **triggers** for the tinnitus. Is there a history of excessive noise exposure (occupational or recreational), a head or ear injury or an increase in stress?

Ask about the **impact on daily life**. How troublesome do they consider it to be? When did the tinnitus start to become annoying to them?

Ask if the tinnitus is disturbing the patient's **sleep or mood**. Does the patient have a history of psychological problems?

Consider a **review of medicines** – have there been any new medicines started, including any over-the-counter medicines? Are there existing medicines that could be implicated, e.g. excessive aspirin use? Has the patient been given any ototoxic medicines such as gentamicin during a hospital admission? (see: "Medicines that may cause tinnitus", over page).

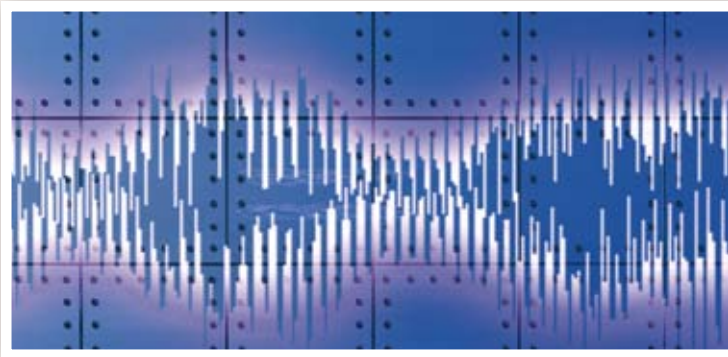
Consider the **age** of the patient – an older patient is more likely to have hearing impairment as the hair cells in the cochlea degenerate with age. This results in a decreased ability to hear higher pitched sounds. There is also some evidence that the distress associated with tinnitus is likely to be worse if it begins at an older age due to a reduction in neuroplasticity.[6]

Is there a **family history of hearing loss**?

Subjective vs. objective tinnitus

Subjective tinnitus is the most commonly experienced form of tinnitus. The tinnitus is only heard by the patient and is usually described as having a more continuous tone rather than being pulsatile. It has no acoustic source. Within the brain the primary auditory centres are normally receptive only to neural activity generated by external sound and transmitted from the inner ear through the classical auditory pathway. If the primary auditory centres become aware of other neural activity, this is interpreted as noise and the patient perceives it as tinnitus. Hearing impairment reduces the influence of the classical auditory pathway. Neural activity which is normally suppressed is processed unconsciously, reinforced by negative emotional influences, detected by the primary hearing centres and interpreted as tinnitus. In the presence of negative emotional associations, a positive feedback loop is generated and there is increased perception of the signal which becomes subjectively louder, more intrusive, more annoying and persistent.[5]

Objective tinnitus is produced by an internal acoustic stimulus from a physiological (often referred to as somatic) source which can include both auditory and non-auditory structures. The eustachian tube may produce an audible click as it opens and closes. Muscles within the middle ear or of the soft palate may fasciculate. Arterial pulsation or a venous hum may be generated by vascular tumours or abnormalities in or close to the ear. Objective tinnitus may be audible on examination using a stethoscope placed on the head in sites around the ear or over the carotid arteries. Obstructing wax in the ear canal and other causes of conductive hearing loss may make any of these somato-sounds more audible to the patient.



Medicines that may cause tinnitus

Medicine group	Effect
Aspirin, NSAIDs and quinine	Can cause reversible, multifactorial impairment of hair cell function
Loop diuretics, e.g. fruosemide	Can cause reversible and dose dependent changes in electrolyte balance in the cells of the inner ear
Aminoglycoside and macrolide antibiotics, e.g. neomycin, framycetin, gentamicin and erythromycin	Irreversibly ototoxic to the hair cells of the cochlea and vestibular system, including after excessive topical application (e.g. with the use of ear drops that contain these antibiotics – sofradex, soframycin, kenacomb)
Antimitotic drugs, e.g. methotrexate, cisplatin, vincristine	Ototoxic – can cause tinnitus and sensorineural hearing loss, which can be permanent

On examination it is important to check the:

- **Ears** – to exclude a build up of wax, infection of the canal or middle ear, tympanic membrane perforation or the presence of a foreign body, e.g. a hearing aid battery or an insect (see: "It sounds like there is a bug in my ear!"). Tympanometry provides information about the mobility of the tympanic membrane and eustachian tube function.
- **Blood pressure** – hypertension has been associated with tinnitus
- **Neck and temporomandibular joints** – for tenderness and crepitus as musculoskeletal problems in either may produce somatosensory tinnitus
- **Auscultation** – listen for carotid bruits and also over the skull around the ears for the presence of tinnitus from non-auditory structures
- **Cranial nerves** – a focused neurological examination is required if there are symptoms to suggest an underlying neurological condition. For example, a facial palsy and reduced corneal reflex may be present in patients with cerebellopontine angle tumours.

Check for hearing loss

Initial tests to screen for hearing loss can be carried out in primary care. Test the patient using a soft whisper or rub their hair between your fingers just behind their ear. Weber and Rinné tests using a tuning fork (see: "How to perform tuning fork tests", Page 34) can help distinguish between a sensorineural (e.g. damage to cochlea or cochlear nerve) or conductive (e.g. blocked ear canal, middle ear effusion) hearing loss.

Applications are now available via some smart phones that can check for sensorineural hearing loss, e.g. "Action on hearing loss" free iPhone hearing check (Search App Store, keyword: deafness or hearing check).

Diagnosing the cause of tinnitus

An algorithm incorporating the characteristics, frequency and site of tinnitus can be helpful in determining the cause (Figure 1). In some cases, referral for further investigations such as MRI is required to make a specific diagnosis.

Manage any underlying conditions

If the cause of tinnitus is found to be an underlying condition that is treatable in primary care, e.g. wax build up, infection, appropriate management of the underlying condition should alleviate the tinnitus.

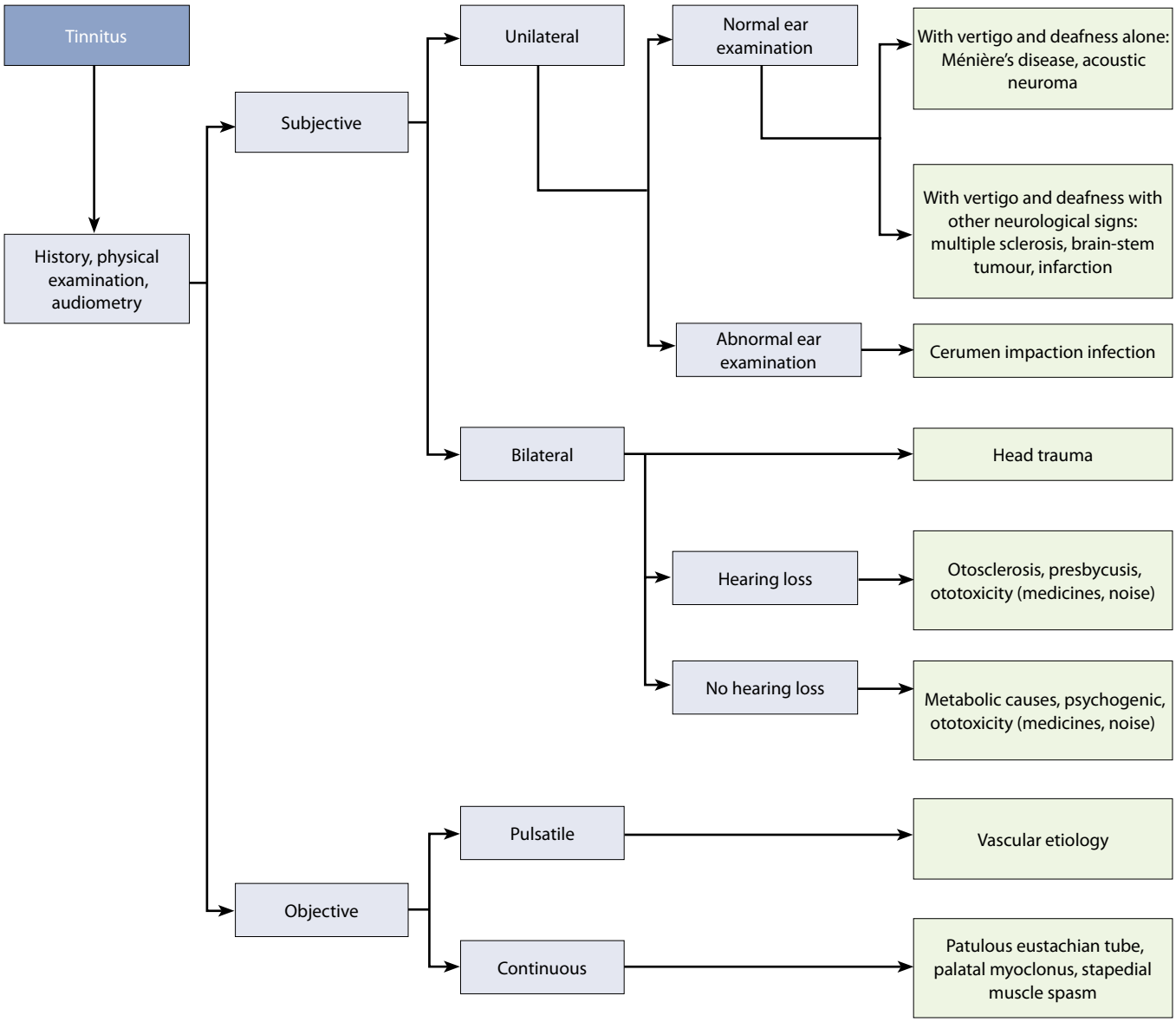


Figure 1: Diagnostic algorithm for tinnitus adapted from Crummer, 2004[13]

A referral for audiometry is recommended for all patients with clinically significant tinnitus once ear wax and infection have been excluded.[15, 16] Treatment of hearing loss with a hearing aid often improves tinnitus (Page 34).

Referral to an otolaryngologist is required if tinnitus is:[11, 17]

- Unilateral
- Pulsatile
- Rapidly progressive
- Associated with sudden hearing loss or fluctuating hearing, fullness or pressure in one or both ears, vertigo or disturbed balance
- Persistent, troublesome and intrusive

These features may indicate an otologic disease such as Ménière's disease, cholesteatoma or a cerebellopontine angle tumour.

Although a rare cause of tinnitus, if an underlying metabolic condition is suspected, laboratory testing may be indicated, e.g. TSH for hypo- or hyperthyroidism, HbA_{1c} for diabetes, FBC for anaemia or infection.

How to perform tuning fork tests

Weber test

Hit the tuning fork lightly on your knee, elbow or firm surface and hold the base on the patient's forehead. If the sound cannot be heard, gently try the bony part of the nose or the middle of the two front teeth. Ask the patient where they hear the sound the best.

In a normal test, or in a patient with a symmetrical hearing loss (e.g. in an older person with age related hearing loss) the sound is heard equally in both ears or in the midline. In an abnormal test, the sound is heard better in one ear than the other. If there is sensorineural hearing loss the sound will be louder in the normal ear. If there is conductive hearing loss the sound is heard better in the ear with the problem. One way to help remember this is to give yourself a conductive hearing loss by blocking one ear with your finger and then speaking or humming - the sound is louder on the side that is blocked, i.e. that has the conductive hearing loss.

Rinné test*

Hold the base of the vibrating tuning fork on the patient's mastoid process. When the patient can no longer hear the sound, move the tuning fork to the ear and ask if they can hear the sound again.

A normal result is when the sound is heard again – air conduction is greater than bone conduction. An abnormal result is when the sound cannot be heard again – bone conduction is greater than air conduction, i.e. there is a conductive hearing loss in that ear, particularly if the Weber test lateralises to that side as well.

In some people with severe asymmetric hearing loss, the Rinné test may appear to be normal because the unaffected opposite ear will detect the sound by air conduction. If you "mask" this ear by making a competing sound, this should be avoided.

* There are various methods

Management of subjective neurophysiological tinnitus

There is no cure and no one specific treatment that is effective for subjective, neurophysiological (idiopathic) tinnitus.[3, 5] Most treatments are aimed at trying to reduce the intensity of tinnitus or to reduce the annoyance and distress that accompanies it.[2]

Patients with bilateral tinnitus, with no impairment of hearing and who report that it is not troublesome usually do not need referral for any further investigations or treatment. Explanation and reassurance is often sufficient and referral should generally be avoided so that the patient does not focus their attention on tinnitus.[17]

Provide education and reassurance

Explain to the patient what tinnitus is and how it may be triggered and influenced by many factors, including hearing impairment, jaw and neck problems, stress, depression and other emotional associations. Aim to improve the patient's understanding of their tinnitus so that they are more able to focus on actual sounds rather than the neural activity they perceive as tinnitus. Provide suggestions for improving desirable sound stimulation, reducing aggravating stimulation from the neck and jaw and disassociating emotional factors.

Local support groups and advice can be found at:
www.tinnitus.org.nz

Review medicines and continuing noise exposure

Where appropriate, medicines which cause tinnitus can be identified and withdrawn. If there is ongoing exposure to excessive noise, advice can be given about hearing conservation and protection.

Consider referral for hearing aids in patients with hearing loss

Straining to hear causes an increase in the sensitivity of the central auditory systems and can allow tinnitus to emerge or, if already present, to worsen.[18] The correction of any associated hearing loss reduces this sensitivity and will also usually help the patient to focus on environmental sounds rather than on the tinnitus. Hearing aids are often useful in patients with tinnitus even if the hearing loss is relatively mild and would not on its own make a hearing aid appropriate.[18] Some people benefit from combination devices – hearing aids which also function as sound generators.

Habituation

Habituation is the ability of a person to become less aware of their tinnitus, and when they are aware of their tinnitus, to avoid associating it with anxiety or distress.[16] The brain can be "trained" to pay less attention to tinnitus in a similar way that people who live next to a busy road can "tune out" the noise of the traffic. It is likely that habituation is part of the natural history of tinnitus, but in some people the process of habituation fails to occur, often when there is a negative emotional significance attached to the tinnitus. This results in the person paying selective attention to tinnitus and beginning a vicious cycle. It has been said that "the difference between a person who experiences tinnitus and one who 'suffers' from it may be the person's ability to habituate to the tinnitus."[16]

Stress reduction

Tinnitus is often associated with stress although it is not always clear whether the stress has exacerbated the tinnitus or tinnitus worsened the stress. In the majority of patients, reassurance and general advice on stress reduction and management will be beneficial.

Promote good sleep hygiene

Tinnitus often disturbs sleep and the tiredness that results may then compound the problem. Encourage good sleep hygiene. Sound enrichment (see below) at night may also be helpful.

Sound enrichment

Many people with tinnitus seek quietness in an attempt to gain relief from the noise of their tinnitus. However, they should be advised to avoid environments that are too quiet because this gives them nothing to listen to except tinnitus. In many patients, particularly those who have hearing loss, quietness may increase the sensitivity of the auditory system and this worsens the tinnitus. The use of sounds that reduce the ratio of tinnitus to other environment noises can be useful and provide an alternative focus. Encourage the patient to play music, have the radio or television on in the background or even turn on a fan. The noise of a water feature or the use of relaxation tapes may be helpful as may electronic sound generators and maskers.

Distraction

Distraction with day to day activities (leisure or work) and regular exercise can be effective.

Dietary triggers

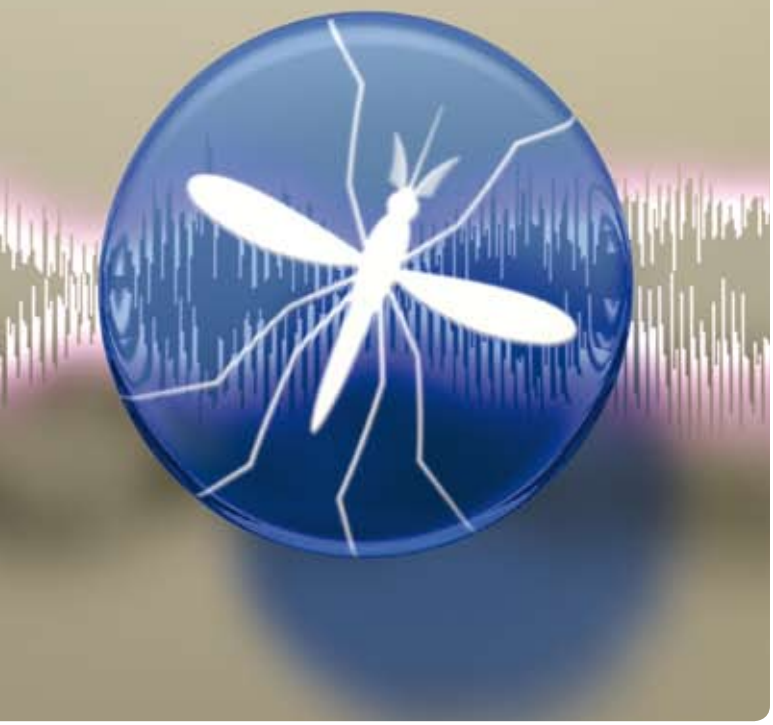
There is anecdotal evidence that for some people excess consumption of caffeine containing foods and drinks, high salt foods and quinine in tonic water may worsen tinnitus. However, there is no robust evidence on whether reducing the intake of these decreases the severity of tinnitus.

Psychological approaches

Cognitive behavioural therapy (CBT) and tinnitus retraining therapy (TRT) combine educational and psychological components, with the aim of achieving habituation by enhancing coping strategies, improving quality of life and assisting people to manage their tinnitus more effectively. Although evidence regarding the effectiveness of these approaches is limited, a recent randomised controlled trial has shown a significant improvement in quality of life for patients and a reduction in the severity of and impairment from tinnitus

"It sounds like there is a bug in my ear!"

A live insect in the ear canal may produce noises that are described in a similar way to tinnitus. A history of a sudden onset of ear noises, often accompanied by pruritus, pain or nausea may suggest the presence of a live insect. Examination of the ear will usually confirm the diagnosis. Provided there is no perforation of the tympanic membrane, irrigation is the simplest way to remove the insect, although, if available, suction removal is recommended, especially if there is suspicion of perforation. If the insect is easily seen and the patient is able to lie still, it may be possible to carefully extract the insect under direct vision with a Tilley forceps. The insect should be killed first using 2% lignocaine, a mineral oil (e.g. baby oil) or emla cream.



using an approach that combines elements of both CBT and TRT.[19] CBT is available through clinical psychologists and TRT through many audiologists.

Pharmacological treatment

The evidence to support the use of pharmacological treatments for tinnitus is inconclusive. Medicines may be appropriate if used to treat conditions associated with tinnitus, e.g. antidepressants in patients who also have anxiety or depression.[5] Both tricyclic antidepressants and SSRIs, along with medicines such as anticonvulsants (e.g. gabapentin and carbamazepine) and benzodiazepines (short-term use) may be trialled in individual patients depending on their circumstances (e.g. the presence of excessive anxiety or insomnia), however, there is anecdotal evidence that suggests that repeated attempts using unsuccessful pharmacological treatments may worsen tinnitus.[18]

Complementary treatments

A number of complementary treatments, including acupuncture, aromatherapy, hyperbaric oxygen therapy, ginkgo biloba, homeopathy and reflexology have been used to try to reduce symptoms of tinnitus. There is no robust evidence that any of these treatments is effective.[5, 20, 21]

Treatments available in secondary care

Intratympanic administration of gentamicin or steroids, or the use of intratympanic or intravenous lignocaine, may be of value for patients who have tinnitus due to Ménière's disease.

Cochlear implants have been found to reduce tinnitus in some patients who have severe or profound bilateral hearing loss.

Repetitive transcranial magnetic stimulation (rTMS) uses a device to generate a brief magnetic field which when held against the scalp produces a weak electric current in the underlying tissues.[22] The hyper-excitability in the auditory cortex associated with tinnitus is suppressed by rTMS. There is some evidence that it may be helpful in patients with moderate tinnitus that has been present for less than four years.[22]

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References

1. Jastreboff P, Jastreboff M. Tinnitus retraining therapy: a different view on tinnitus. ORL J 2006;68:23–30.
2. Langguth B. A review of tinnitus symptoms beyond 'ringing in the ears': a call to action. Curr Med Res Opin 2011;27(8):1635–43.
3. Henry J, Zaugg T, Myers P, et al. A triage guide for tinnitus. J Fam Pract 2010;59(7):389–93.
4. Snow J. Tinnitus. Philadelphia, PA: University of Pennsylvania; 2007.
5. Baldo P, Doree C, Lazzarini R, et al. Antidepressants for patients with tinnitus. Cochrane Database Syst Rev 2012;(9):CD003853.
6. Schlee W, Kleinjung T, Hiller W, et al. Does tinnitus distress depend on age? PloS ONE 2011;6(11):e27379.
7. Shargorodsky J, Curhan G, Farwell W. Prevalence and characteristics of tinnitus among US adults. Am J Med 2012;123:711–8.
8. Thorne P. Noise-induced hearing loss and tinnitus. Proceedings of 'Tinnitus Discovery': Asia-Pacific Tinnitus Symposium, 11-12 Sept 2009, Auckland, New Zealand. N Z Med J 2010;123.
9. Mazurek B, Haupt H, Olze H, Szczepek A. Stress and tinnitus - from bedside to bench and back. Front Syst Neurosci 2012;6(47):DOI:10.389.
10. Welch D, Dawes P. Personality and perception of tinnitus. Proceedings of 'Tinnitus Discovery': Asia-Pacific Tinnitus Symposium, 11-12 Sept 2009, Auckland, New Zealand. N Z Med J 2010;123.
11. Newman C, Sandridge S, Bea S, et al. Tinnitus: Patients do not have to 'just live with it'. Clev Clini J Med 2011;78(5):312–9.
12. Magnusson J. Similarities between chronic pain and tinnitus: what we've learned from chronic pain and how it applies to tinnitus. Proceedings of 'Tinnitus Discovery': Asia-Pacific Tinnitus Symposium, 11-12 Sept 2009, Auckland, New Zealand. N Z Med J 2010;123.
13. Crummer R. Diagnostic approach to tinnitus. Am Fam Physician 2004;69(1):120–6.
14. Ruppert S, Fay V. Tinnitus evalutation in primary care. Nurse Practit 2012;37(10):20–6.
15. Goodey R. Medical evaluation and management of tinnitus. Proceedings of 'Tinnitus Discovery': Asia-Pacific Tinnitus Symposium, 11-12 Sept 2009, Auckland, New Zealand. N Z Med J 2010;123.
16. Searchfield G. The management of tinnitus. N Z Fam Prac 2003;30(5):345–9.
17. Department of Health. Provision of services for adults with tinnitus: A good practice guide. United Kingdom; 2009. Available from: www.dh.org.uk (Accessed Oct, 2012).
18. British Tinnitus Association (BTA). Raising awareness in primary care: Ten top tips for GPs. BTA, UK; 2012. Available from: www.tinnitus.org.uk (Accessed Oct, 2012).
19. Cima R, Maes I, Joore M, et al. Specialised treatment based on cognitive behaviour therapy versus usual care for tinnitus: a randomised controlled trial. Lancet 2012;379(9830):1951–9.
20. Kim J, Choi J, Lee D, et al. Acupuncture for the treatment of tinnitus: a systematic review of randomized clinical trials. BMC Comp Alt Med 2012;12(79):DOI: 10.1186.
21. Hilton M, Stuart E. Ginkgo biloba for tinnitus. Cochrane Database Syst Rev 2010;(1):CD003852.
22. Stinear CM. Tinnitus management with repetitive transcranial magnetic stimulation. Proceedings of 'Tinnitus Discovery': Asia-Pacific Tinnitus Symposium, 11-12 Sept 2009, Auckland, New Zealand. N Z Med J 2010;123.

Care pathways for long-term conditions: Using type 2 diabetes as an example

A care pathway is a tool that enables practitioners to provide better health care and better patient outcomes at a lower cost. A diabetes care pathway helps guide decisions and timing for diagnosis, interventions, appropriate follow-up, escalation of treatment and referral to secondary care. This introduction to care pathways places the concept of a pathway in the context of managing long-term conditions, and highlights the difference between a care pathway and a care plan.

Over 208,000 people in New Zealand have been diagnosed with diabetes.[1] In 2011, on average 50 people were newly diagnosed with diabetes every day, with up to 100,000 more people believed to have undiagnosed diabetes.[1] Diabetes is strongly associated with ethnicity – the prevalence of type 2 diabetes is three times higher in Māori and Pacific peoples than in New Zealand Europeans.[2] Also of concern is the rapid rise in prevalence of diabetes within the South Asian population. Between 2002/3 and 2006/7 there was a four-fold increase in the number of South Asian people receiving treatment for diabetes.[3] As the number of people with diabetes continues to grow, practices must consider how they will manage their limited resources to provide care for this patient population. An evidence-based, risk adjusted approach to early detection and structuring care, i.e. a care pathway, can help to ensure that management is sustainable into the future.

What is a care pathway?

A care pathway is, at its simplest, a set of management guidelines, usually in the form of a flow chart, applied to a group of patients with the same condition. It is a tool used to improve the quality of healthcare by recommending a recognised best practice approach at a certain stage of a disease or condition. At its most complex, a care pathway can act as a fully integrated information system, guiding and monitoring a patient's journey of care between health professionals and across sectors.

Applying care pathways to long-term conditions

When care pathways are applied to long-term conditions they provide primary care clinicians guidance on:

- When to make an intervention
- Lifestyle reinforcement
- Therapeutic changes
- Checking for and monitoring complications
- Referral to other health professionals
- Intensity and content of follow-up

It is important for clinicians to have an understanding of the stages of a long-term condition (Figure 1) and how this is representative of their practice population. The type of support an individual patient requires changes as they move from one

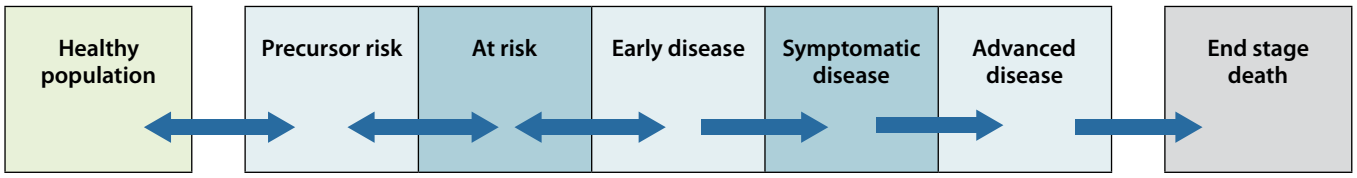


Figure 1: Stages of a long-term condition

stage to another. Those furthest along are seen more often, have lower thresholds for referral and require more intensive management. Care pathways guide the specific detailed interventions needed at each point as the patient progresses over time, from precursor risk through to advanced disease and then palliative care.

People identified as being at high risk of complications should receive more intensive intervention and follow-up, e.g. a single annual diabetes check up is not sufficient to properly manage a person with several risk factors for diabetes complications, such as poor glycaemic control, raised blood pressure and signs of kidney damage represented by an elevated urine albumin creatinine ratio.

The volume of patients at the different stages of a long-term condition is often represented as a pyramid (Figure 2). The number of patients with precursor risk and established risk factors is considerably greater (the bottom of the pyramid) than the number with multiple complications from advanced disease (top of the pyramid). When the numbers of patients at each stage are identified in a practice, the implications of how their needs are to be met and how services can be delivered become clearer. Care pathways have to take into account the type of care that can be provided by practices for the expected numbers of patients at any particular stage. For example, the greatest need is in providing support for self-management, therefore group work may be a more practical solution than providing one-to-one care. More intensive clinical care should be focused on patients who need monitoring for complications and therapeutic changes.

How effective are care pathways?

The implementation of a care pathway has been shown to reduce the variability in clinical practice, reduce healthcare costs and improve patient outcomes.[4, 5]

A Cochrane systematic review of care pathway implementation found that for every 18 people treated on a care pathway, one serious complication would be prevented. For hospital care, healthcare pathways were shown to reduce length of stay, the incidence of hospital-acquired pneumonia and the cost of care.[4]

A care pathway is different from a patient's care plan

A care pathway represents the ideal way to manage a patient population with a specific problem or long-term condition. A care plan is for an individual. The care pathway provides recommendations which should be included and enacted within a care plan. Care plans promote self-management

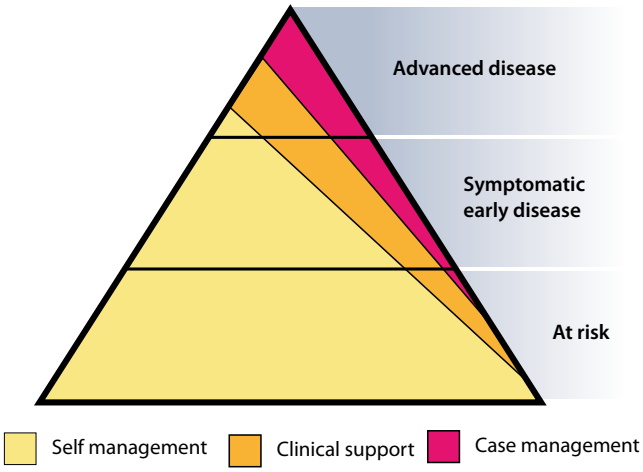


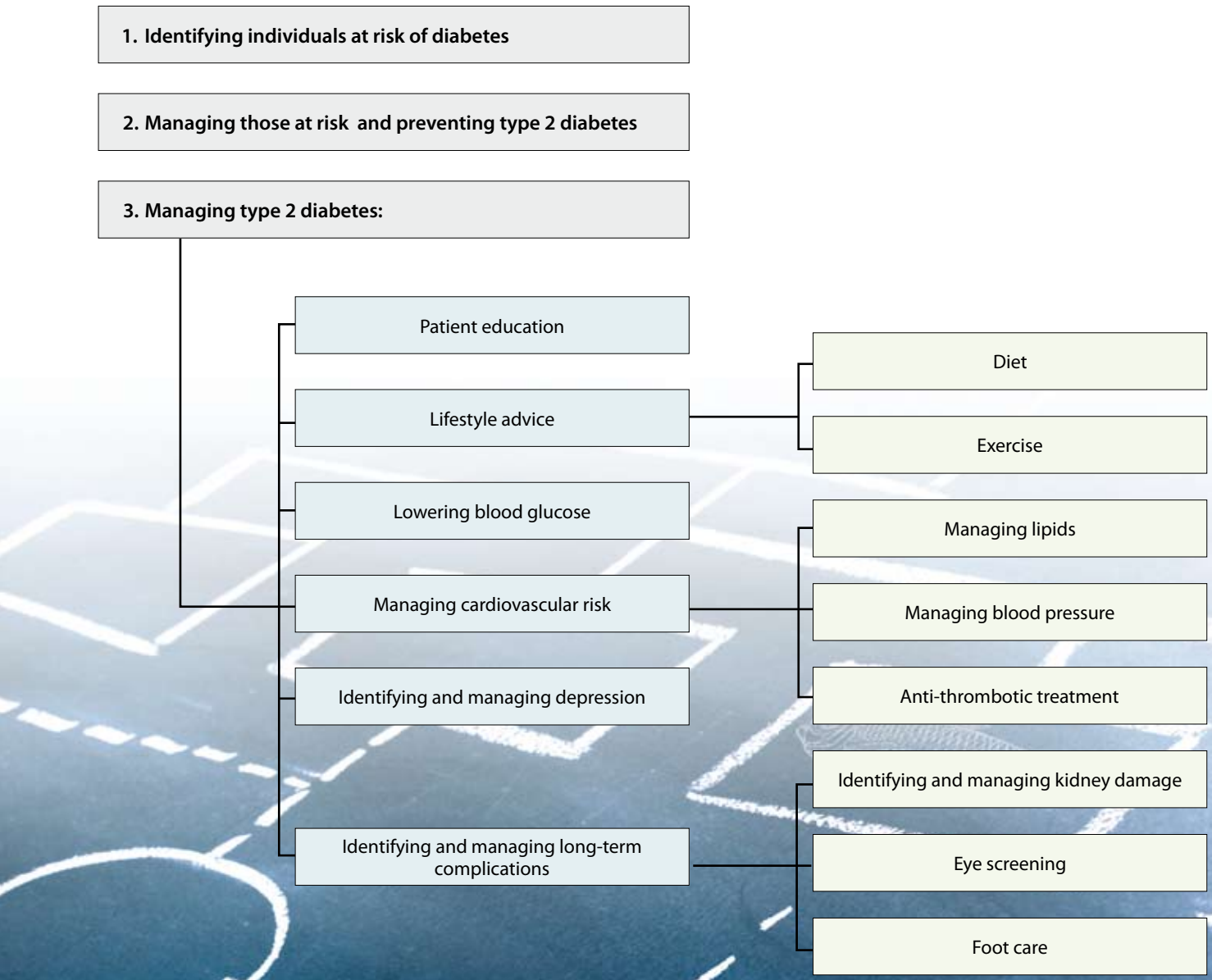
Figure 2: Representation of the volume of patients at each stage of a long-term condition

by encouraging patients to take an active role in their own care. Care plans are useful in educating patients about their condition, and include their individual clinical circumstances, their risk-factors, co-morbidities and management, as recommended by the care pathway. The patient's care plan will change as their risks change and complications occur, as opposed to the pathway which is a rigid overview of recommendations and only changes with the evidence. Care pathways and care plans, when combined, provide patients with an individualised best practice approach for their care and are increasingly recognised as being an essential element for improved outcomes for long-term conditions.

Will pathways reduce clinical judgment and individual choice?

Clinical judgement and individual patient preference remain of paramount importance. Choice should not be reduced by the use of a care pathway. For example, currently the usual recommendation for a target HbA_{1c} in a person with type 2 diabetes is 50 – 55 mmol/mol, however, in an elderly person this target level of control may be individualised, due to the complexities and increased risks associated with hypoglycaemia in this age group. Care pathways are not about standardising care for every individual. The setting of agreed individual targets within a care plan allows for flexibility and achievable goal setting.

The type 2 diabetes care pathway



The two indicators for diabetes are still funded

Most DHBs have yet to release details on their approach to the Diabetes Care Improvement Package (DCIP). The two PHO Performance Programme (PPP) Indicators for diabetes, "Diabetes detection" and "Diabetes annual review", remain as funded indicators and will, for now, continue under any new DCIP programmes. Funding will be allocated for the total and the high needs groups; the high needs group for both indicators includes Māori and Pacific peoples and people living in NZDep decile 9 and 10 socioeconomic areas. The two indicators represent 16.5% of the total allocated PPP funding.[6]

In some regions, free annual check-ups are no longer available for all patients. This is likely to make meeting the annual diabetes review indicators significantly more difficult, however, by working with patients and communicating the need for regular review and highlighting the risks of not doing so, general practice can continue to provide consistent, high-quality care to all people with diabetes.

Diabetes Detection

The PPP Indicator for Diabetes detection is measured as the percentage of the population estimated to have diabetes that has been diagnosed with diabetes.

The programme's goal is for: at least 90% of the people aged 15 – 79 years who would be expected to have diabetes to be coded as having diabetes.

The indicator accounts for 7.5% of the annual PPP funding; 2.5% for the total population and 5% for the high needs group.

For further information on diagnosing diabetes, see: "The new role of HbA_{1c} in diagnosing type 2 diabetes", BPJ 42 (Feb, 2012).

Diabetes Annual Review

The PPP Indicator for Diabetes annual review is the percentage of people with diabetes who have had an annual check-up.

The programme's goal is for: at least 90% of people aged 15 – 79 years with diabetes to have a record of a Diabetes Annual Review during the reporting period.

The indicator accounts for 9% of the annual PPP funding; 3% for the total population and 6% for the high needs group.

For further information on how to perform a diabetes annual review, see: "Diabetes follow-up: what are the PHO Performance Programme goals and how are they best achieved?", BPJ 39 (Oct, 2011).

References

1. District Health Boards New Zealand. National summary of PHO performance as at 1 July 2012. DHBNZ; 2012. Available from: www.dhbsharedservices.health.nz/Site/Current-Issues/Performance-Results-Dec-2011.aspx (Accessed Oct, 2012).
2. Ministry of Health (MoH). Diabetes in New Zealand: models and forecasts 1996-2011. MoH: Wellington, New Zealand; 2002. Available from: www.health.govt.nz (Accessed Oct, 2012).
3. Scragg, R. Asian Health in Aotearoa in 2006 - 2007: trends since 2002-2003. Auckland: Northern DHB Support Agency, 2010.
4. Rotter T, Kinsman L, James E, et al. Clinical pathways: effects on professional practice, patient outcomes, length of stay and hospital costs. Cochrane Database Syst Rev 2010;(3):CD006632.
5. Giorda CB. The role of the care model in modifying prognosis in diabetes. Nutr Metab Cardiovas 2012;In Press, Corrected Proof.
6. DHBNZ. PHO Performance Programme. Indicator definitions. Version 5.5 . 2012. Available from: www.dhbnz.org.nz/Site/SIG/pho/Operational-Documents.aspx (Accessed Oct, 2012).

Using the New Zealand Formulary

Hidden Gems: Locating product and funding information

At the click of a button, The New Zealand Formulary provides up-to-date information on product specifications/descriptions, excipients, availability and funding , with links to the Medicine Datasheet and Special Authority forms.

How to find detailed product information for medicines in New Zealand Formulary (NZF)

Comprehensive product information can be found in the New Zealand formulary, by expanding the ✚ icon for each heading in the drug monographs. The information is intentionally not visible at first, to give an uncluttered, single-frame view of the forms of a medicine that are available.

Below the Dose field is the preparation group box; click on the ✚ icon beside each **product/strength** for product specific details, or the ✚ sign beside the blue Medicine heading to view all products that are available for this medicine. The process is the same for combination product monographs.

Using the "hovers"

The New Zealand Formulary frequently uses hover functionality, to keep the screen as uncluttered as possible until further information is required. Hovering the computer mouse over a symbol or words with a dashed underline, will reveal a box with information relating to that icon. The information will remain visible while the mouse is hovered. Click rather than hover the mouse if you would like the information to stay in a fixed display; to close the hover display box, click the × button in the top right hand corner.

Links to other information

Words or phrases with solid underlines can be clicked for links to other places in the formulary, or to other relevant web pages. Users are directed to the appropriate place within other websites, rather than having to search a website for the required information.

Please place feedback if any links are not working for you.

Understanding the icons

Italics Indicates brand name; there can be several brands available for any medicine.

Indicates the product is a prescription medicine. Further details of the legal classification that relate to pack sizes, saleable quantities and criteria for supply by registered allied health practitioners are found by hovering over 'legal classification' at the top right corner of the preparation group box.

C2 Indicates the medicine is a Controlled Drug; the class (B1 to B3, C1 to C7) is specified.

Links to the Medicines Datasheet; datasheets are brand specific. Not all medicine products have a datasheet but all available datasheets are included in the link.

Links to the Consumer Medicine Information (CMI) sheets, where available. Pharmaceutical companies are responsible for producing CMI. However, in New Zealand it is not a legal requirement for them to do so. The CMI is written by pharmaceutical companies using guidelines set by Medsafe.

Indicates the product is fully subsidised by PHARMAC. Any

restrictions, including a link to any Special Authority forms, and any direction that a pharmacist must dispense a three-month supply all at once, are found here.

Indicates the product is partially subsidised by PHARMAC. The patient will be required to pay the difference between the subsidised amount and the actual cost of the medicine, including mark-ups and taxes. The total payable by the patient will therefore depend on the quantity prescribed.

Indicates a Restricted Medicine, which may be sold in a pharmacy by a registered pharmacist, in accordance with legal limitations (provision of advice, quantity, labelling; hover over legal classification' for full details).

Indicates a Pharmacy Only Medicine, which may be sold in a pharmacy in accordance with legal limitations (provision of advice, quantity, labelling; hover over 'legal classification' for full details).

A Restricted or Pharmacy Only Medicine may also be included in the New Zealand Pharmaceutical Schedule and subsidised on a prescription written by a doctor.

Indicates a General Sale Medicine

CALs Cautionary Advisory Labels provide brief, important information for patients. Medicines dispensed by pharmacies usually include this information within their labelling or as a supplementary yellow label. Additional comments provide practical advice on how to take the medicine.

Section 29 Indicates Section 29 medicines that are not approved by Medsafe for marketing within New Zealand. Unapproved medicines are not usually subsidised by PHARMAC.

Excipients can be clinically important

An excipient is a component or ingredient in a product formulation that is not the active ingredient. Excipients can cause adverse reactions, such as allergy. In addition, large quantities of some salts and preservatives may cause a clinically significant adverse effect.

When the information is available from the manufacturer, the excipients that may be of risk are noted; not all excipients are listed. Examples include aspartame, benzyl alcohol, polysorbate and gelatine. More information can be obtained from medicines manufacturers, or a Medicines Information service.

A reminder about searching

New Zealand Formulary feedback has highlighted some problems users are having with searching for a medicine. Note that the left hand search box requests the generic name of a medicine. It is necessary to use the right hand box if searching for a brand name, condition or phrase.

There are two spellings for some medicines

The New Zealand Formulary is based on the British National Formulary. In recent years, the British National Formulary has moved towards using rINNs (recommended International Nonproprietary Names), such as sulfasalazine or beclometasone, instead of BANs (British Approved Names), such as sulphasalazine or beclomethasone. This is also in line with the World Health Organisation's recommendations for the nomenclature of drugs.

The New Zealand Formulary uses rINNs for drug names. However, both spellings are currently included in medicine monographs, and are searchable within the formulary.

Special Authority forms

Links to printable PHARMAC Special Authority forms are found alongside the full or partial subsidy icons or by hovering over then clicking on (restrictions).

User feedback

Feedback to the New Zealand Formulary is welcome. Use the "feedback" tab alongside the "search" and "browse" tabs at the top of each page. Include a contact email to ensure you receive a personal reply.

Visit: www.nzformulary.org

Risk of toxicity from nasal application of mupirocin?

Dear Editor,

Re: "Managing skin infections in Māori and Pacific families" (BPJ 45, Aug 2012) – use of intranasal bactroban. Do we need to be concerned about the propylene glycol content, as we have been warned about in the past, when applied up the nose?

Sarah Bull
Pharmacist

Fusidic acid is the first-line choice for treating recurrent skin infections and community outbreaks (e.g. impetigo), where nasal carriage is suspected. Mupirocin is also effective, but it is active against MRSA so use should be reserved for when susceptibility testing shows MRSA to be present.

The topical mupirocin formulation (Bactroban ointment) available in New Zealand does contain a polyethylene glycol base and can cause irritation to the mucous membranes when applied intranasally. However, the amount of polyethylene glycol absorbed from nasal application is very unlikely to cause systemic toxicity to the patient. Caution is only required in people with moderate to severe renal impairment, if Bactroban ointment is being applied to a large area of broken skin, where systemic absorption of polyethylene glycol may worsen renal impairment (due to renal excretion).

Given that there is no alternative form available, and that mupirocin is the recommended treatment for infection or eradication of carriage of MRSA, the benefits of using it are likely to outweigh the risks.

PCR testing for pertussis: what swab to use?

Dear Editor,

The recommendation to use a "dry orange top tube" for PCR testing for pertussis [in "Pertussis: an avoidable epidemic" BPJ 45 (Aug, 2012)], has caused a bit of confusion.

Laboratories seem to have differing sample requirements and instructions. For example, LabPlus instructions are for swabs in transport media and Canterbury Health Laboratories request a dry swab only, but have no reference to an orange top.

Medical Laboratory Scientist, *North Island*

The "orange topped tubes" referred to in the Pertussis article are the flock-topped nasal swabs with a flexible head. This is the recommended swab to use, as the flock top picks up (and releases) more material than the conventional cotton or Dacron swab, and the flexible head allows for the best chance of correct nasopharyngeal swabbing technique.

A swab which uses a charcoal medium for transport of the specimen is not recommended because a PCR analysis cannot be conducted on a sample that has been placed in charcoal medium, and the swab will therefore be rejected by the laboratory.

The charcoal swab is used for pertussis culture, which is less useful than PCR as it has a lower sensitivity and a narrow time frame in which a sample will return a positive result. Pertussis culture is no longer recommended, although it is still available from some laboratories.

It appears that there are inconsistencies in the type of swab recommended by New Zealand laboratories. Most laboratories will accept a swab in a dry tube or a tube with universal viral transport medium, regardless of the swab or tube colour. Wire swabs are also acceptable, as long as the thinner, flexible head wire swab is used. The thicker wire swabs used for nose or throat swabs are not recommended as they are not flexible enough to be used to take a correct nasopharyngeal sample.

In summary, the three most common swabs that can be used for pertussis PCR testing are:

- The orange top, flock-topped, flexible head swab, with dry tube
- The blue top, wire swab placed back in the dry tube (not in the charcoal medium tube)
- A flock-topped, flexible head swab that is broken off into a short tube with universal viral transport medium

Other types of swab may be available and it is recommended that practitioners check with their local laboratory as to what the preferred sample method and materials are.

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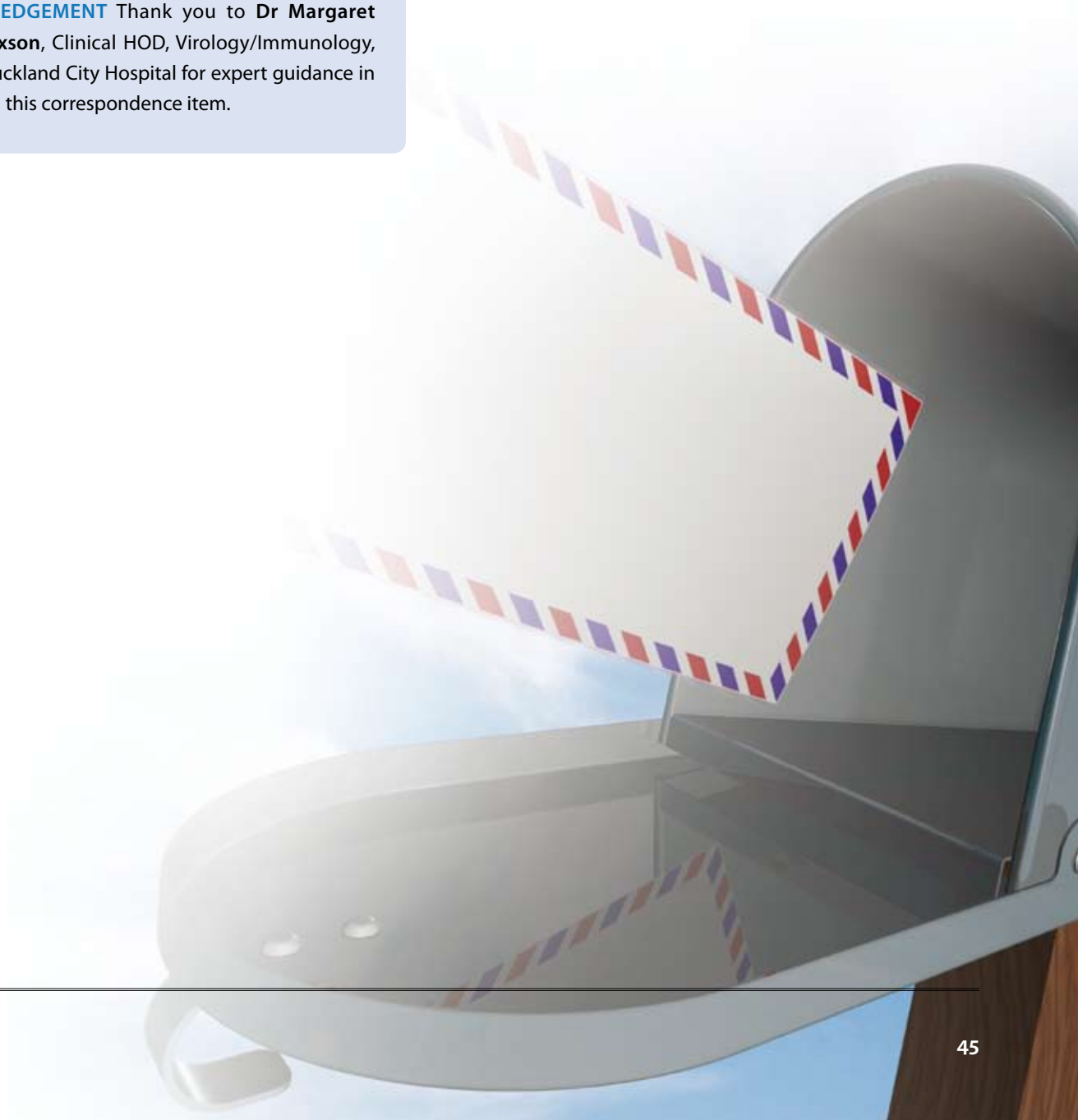
We value your feedback. Write to us at:
Correspondence, PO Box 6032, Dunedin
or email: editor@bpac.org.nz



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