BEST PRACTICE

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OCTOBER 2010

Which Antihypertensive?
The Warfarin Dilemma
Clopidogrel
The Diabetic Foot

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better medicine

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

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

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Best Practice Journal (BPJ)

ISSN 1177-5645

BPJ, Issue 31, October 2010

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

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

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

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

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

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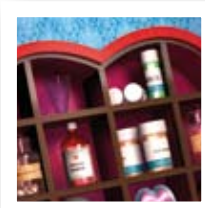

Which antihypertensive?

Once the decision has been made to initiate antihypertensive treatment, choice of medicine should be based on individual patient characteristics including age and co-morbidities. Combination treatment is ultimately needed to control blood pressure in the majority of patients so it is less important which antihypertensive is used initially. In non-frail, older people without co-morbidities a low dose thiazide diuretic is suitable as first-line treatment, unless contraindicated or if indications are present for one of the other treatment options (ACE inhibitor, calcium channel blocker or beta blocker).

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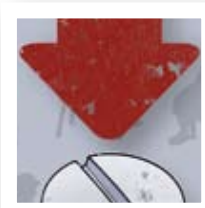

The warfarin dilemma

Oral anticoagulation with warfarin in older people with atrial fibrillation

Evidence suggests that warfarin is under utilised in older people. The dilemma is that in older people with atrial fibrillation, the factors indicating a need for anticoagulation with warfarin are also the risk factors for intracranial haemorrhage. Providing bleeding risks can be managed, warfarin is still the most effective treatment in this group of people and should be considered for individual patients, based on an assessment of bleeding risk, stroke risk, co-morbidities, concurrent medicines and likely compliance with monitoring. Increasing age alone is not a contraindication for warfarin use.

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Access to clopidogrel now widened

Clopidogrel, an antiplatelet medicine, is now able to be prescribed without Special Authority approval. Clopidogrel is not recommended for use in primary prevention of cardiovascular disease (CVD), but it can be considered for use in people with established CVD in place of aspirin, when aspirin is not tolerated or contraindicated. Clopidogrel may also be used in secondary stroke prevention as an alternative to aspirin/dipyridamole, in acute coronary syndrome without ST-segment elevation and in post-revascularisation procedures.

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Screening and management of "the diabetic foot"

Foot ulceration and damage is one of the most common complications of diabetes and without regular screening and effective management, patients are at high risk of lower extremity amputation. Feet should be checked at least once per year in every person with diabetes and more often in those who are at higher risk of developing foot complications. Management focuses on prompt treatment and referral for any detected foot problems and providing patient education about foot care.

Supporting the PHO Performance Programme

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INR point of care testing in community pharmacies – is this the future?

The international normalised ratio (INR) is used to monitor patients receiving warfarin for treatment or prevention of thrombosis and embolism. The therapeutic range of warfarin is narrow, so monitoring of INR is performed to avoid complications from both over-dosage (which increases the risk of haemorrhagic events) and under-dosage (which may result in thromboembolic events).

In New Zealand, most patients prescribed warfarin have their INR levels measured in a laboratory and receive advice from general practice on their next dose and testing frequency. Some general practices have moved away from laboratory based INR testing and have adopted surgery based INR point-of-care testing (POCT) using portable coagulometers.

In 2009 a pilot was undertaken for INR testing and warfarin management using POCT in a community pharmacy setting. It is now being followed by a larger pilot study. The future of INR monitoring could be changing with testing and management increasingly coming into the care of community pharmacies.

It is likely that there will be a mixed response to community pharmacists adopting the role of INR testing. Some GPs and practice nurses may not be overly concerned and may even be pleased by the prospect of having one less task to worry about in their already-stretched workload. Others may feel that if the task of INR monitoring is mismanaged there could be serious consequences and so may be concerned to see it delegated outside of general practice.

Community pharmacy based INR point-of-care testing pilot

New Zealand's first trial in community pharmacy-based POCT of INR took place in 2009. An anticoagulation clinic was set up at one community pharmacy using a portable coagulometer and a web-based management

support tool, which allowed a revised warfarin dose to be calculated from the INR result. Patients were shown a pictorial representation of their warfarin dose and informed when their next test was required. The process took on average less than ten minutes to complete and also provided a chance to counsel patients about their warfarin management at each appointment.

The pilot study ran from July to November 2009, and involved 40 patients with prior consent from their GP. Results have not yet been published.

Larger pilot trial currently planned

After observing the original pilot study, the Pharmaceutical Society of New Zealand supported the extension of the programme nationally and has planned a community pharmacy Anticoagulation Management Service pilot. This pilot will involve 15 pharmacies, each of which will enrol 50 patients with atrial fibrillation using warfarin. The pilot will run for one year, with an evaluation planned before the end of 2011.[1]

As with the original pilot study, the accredited pharmacists during this POCT of INR trial will:

- Check the patient's INR levels
- Input the result into a computer programme for dose recommendation
- Advise the patient of their next appropriate warfarin dose
- Notify the GP of the blood result, dose and date of next test for the patient

The pharmacists will perform these tasks under standing orders from the patient's GP, with communication protocols in place to ensure the GP remains fully informed and in charge of their patient's care. The aim is to maintain INR levels within safe parameters. Appropriate protocols will be put in place for referral back to the patient's GP, if required.[1]

INR point-of-care-testing is quick and simple to perform

POCT of INR is performed by obtaining a drop of capillary blood from a patient via fingerprick which is then processed in a portable coagulometer. An INR result is usually obtained within three minutes.

Advantages of POCT of INR include:

- INR results are obtained sooner allowing discussion of the result and any change in management at the same visit as the INR testing
- It is a more acceptable method for people who have fears of venepuncture
- It is more convenient for patients especially if they live some distance from phlebotomy services
- Possibly improved compliance with warfarin as a result of having face-to-face guidance given rather than over-the-phone

The risks of POCT are those associated with obtaining the capillary sample including: localised bleeding, bruising and vasovagal episodes. There is also a risk of needle-stick injury when obtaining the sample but this is unlikely to pose any additional risk to that associated with venepuncture for laboratory-based INR testing.

Reaction to the proposed pilot study

The pilot POCT study is an opportunity for a partnership between pharmacy and general practice and for maximisation of pharmacists skills. However, it has been suggested that trialling of POCT for INR should also be carried out in a general practice setting for a true comparison of services.

There is concern that patients undergoing anticoagulation management, who are not regularly seen in general practice, will have compromised care. Anticoagulation management is multifactorial and complex, and patients have multiple needs in addition to a check of their INR level.

On the other hand, the number of people receiving oral anticoagulation therapy, most commonly for stroke prevention in non-valvular atrial fibrillation, is growing each year, with the increasingly ageing population. Increasing demand for monitoring could stretch the resources of general practice, if it is continued to be managed there alone.[2]

The question of cost has not been fully addressed. It is unclear whether individual pharmacies will purchase the necessary equipment for POCT and in turn, what cost will be passed on to the patient, bearing in mind that current laboratory testing of INR is fully funded.

It is likely that the results of the Anticoagulation Management Service pilot will answer some of these questions and help to determine whether INR testing fits into the community pharmacy setting, both in terms of patient and clinician satisfaction and improved patient safety.

The benefits of point-of-care testing of INR

An essential aspect of improving health services in New Zealand is to encourage the provision of better and convenient access to healthcare for all patients, particularly those with chronic illnesses and those who encounter barriers to accessing services. POCT represents a way to provide a convenient service, faster results and to facilitate quicker clinical decisions.

General Practices, with a desire to provide this service and the necessary resources and capacity, are an ideal setting for POCT of INR. It is also increasingly recognised that pharmacists are well-placed to provide patient-centred services such as POCT. Similar developments involving community pharmacists are taking place in the UK, Canada and South Africa.[2] Community delivery of POCT for INR could also take place in residential aged-care facilities, led by pharmacists or general practice staff.

POCT of INR does not appear to be better than laboratory testing of INR, but it is at least as accurate and at least as effective in maintaining INR in the target range. It is also likely to be more convenient for many patients.

Is POCT of INR accepted by patients?

One study of POCT of INR by nurses in a general practice setting, found that significantly more patients preferred POCT of INR compared to usual care i.e. laboratory testing. This was due to factors such as improved capacity to make appointments, less time spent at appointments, less pain associated with the test and improved communication about medicine dose.[3]

Research in the UK, as part of a project on monitoring of diabetes and chronic heart disease using POCT, indicated that 34% of patients chose their pharmacy to monitor their condition instead of their GP. Of this 34%, almost all (97%) rated the pharmacy service better or equal to their GP. Convenience, both in terms of location and opening hours, is a key advantage of pharmacy based POCT.[2]

Is POCT of INR as accurate as laboratory testing?

In a general practice based study, calibrations of POCT and laboratory testing showed dependable INR levels from both systems.[4]

Community pharmacy-based POCT of INR has also been shown to be as accurate as laboratory INR monitoring. A study involving POCT at 16 rural pharmacies in Australia found the same results when 120 INR tests performed in the pharmacy setting were compared with laboratory tests taken within four hours.[5]

Are target INR levels achieved with POCT?

There is mixed evidence of the benefit of POCT compared to laboratory testing in maintaining INR within the target range, however POCT appears to be at least as effective.

A US-based observational study in a primary care clinic found a significant improvement in the percentage of visits in which a patient's INR result was in the target range after POCT was implemented (from 34% to 67% over one year).[6] In a more recent randomised controlled trial of POCT in general practice in Australia, there was no significant difference between the POCT and control groups (who received the usual laboratory based testing) in terms of the number of patients with results in the target range for INR (57% POCT vs 61.5% control, p=0.24).[7] Another randomised controlled trial also found that there was no significant improvement in the time spent in the INR target range, between those who received POCT in a community clinic and those who received laboratory testing.[4]

One pharmacy-based study found that more than 80% of patients receiving POCT of INR had values within their targeted range 60% or more of the time, which is comparable with values reported for anticoagulation clinics.[8]

Is POCT of INR cost-effective?

There is currently no strong evidence of the cost–effectiveness of POCT of INR in either the general practice or community pharmacy setting. New Zealand specific data is required in order to accurately estimate the cost of POCT in this country.

A large trial in a general practice setting in Australia found there was no significant difference in overall costs between POCT and laboratory testing. There was a non-significant decrease in hospital admissions for patients using POCT of INR. POCT of INR increased the number of tests that people were receiving compared to those receiving laboratory tests. Overall, it was concluded that POCT of INR was not cost-effective in the general practice setting compared to usual care.[9]

Several other studies have concluded that POCT in a general practice setting is more expensive than laboratory testing.[7, 10] However, other studies have found that POCT provided an overall saving for health care providers or from a patient perspective, through a reduction in patient visits to the GP.[6, 11–13]

A Canadian study found that both physician and pharmacist-managed anticoagulation services were associated with improved INR control, but pharmacist-managed services may be more expensive in the long-term.[14]

It is difficult to calculate cost in terms of just economic value. Costs may be offset by prolonged life or reduced hospital stays.[9]

Warfarin is underused in areas with limited access to pathology services

Research shows that warfarin may often be underused in areas in which access to pathology services for INR monitoring is limited. Patients in these areas who do use warfarin are also potentially at increased risk of under- or over-dosing events.[15] The availability of portable INR monitors in such settings would be likely to increase the level and safety of warfarin use.

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ANTIMICROBIALS

How should they be used in primary care?

Contributed by **Dr Rosemary Ikram**, Clinical Microbiologist, MedLab South

The first article in this series (BPJ 30, Aug 2010) outlined the problem of antibiotic resistance in the community. This article considers what interventions could lead to improved use of antimicrobial agents and allow the best chance of slowing the spread of resistant bacteria.

Ideally antibiotics should be reserved for the treatment of known bacterial infection but it is well recognised that they are often prescribed empirically "just in case" or inappropriately when the infection is highly likely to be viral. For a specific infection, the antibiotic with the narrowest useful spectrum should be selected and the entire treatment course should be taken. To accomplish this, in some cases it may be appropriate to take a sample for testing or consult antibiotic susceptibility guidelines.

Is this a bacterial infection?

Deciding whether a patient has a bacterial infection can sometimes be challenging. The difficulties and uncertainties are partly reflected by the variability in microbiology test ordering patterns in primary care. A United Kingdom based study investigated microbiology test ordering rates for different practice localities and found a 200% variation in rates for urine samples and an 800% variation for wound swabs.[1] This suggests that more education is required to guide practitioners on appropriate microbiological testing along with the implementation of guidelines. In this era of increasing antibiotic resistance it may be necessary to re-evaluate some of the current practices. For example, we know that more resistant bacteria will be isolated from patients who have had previous antibiotic treatment[2] and the antibiotic susceptibility of organisms such as *E. coli* is less predictable in those who have travelled to or lived in areas with high levels of endemic resistance.[3]

A useful approach is to ask the question; "How likely is this to be a viral infection?" It is clear that most respiratory tract infections such as sore throats, acute bronchitis, acute otitis media and coryza are usually viral in origin. There may be uncertainty as to the likelihood of a bacterial infection as well as an expectation from the patient or parent/caregiver that an antibiotic should be prescribed. The United Kingdom National Institute of Health and Clinical Excellence (NICE) recently published a short clinical guideline on antibiotic prescribing for respiratory tract infections.[4] After a face-to-face consultation, including patient history and an examination, patients can be categorised into three different management groups - antibiotics are not recommended, a delayed ("back pocket") prescription is given or antibiotics are prescribed.

Prescribers are encouraged to download a copy of the NICE guideline and use it to help inform their prescribing decisions: **www.nice.org.uk/nicemedia/live/12015/41322/41322.pdf**

Interventions to improve prescribing – what works?

There is currently insufficient research to determine which single approach to rational use of antimicrobials is the most effective. A recent Cochrane review suggests that multifaceted approaches and interventions targeting patients show the most promise. The main conclusions were that:[5]

- Patient based interventions including information, education and delayed or "back-pocket" prescriptions, consistently decreased patient antibiotic use (a patient information pamphlet is available from bpac[nz])
- Multifaceted interventions which combined education for doctors and patients with public information campaigns consistently reduced antibiotic prescribing for inappropriate conditions
- Educational outreach including reminders to doctors and audits had mixed effects on prescribing practices
- Educational meetings improved antibiotic prescribing, but effects were variable and generally modest
- Printed educational material such as flyers or leaflets had little effect on prescribing behaviour

The authors suggested that the most effective interventions are likely to be those that address local prescribing behaviours and barriers to change, and include patients and the public in the educational programme. Local barriers should be addressed before major educational efforts are implemented. An example of this is the variable rate of rheumatic fever in New Zealand – some areas, particularly in Northland, have very high rates but in the South Island much lower rates occur. Therefore a protocol implemented across the whole population will be neither the most appropriate nor worthwhile intervention.

Should children be educated about antimicrobial use?

In some countries children are taught the fundamentals of antimicrobial use at primary and secondary school level. The main issues covered are resistance and appropriate use, e.g. antibiotics are ineffective for colds and influenza. Finland and Moldova were the first countries to implement this as part of the school curriculum and a positive effect on parent knowledge and education about antibiotics has been observed.

These initiatives have recently been expanded in Europe with the development of a web site for school educational use (e-bug). Teaching children about antibiotic use would seem a logical approach given the need for wider public knowledge of the issues. Several pilot studies have been carried out in New Zealand, but the concept is not yet widespread. Bpac[nz] supports this initiative.

www.e-bug.eu

Delayed prescriptions

A study in Auckland reported that delayed ("back pocket") antibiotic prescriptions effectively reduced antibiotic use.[6] Interestingly, GPs valued empowering patients to be more involved in decision making about their health care management more than patients did. GPs generally viewed the strategy as providing reassurance to patients and meeting their expectations. Both patients and physicians agreed that delayed prescribing is not appropriate for everyone, but currently no consistent criteria have been established.

Antibiotic choice and use

When prescribing antimicrobial treatment it is important that a narrow spectrum antibiotic is chosen in most cases and the length of treatment is kept as short as possible. Antibiotic treatment affects both the pathogen it is targeted against, and the whole bacterial flora of the patient. There is evidence that antibiotic treatment leads to the presence of more resistant bacteria in the normal flora and also in subsequent infections.[2] In general practice it has been shown that this effect is prolonged and can also be related to the length of treatment. Broad spectrum antibiotics have more effect on the flora than narrower spectrum agents.

It is necessary to provide local antibiotic susceptibility data to the primary sector to allow antibiotic guidelines to be formulated locally. To enable this to happen there needs to be communication between the laboratories testing microbes from the community, referrers and local experts in the treatment protocols relevant to specific geographical areas. In the United Kingdom the Health Protection Agency have produced a document: "Management of Infection Guidance for Primary Care for Consultation and Local Adaptation".[7] Using this document and other guidelines it should be possible to develop a similar document for New Zealand primary care.

In Summary...

Both health professionals and patients need to review how antimicrobials are currently being used. This involves being aware of the susceptibility of bacteria locally, having a clear understanding of when antimicrobials are not indicated and using resources such as education for both prescribers and patients to enable optimal use of these valuable medicines. If this can be achieved we shall be on the way to at least slowing the spread of antimicrobial resistance in New Zealand.

ACKNOWLEDGMENT Thank you to **Associate Professor Mark Thomas**, Infectious Disease Specialist, University of Auckland for his contributions to this article.

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RESISTANCE TO ANTIMICROBIALS

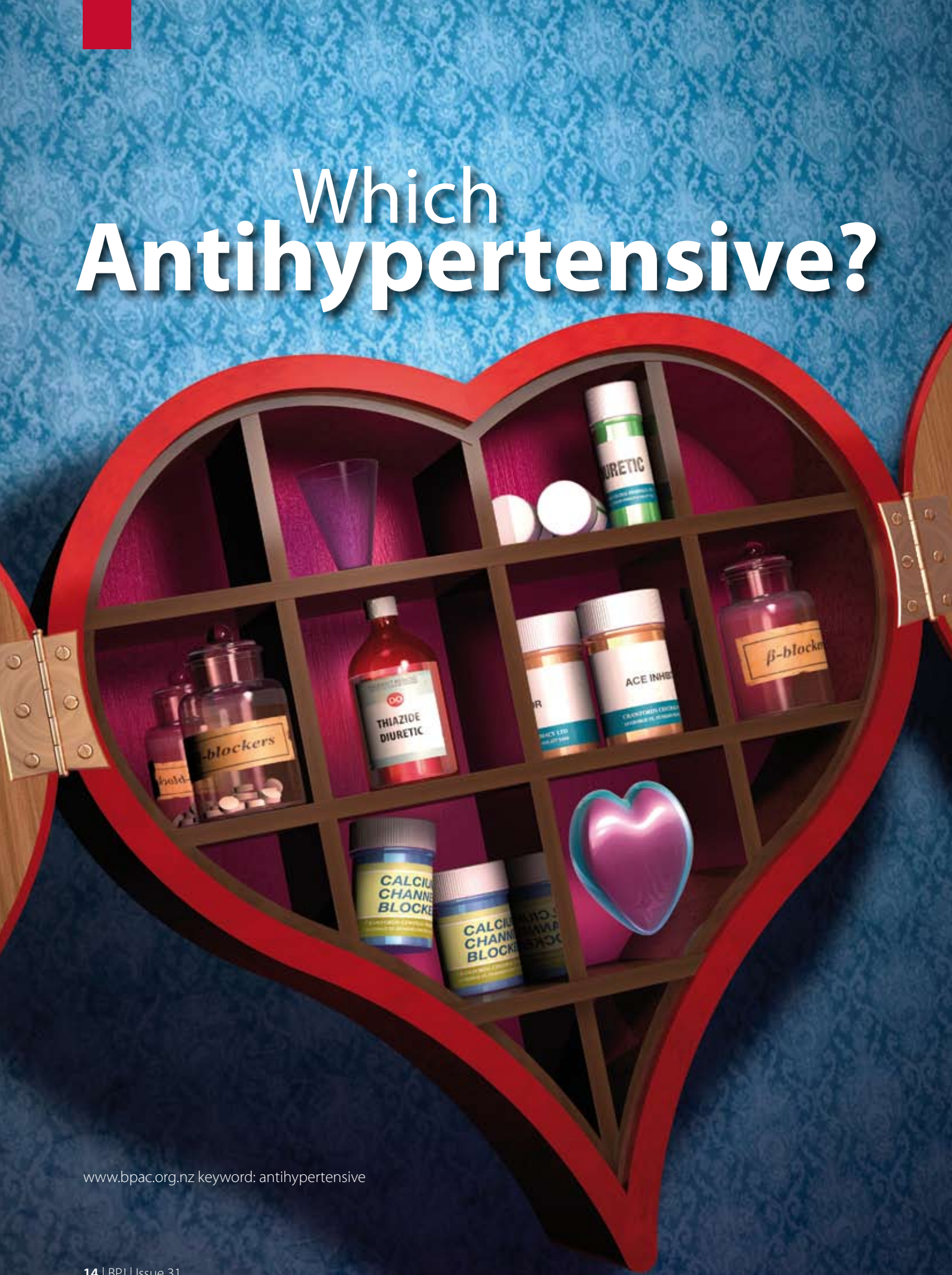
is an increasing problem in our community

We challenge you to examine the use of antimicrobials in your practice and to consider ways in which you may contribute to reducing resistance in our communities.

Prescribers are invited to complete a questionnaire about antimicrobial use in primary care. This is available online at:

www.bpac.org.nz

Which Antihypertensive?



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Choosing an antihypertensive medicine

The main benefit of any antihypertensive treatment is lowering of blood pressure and this is largely independent of the class of medicine used.[1] Once the decision has been made to initiate antihypertensive treatment, choice of medicine should be based on individual patient characteristics including age and co-morbidities.

The main classes of antihypertensive medicines are; thiazide diuretics, angiotensin converting enzyme (ACE) inhibitors (or angiotensin receptor blocker [ARB] for those who are not able to tolerate an ACE inhibitor),calcium channel blockers and beta blockers.

There is much debate on which antihypertensive medicine is the most appropriate first choice. In practice, combination treatment is ultimately needed to control blood pressure in the majority of patients so it is less important which antihypertensive is used initially.[2] Some patients may respond well to one medicine but not to another.[1]

Beta blockers are not usually considered for first line treatment of hypertension, except when used for their protective effect in ischaemic heart disease and heart failure, and for their rate-controlling effect in atrial fibrillation.[3] The effectiveness of beta blockers in reducing major cardiovascular events (stroke in particular) compared to other antihypertensive agents is currently under review.

Key concepts

- In patients with uncomplicated, mild hypertension and in elderly people, initiating a single antihypertensive medicine is appropriate first-line treatment
- Selecting which antihypertensive to use can be based on co-morbidities and individual patient characteristics
- Thiazide diuretics, ACE inhibitors and calcium channel blockers are all appropriate initial choices and beta blockers may be used first line in selected groups of patients
- In general, an ACE inhibitor may be selected for a younger patient (<55 years) and a diuretic or calcium channel blocker selected for an older patient, if there are no compelling indications for another choice
- If blood pressure targets are not achieved with monotherapy, consider initiating combination therapy - the majority of people with hypertension will require at least two antihypertensive medicines to achieve recommended targets
- In patients with moderate to severe hypertension or high to very high cardiovascular risk, combination therapy can be initiated as first-line treatment
- The choice of antihypertensive combination can be based on selecting medicines with different actions and on individual patient characteristics. An ACE inhibitor plus a diuretic or calcium channel blocker is a commonly used regimen.
- “Start low, go slow” unless otherwise indicated
- If patients experience adverse effects, changing early to a more tolerated medicine will improve adherence

Monotherapy is a practical starting point

> **"Monotherapy is recommended initially, especially for patients with mildly elevated blood pressure and low to moderate total cardiovascular risk. A low dose thiazide diuretic is recommended as first-line treatment, unless contraindicated or if indications are present for one of the other treatment options."**

In patients with uncomplicated, mild hypertension and in elderly people, antihypertensive therapy can be initiated gradually after a period of life style changes, e.g. three to six months. Monotherapy is recommended initially, especially for patients with mildly elevated blood pressure (140 – 159/90 – 99 mmHg), and low to moderate total cardiovascular risk.[2]

The New Zealand Guidelines recommend a low dose thiazide diuretic as first-line treatment, unless contraindicated or if indications are present for one of the other treatment options.[4] For example, a beta blocker may be appropriate as a first-line treatment when there are co-existing cardiac problems such as ischaemic heart disease and heart failure. ACE inhibitors or calcium channel blockers can also be used initially. Choice is based on individual patient characteristics, including age, ethnicity, contraindications or compelling indications for specific medicines, adverse effects and relative cost effectiveness (Table 1).[5]

Treatment should be initiated at a low dose. If blood pressure is not controlled after six weeks, either a full dose of the initial medicine can be given, or patients can be switched to a medicine of a different class (starting at a low dose and then increasing). If blood pressure control is not reached, low doses of two medicines is preferable to increasing to a maximum dose of a single medicine. This approach maximises efficacy while minimising adverse effects.[6]

Best Practice Tip: Starting with even a low dose of an antihypertensive medicine can cause an exaggerated response in some people. Inform patients of the signs of hypotension especially in the early stages of treatment.

Patient co-morbidity influences antihypertensive choice

There are specific indications, limitations or contraindications for each of the antihypertensive medicine classes for individual patients, depending on their co-morbidities.[7]

Compelling indications include the use of ACE inhibitors or ARBs in patients with nephropathy and beta blockers in patients who have had a myocardial infarction.[4] Equally, there may be clinical reasons to avoid a particular class of antihypertensive (Table 1).

Age influences antihypertensive choice

Unless a patient has a specific indication for a particular antihypertensive class, there are some medicines which may be best suited to them based on their age.

ACE inhibitors for younger patients: Treatment guidelines from the United Kingdom recommend that ACE inhibitors or ARBs are initiated for younger patients (aged under 55 years) with hypertension.[3]

In practice, many younger patients are started on an ACE inhibitor. Special Authority criteria apply for the prescription of an ARB. A limited number of studies have found ACE inhibitors and beta blockers to be more effective at lowering blood pressure in younger people compared to calcium channel blockers or thiazide diuretics.[8] One study found significantly greater responses in blood pressure levels in a group of younger patients (age 22 to 51 years) when treated with an ACE inhibitor and also when treated with a beta blocker, compared to when they were treated with a calcium channel blocker or a diuretic.[9] In the absence of a compelling indication, beta blockers are not commonly used for initial monotherapy.

Thiazide diuretics and calcium channel blockers for older patients: United Kingdom guidelines recommend diuretics

Table 1: Choice of antihypertensive in patients with co-morbidities[6, 10]

Condition	Potentially beneficial	Cautions
Angina	Beta blockers (without ISA)* Calcium channel blockers ACE inhibitors	No specific cautions
Post myocardial infarction	Beta blockers (without ISA)* ACE inhibitors	No specific cautions
Atrial fibrillation	Rate control: beta blockers Verapamil, diltiazem	No specific cautions
Heart failure	ACE inhibitors, ARBs Thiazide diuretics Beta blockers e.g. carvedilol, metoprolol controlled release	Caution: Calcium channel blockers (especially verapamil, diltiazem) Contraindicated: Alpha blockers in aortic stenosis, beta blockers in uncontrolled heart failure
Chronic kidney disease	ACE inhibitors, ARBs	
Post stroke	ACE inhibitors, ARBs Calcium channel blockers Low dose thiazide diuretics	Thiazides in very elderly people or those with poor fluid intake could contribute to hypoperfusion
Diabetes	ACE inhibitors, ARBs Calcium channel blockers	Beta blockers Thiazide diuretics (risk of metabolic adverse effects mainly associated with high doses)
Symptomatic benign prostatic hypertrophy	Alpha blockers (add-on) e.g. doxazosin, prazosin	Alpha blockers could lead to postural hypotension in elderly people
Asthma/COPD	No specific recommendations	Beta blockers Cardioselective beta blockers e.g. metoprolol, atenolol, can be used cautiously in stable COPD, especially if specifically indicated, e.g. in heart failure Beta blockers are generally contraindicated in asthma
Gout	No specific recommendations	Thiazide diuretics: precipitation of gout unlikely especially if controlled with allopurinol

* ISA = intrinsic sympathomimetic activity. Beta blockers with ISA are: pindolol, oxprenolol and celiprolol, all other beta blockers are without ISA

or calcium channel blockers for older patients (aged 55 years or older) with hypertension.[3] Australian guidelines recommend thiazide diuretics as first line treatment in patients aged 65 years and older.[6] In very elderly or frail patients the decision to treat hypertension should be made on a case by case basis.

Older patients often respond best to a thiazide diuretic or calcium channel blocker and therefore these may be more effective initial choices in this group.[1] The use of thiazide diuretics and calcium channel blockers in older patients may have the additional benefit of managing isolated systolic hypertension. This is more prevalent in elderly people due to large vessel stiffness associated with ageing.[10] Older patients usually have lower plasma renin activity than younger patients, therefore ACE inhibitors and beta blockers may not be as effective.[1]

Hypertension in pregnancy

Suitable first line medicines for women with hypertension who are planning a pregnancy include labetalol, methyldopa and clonidine.[6]

ACE inhibitors, ARBs and diuretics are contraindicated at all stages of pregnancy. Calcium channel blockers are contraindicated in early pregnancy but have been shown to be safe and effective in the late second and third trimesters. Specialist referral is recommended for all pregnant women with hypertension.[6]

Combination diuretic therapy

"Most patients will require more than one antihypertensive medicine to reach their treatment target."

An estimated 50–75% of patients with hypertension will not achieve blood pressure targets with monotherapy.[6] Most patients will require more than one antihypertensive medicine to reach their treatment target.[4]

A combination of two medicines at low doses may also be used as initial therapy in patients with moderate to

Recommended doses for commonly used antihypertensives [6, 11]

Class	Commonly used medicines	Usual dose range
Thiazide diuretics	Bendrofluazide	2.5 mg once daily
ACE inhibitors	Cilazapril	0.5–5 mg once daily
	Quinapril	2.5–40 mg once daily or in two equally divided doses
	Enalapril	2.5–20 mg once daily or in two equally divided doses
ARBs	Candesartan	4–8 mg once daily (maximum 32 mg)
	Losartan	25–50 mg once daily
Calcium channel blockers (dihydropyridine)	Felodipine	2.5–10 mg once daily (controlled release)
	Amlodipine	2.5–10 mg once daily
Beta blockers	Metoprolol tartrate	50–100 mg twice daily
	Metoprolol succinate	23.75–190 mg once daily (controlled release)
	Atenolol	25–50 mg once daily
ACE Inhibitor with diuretic	Cilazapril (5 mg) with hydrochlorothiazide (12.5 mg)	
	Quinapril (10 mg or 20 mg) with hydrochlorothiazide (12.5 mg)	

Notes:

- Initial doses in older people or in those with renal impairment should be at the lowest end of the dose range.
- Atenolol is recommended only in combination with other agents. For patients on atenolol monotherapy, consider substituting for another beta blocker or another medicine class (due to adverse outcomes in meta-analyses of monotherapy clinical trials).[12]

Adherence to antihypertensive therapy

International studies suggest that up to one quarter of patients discontinue their antihypertensive treatment after six months, and this is associated with increased risk of hospitalisation for cardiovascular problems. In a recent large Canadian study, 22% of patients stopped their treatment completely within the first six months. Factors associated with an increased likelihood of continuing treatment were; better medical management and communication by the prescriber, early changes in treatment (if adverse effects are experienced), more follow up visits and non-diuretics as initial choice of therapy.[13] This study emphasises the importance of monitoring treatment and adverse effects, and making appropriate changes promptly to improve adherence.

highly elevated blood pressure or high to very high total cardiovascular risk.[2]

There is an additive effect when two antihypertensives from different classes are combined, and this is greater than the effect of increasing the dose of a single medicine.[4] The most effective combinations involve medicines that act on different physiological systems.[2] Most guidelines recommend renin angiotensin system inhibitors i.e. ACE inhibitors or ARB, in combination with a diuretic or calcium channel blocker as the preferred combination therapy.[3, 6, 14]

The combination of a thiazide diuretic and a beta blocker, although still effective, is not routinely recommended in people with glucose intolerance, metabolic syndrome or established diabetes.[2, 6] This is because of the additive combination of metabolic adverse effects,

An ACE inhibitor or ARB is likely to be less effective when used in combination with a beta blocker, since beta blockers reduce renin secretion and therefore angiotensin II formation.[1]

Occasionally a combination of more than three antihypertensive drugs may be required to achieve adequate blood pressure control. If patients continue to have an elevated blood pressure despite triple therapy, the possibility of secondary hypertension should be considered, although factors such as non-compliance, non-steroidal anti-inflammatory use or alcohol misuse may contribute to resistance.[4] Patients with suspected secondary hypertension need to be further investigated for the cause e.g. sleep apnoea, chronic kidney disease, Cushing's syndrome, phaeochromocytoma.

ACKNOWLEDGMENT Thank you to **Dr Sisira Jayathissa**, General Physician and Geriatrician, Clinical Head of Internal Medicine, Hutt Valley DHB, Wellington for expert guidance in developing this article.

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What's up with the men folk?

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The warfarin dilemma

Oral anticoagulation with warfarin in older people with atrial fibrillation

Atrial fibrillation and increasing age are both risk factors for stroke

Atrial fibrillation (AF) is associated with increased morbidity and mortality from stroke, thromboembolism and heart failure. AF increases the risk of ischaemic stoke approximately five-fold and the prevalence of AF increases with age. It is estimated that 5% of people aged over 65 years and 10% of people aged 80 years and older have AF.[1]

Stroke risk doubles every ten years after age 55, with over 50% of strokes occurring in people aged over 75 years.[2] Approximately one-third of strokes in patients aged 80–89 years are related to AF.[3] Evidence also shows that people with AF related strokes have a poorer prognosis when compared with people who have non-AF strokes, with larger neurological deficits, increased medical complications and higher inpatient mortality.[4] In people with non-valvular AF, anticoagulation with warfarin is more effective at reducing stroke than the antiplatelet agent aspirin.[5]

Older people with AF are at the highest risk of stroke, so they stand to benefit the most from treatment. Providing bleeding risks can be managed, the most effective treatment, i.e. warfarin, should be offered. Evidence suggests however, that warfarin is under utilised in elderly people, both in primary care and hospital practice.[6] The dilemma in older patients with AF is that the same factors indicating a need for anticoagulation with warfarin, e.g. hypertension, older age, previous stroke, are also the risk factors for intracranial haemorrhage.

Key Concepts

- Evidence suggests that warfarin is under utilised in older people
- The dilemma is that in older people with atrial fibrillation, the factors indicating a need for anticoagulation with warfarin are also the risk factors for intracranial haemorrhage
- Providing bleeding risks can be managed, warfarin is still the most effective treatment in this group of people and should be considered on an individual basis
- The decision whether to prescribe warfarin is based on an assessment of bleeding risk, stroke risk, co-morbidities, concurrent medicines and likely compliance with monitoring
- Increasing age alone is not a contraindication for warfarin use

Warfarin or aspirin?

Both warfarin and aspirin increase the risk of bleeding via different mechanisms. Warfarin requires careful monitoring and is susceptible to drug interactions which increases the hazards associated with its use compared with aspirin. However, bleeding rates in comparative clinical trials between aspirin and warfarin for AF are generally very similar, which may partly reflect close monitoring in the study situation. Until recently, clinical trials for stroke prevention in AF did not include, or were under-represented by, older people. Trials such as the Birmingham Atrial Fibrillation Treatment of the Aged Study (BAFTA) and recent review articles indicate that warfarin has significant net beneficial effects compared with aspirin, in people with AF aged 75 years and older, who are at the highest risk of stroke.[7,8] In the BAFTA trial, which included people with AF aged over 75 years, the risk of a primary endpoint (stroke, intracranial haemorrhage or arterial embolism) was significantly lower with warfarin (1.8%) compared with aspirin (3.8%), and there was no evidence that warfarin caused more bleeding complications than aspirin.[7]

The use of warfarin in older people

The decision to use warfarin in an older person requires consideration of the following:

- Risk factors for bleeding
- Tools to evaluate baseline stroke risk
- Individual assessment of the patient with regard to co-morbidities, medications and ability to comply with monitoring

Warfarin-related bleeding

The risk of bleeding while on warfarin is greatest in patients who have not previously received warfarin, and in the first 90 days of treatment.[9] A lower starting dose is recommended in older people as they are more sensitive to the effects of warfarin. Lower maintenance doses are also often required, e.g. 2–4 mg.[10] The potential for bleeding complications in older people is also increased by pathological changes that accompany ageing.

Most bleeding related to the use of warfarin occurs in the gastrointestinal tract, urinary tract, soft tissues and oropharynx with gastrointestinal haemorrhage being the most severe.[10] Patients who have an extracranial haemorrhage while on warfarin are less likely to die from the initial event or in the first month after discharge and also less likely to have long term functional deficits, than those who have intracranial haemorrhage.[11]

Although the absolute risk is relatively low at 0.2% per year, intracranial haemorrhage is the most serious complication of anticoagulation-related bleeding with a mortality rate reported of up to 50%.[5,10,11] Intracranial haemorrhage includes bleeding that is intracerebral (approximately 70%), subdural or subarachnoid and is the cause of approximately 90% of the deaths from warfarin associated bleeding.[11] Patients who initially survive an intracranial haemorrhage are likely to be discharged with significant functional deficits or to die within the first 30 days after discharge.[11]

There is good evidence that older age (>75 years), elevated INR level (>3.0), uncontrolled hypertension (e.g. systolic blood pressure > 160 mmHg) and a history of ischaemic stroke increase the likelihood of an intracranial haemorrhage. However, a previous stroke, hypertension and older age are also risk factors for ischaemic stroke.[10,12,13] Risk factors for warfarin associated intracranial haemorrhage also overlap with risk factors for spontaneous intracranial haemorrhage (see sidebar over page). However, for older people on warfarin the beneficial reduction in the risk of stroke is greater than the small increase in the risk of serious haemorrhage.[14]

Many other risk factors for warfarin associated bleeding have been investigated, however, there is conflicting evidence and often a lack of consistency in the proposed risk factors. There is some evidence to support a higher risk of bleeding complications in people with the following risk factors:[8,12,13]

- Concomitant use of aspirin, other antiplatelet medicines or NSAIDs
- Polypharmacy - seven or more medications
- Other co-morbidities e.g. diabetes, anaemia, alcohol or drug misuse, smoking, falls risk
- Patient factors e.g. Insufficient education on the use of warfarin, poor compliance, confusion

One of the most recent models that attempts to establish the risk of bleeding in older people on warfarin is the HAS-BLED Bleeding Risk Score (Table 1).[15] This model would be ideal for use in general practice. It aims to provide a rapid, simple method to predict bleeding risk. A score of three or more indicates a patient who may be at high risk of bleeding complications and who therefore may benefit from more regular review of warfarin therapy.[15]

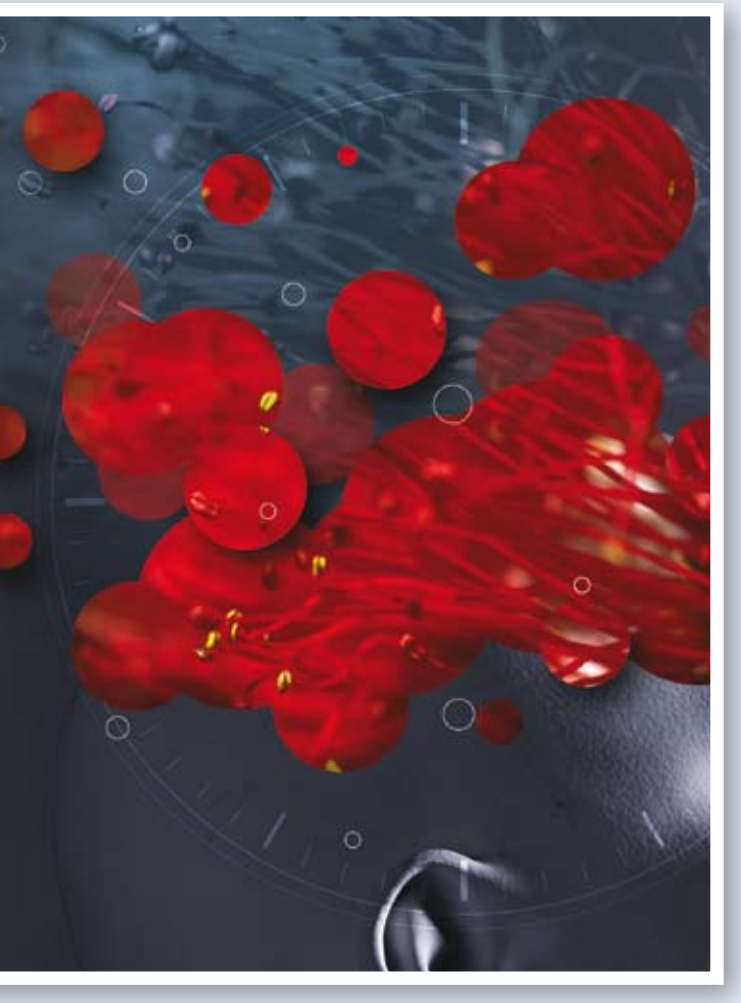
The clinical characteristics that may predict a high risk of bleeding are often thought of as contraindications to warfarin use in older people, however, the majority of these may be considered relative contraindications and will depend on individual patient characteristics and the clinical situation. In many cases the overall benefit of warfarin may still outweigh the potential risk of treatment. For patients in such clinical situations, for whom embolic risk is deemed to be high, consultation with a stroke specialist should be considered to discuss the possibility of treatment with warfarin or the use of other alternatives.

Table 1: HAS-BLED Bleeding Risk Score (adapted from Pisters et al, 2010[15])

Letter	Clinical Characteristic	Points
H	Hypertension (systolic blood pressure > 160 mm Hg)	1
A	Abnormal renal and liver function	1 point each
S	Stroke (past history)	1
B	Bleeding (previous history of bleeding or predisposition to bleeding)	1
L	Labile INRs (unstable, high or insufficient time within therapeutic range)	1
E	Elderly (> 65 years)	1
D	Drugs or alcohol (including concomitant use of aspirin, other antiplatelet agents and NSAIDs)	1 point each
		Max 9 points

Spontaneous intracranial haemorrhage [13]

The rate of spontaneous intracranial haemorrhage in people aged over 70 years is approximately 0.15% per year. Risk factors for spontaneous intracranial haemorrhage include; uncontrolled hypertension, increasing age, an underlying pathological condition e.g. tumour, infection, vascular malformation, ethnicity (increased risk in people of Asian descent), and illicit drug use e.g. cocaine, amphetamine.



Clinical situations that may be a contraindication to warfarin use include:[16,17]

- Actual or potential haemorrhagic conditions e.g. peptic ulceration (or history of within the previous six months)
- Uncontrolled or severe hypertension (blood pressure consistently above 160/90 mmHg)
- Severe renal or liver disease
- Recurrent unexplained syncope or recurrent falls
- Planned surgery
- Unsupervised dementia

Tools to evaluate baseline stroke risk

The risk of stroke can be evaluated using a risk stratification tool such as $CHADS_2$ or the updated version, CHA_2DS_2-VAS, which now includes additional stroke risk factors (see sidebar next page).[6,18] $CHADS_2$ is a simpler tool for use in general practice. These tools can be used to help decide whether to use warfarin in patients with non-valvular AF. However, they do not take into account bleeding risks, monitoring requirements and other factors that may make warfarin less suitable or potentially hazardous in a particular patient.

In general, warfarin is recommended in people at high risk of stroke ($CHADS_2$ score ≥2 or CHA_2DS_2-VAS score >1). The updated tool attempts to simplify the decision of which agent to use for anticoagulation by also recommending warfarin for patients who have a CHA_2DS_2-VAS score = 1.[18] Aspirin is still included as an option for those who score 1 but a clear preference is stated for anticoagulation with warfarin. Patients who score 0 are now considered truly low risk and although may still be prescribed aspirin, choosing not to use antithrombotic therapy may be preferred.[8] Most older people will be in a higher risk group where warfarin is the most beneficial treatment.

If warfarin is contraindicated, not indicated or is declined by the patient, aspirin may be prescribed, as it reduces the risk of stroke compared to no treatment.

The importance of INR monitoring

Appropriately monitored and dose adjusted warfarin is effective and relatively safe in elderly patients. However, warfarin may be unsuitable or hazardous in some people if they are unable to manage the treatment and its monitoring.

Monitoring of INR is important in the context of both safety and effectiveness. Interactions, diet changes and unintentional overdosing can all increase INR and bleeding risk. The ability of the patient to commit to ongoing monitoring for the duration of warfarin therapy needs to be assessed. Older people may be at higher risk of bleeding for many reasons including poor monitoring of INR.

For warfarin to be effective in preventing stroke it has been estimated that the INR should be in the target range of 2.0 to 3.0 ≥65% of the time.[8] There is no lower threshold of INR that does not accentuate the risk of intracranial haemorrhage therefore targeting a lower INR range, e.g. 1.5 to 2.0, does not reduce the risk of bleeding and is less likely to prevent stroke.[13,19]

A target INR of 2.5 within a therapeutic range of 2.0–3.0 is widely recommended for older patients. The rate of intracranial haemorrhage increases markedly in older people if the INR is >3.5 and to a lesser extent if the INR is above 3.0.[8,12] The difficulty is that although an INR above 3.0 increases the risk of intracranial haemorrhage, the majority of people on warfarin who have warfarin associated intracranial haemorrhage have been found to have an INR within the therapeutic range e.g. 2.0–3.0.[13] Minimising the risk of intracranial haemorrhage therefore requires not only good control of anticoagulation but also effective management of other modifiable risk factors, particularly hypertension.[13]

Individual patient assessment is essential

An individual assessment of the patient with regard to co-morbidities, medications and the ability to comply with monitoring is essential for the safe use of warfarin.

Stroke assessment tools for patients with AF[6,18]

The updated stroke assessment tool CHA_2DS_2-VAS puts greater emphasis on increasing age (≥75 years) and also incorporates additional risk factors for stroke – female gender, age group 65 – 75 years and a history of vascular disease e.g. myocardial infarction, peripheral arterial disease. Scores for each tool are calculated as follows:

$CHADS_2$	Score
Congestive heart failure	1
Hypertension	1
Age 75 years or older	1
Diabetes mellitus	1
Previous **S**troke or TIA	2
Maximum score	**6**

CHA_2DS_2-VAS	Score
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age ≥75 years	2
Diabetes mellitus	1
Stroke/TIA	2
Vascular disease (prior MI, peripheral vascular disease)	1
Age 65–75 years	1
Sex category (i.e. female gender)	1
Maximum score	**9**

N.B. The maximum score for CHA_2DS_2-VAS is 9 as only one age score is used in the calculation.

Co-morbidities may be risk factors for bleeding and they may also increase the potential for falls.

A review of medicines is recommended to avoid concomitant use of those that may increase the risk of bleeding e.g. aspirin, NSAIDs, Cox-2 inhibitors, dipyridamole. In addition bleeding risk should be reassessed when new medicines, including those used for short periods such as quinolone or macrolide antibiotics, are introduced.

Before initiating warfarin the possibility of non-adherence and monitoring should be considered. Factors to take into account may include any cognitive impairment, mental illness or an inability to access services.

Warfarin interacts with multiple other prescriptions medicines, as well as nutritional supplements, over-the-counter medicines and some foods e.g. foods that contain high levels of vitamin K such as broccoli, spinach and cabbage. Discuss the possibility of these interactions with patients and encourage them to consult about any major dietary changes they are planning to make. Also inform patients that their general wellbeing may also affect their warfarin therapy, e.g. a new illness such as fever or diarrhoea or a condition such as congestive heart failure. Poor quality patient education has been found to be a significant risk factor for both ineffective anticoagulation and warfarin associated bleeding in older patients.[12]

Bottom-line

Increasing age alone should not prevent the use of warfarin. The decision to use warfarin involves identification and assessment of those patients who are at high risk of ischaemic stroke without warfarin and weighing this against the risk of intracranial haemorrhage with warfarin treatment.[11] Further research that includes older participants and the ongoing development of risk assessment tools are first steps toward solving this problem.

Once the decision to use warfarin is made on an individual basis, prevention of bleeding complications relies on maintaining an INR between 2.0 and 3.0, appropriately monitoring and adjusting doses as required, providing quality patient education and effectively managing any modifiable risk factors.

ACKNOWLEDGMENT Thank you to **Dr Anna Ranta**, Lead Stroke Physician, Consultant Neurologist and Head of Department, Department of Neurology and Acute Stroke Services, Midcentral DHB, Palmerston North and Associate Dean of Undergraduate Studies, University of Otago, Wellington for expert guidance in developing this article.

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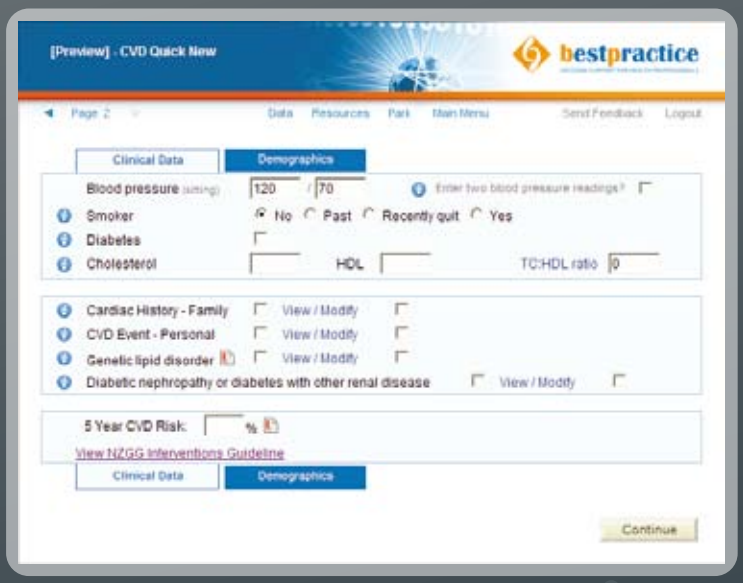

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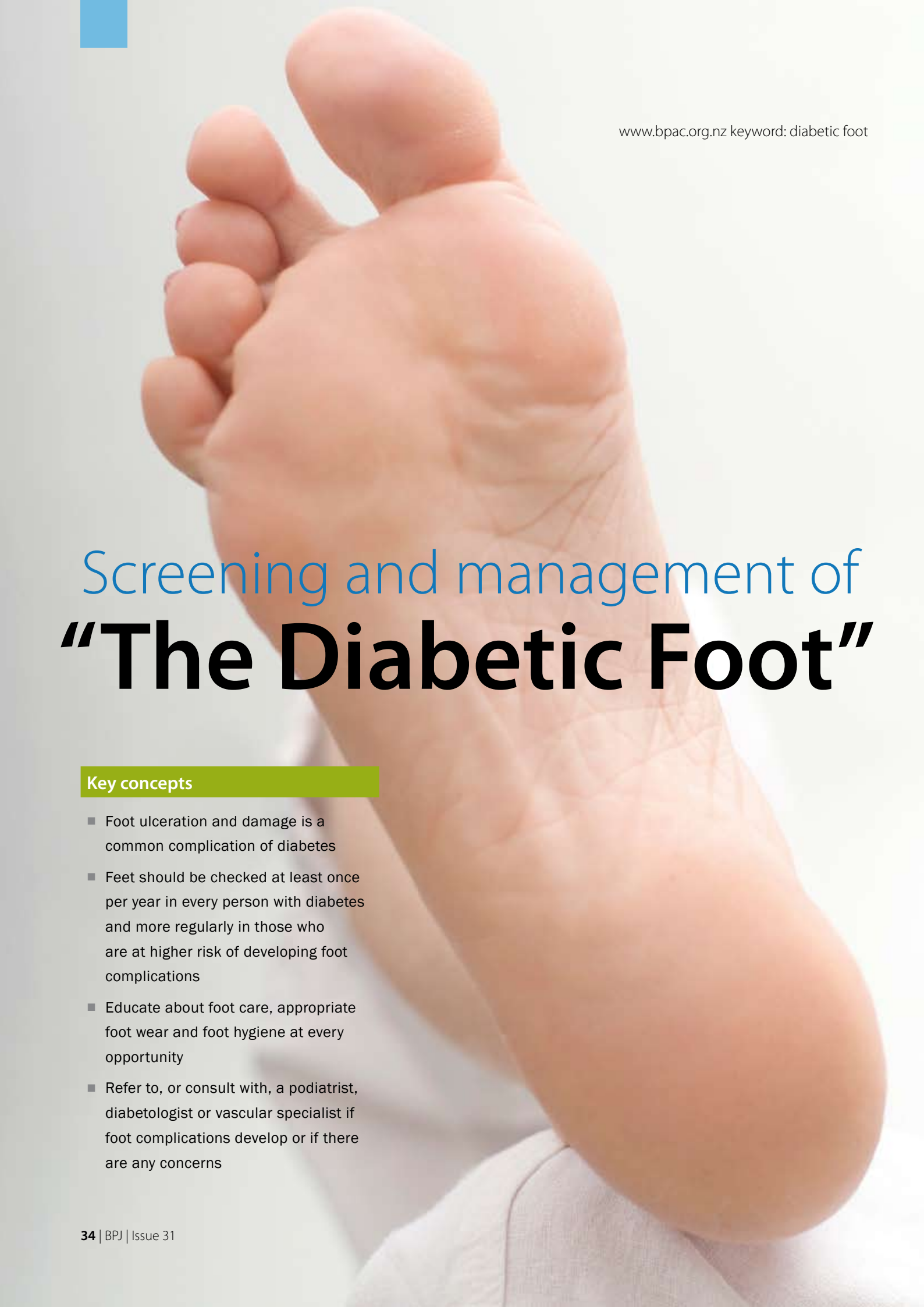

Screening and management of "The Diabetic Foot"

Key concepts

- Foot ulceration and damage is a common complication of diabetes
- Feet should be checked at least once per year in every person with diabetes and more regularly in those who are at higher risk of developing foot complications
- Educate about foot care, appropriate foot wear and foot hygiene at every opportunity
- Refer to, or consult with, a podiatrist, diabetologist or vascular specialist if foot complications develop or if there are any concerns

Diabetes in New Zealand – a growing concern

In 2000 the Ministry of Health acknowledged that diabetes was a major concern in New Zealand and a range of preventative measures and increased treatment options were introduced.[1] At that time there were approximately 125,000 people with Type 2 diabetes in New Zealand and it was projected that this number would increase to 180,000 in 2011.[1] This estimate has already been far exceeded with approximately 270,000 people in New Zealand (5 – 7% of the population) currently diagnosed with diabetes.[2]

The Ministry of Health has labelled this the "Diabesity" epidemic, relating the significant rise in the number of people with Type 2 diabetes to the rising rates of obesity. People of Māori, Pacific or Indo-Asian ethnicity are two to three times more likely to have diabetes and this is a major contributor to increasing health inequalities.[2]

Interventions to reduce morbidity and mortality from diabetes focus on education, prevention and early detection of diabetes and its complications.

"Get Checked" yearly to prevent diabetes complications

Part of the Ministry of Health's "Diabetes 2000 Initiative" was the implementation of the annual, free "Get Checked" programme. The goal of the programme is to increase intervention before more serious complications of diabetes develop.

The "Get Checked" annual health review includes:

- A HbA_{1c} level
- Blood pressure, lipid profile, height and weight
- Kidney function (microalbuminuria)
- Assessment of peripheral circulation and sensation of the feet
- Retinal check (at least every two years)
- Follow-up plan for care

Information for the annual review can be collected throughout the year, or alternatively as a more formal "one-off" process.

An annual check for people with diabetes is also a PHO Performance Programme (PPP) indicator.

> The PPP goal is for at least 80% of all people with diabetes enrolled in a practice to have an annual diabetes review.

"The Diabetic Foot" is a common complication of diabetes

Peripheral neuropathy and arterial disease are common complications of diabetes and are the main risk factors for the development of ulcers, infection and ultimately lower extremity amputation.[3, 4]

Neuropathy results in ulcer formation and other foot complications by decreasing pain sensation and perception of pressure. This also causes muscle imbalance that leads to foot deformity and impaired microcirculation and integrity of the skin.[5]

A foot affected by neuropathy is described as warm, dry and numb,[3] although sensory neuropathy can be very painful. Pain, burning and tingling that is especially worse at night and relieved by getting up and walking, is highly suggestive of diabetic peripheral neuropathy.

A foot affected by peripheral arterial disease is described as cold and without detectable pedal pulses. The patient often experiences pain when walking or if severe, at rest. Once ulcers form, the capacity for them to heal is compromised by diminished blood flow in the foot.[5] Wounds can deteriorate rapidly and patients are at increased risk of developing serious infection.[3]

These syndromes are collectively referred to as "The Diabetic Foot" and this is one of the most common complications occurring among people with diabetes in New Zealand.[3]

Management of "The Diabetic Foot" in primary care focuses on:

1. Regular (at least yearly) screening for foot problems in people with diabetes to prevent ulcer formation
2. Prompt treatment and referral, if required, for any detected foot problems
3. Education about preventing foot problems from occurring or worsening

Screening for diabetic foot complications

Foot checks should begin immediately after a person has a confirmed diagnosis of type 2 diabetes and at least yearly thereafter (as part of an annual diabetes review). If the patient has characteristics that increase their risk of foot complications (see opposite page) or once evidence of diabetic foot complications has been detected, feet should be checked every three to six months.[3] Some patients at very high risk of foot damage e.g. loss of feeling in the foot, no detectable pedal pulses or active ulceration may be considered for review even more frequently, i.e. every one to three months. These recommendations are summarised in Table 1.

Table 1: Recommended frequency of examination for diabetic foot complications

Stage of progression	Recommended frequency of foot check
Confirmed diabetes	As soon as possible after diagnosis, annually thereafter
First signs of foot problems identified or patient at high risk	Every three to six months
Active ulceration and infection or very high risk	Regular assessment until active problems resolved, then every one to three months

Patients who have the following characteristics are at high risk of developing foot complications:[3, 4]

- Peripheral neuropathy
- Peripheral arterial disease
- Previous foot ulceration or amputation
- Structural foot deformity
- Plantar callus
- Older age (> 70 years)
- Māori or Pacific ethnicity
- Longer duration of diabetes
- Smoking
- Other diabetic complications e.g. retinopathy
- Renal impairment
- Continual use of inappropriate footwear
- Living in a lower socioeconomic area

Performing a foot check[3-5]

1. **Examine the foot** to identify deformity e.g. abnormal foot shape, clawed or hammer toes, ulceration, skin abrasions, erythema, swelling and pressure points. Assess the skin status i.e. colour, thickness, dryness, cracking. Check if the foot is fixed or flexible by asking the patient to stand and observe whether the toes straighten. Assess how well the patient cares for their feet by checking for cleanliness and trimmed nails. Examine carefully between the toes for tinea pedis. Check whether the patient can both reach and see their feet.

2. **Ask the patient** if they experience numbness or pain, including what type of pain e.g. burning, tingling, and at what times e.g. walking, resting, day-time, night-time. Ask about the normal temperature of the foot.

3. **Assess for neuropathy** using a 10 g monofilament (see sidebar over page). A vibration test, using a 128 Hz tuning fork or a biothesiometer may also be performed. Absent touch pressure, pin prick or vibration sensation (in a "stocking distribution"), absent ankle reflexes, altered temperature sensation and dry, scaly skin are suggestive of neuropathy.

4. **Assess peripheral circulation** with thorough palpation of pedal pulses (dorsalis pedis and posterior tibial). If there are no palpable pulses, and if a Doppler machine is available, calculate ankle brachial index (see sidebar over page) or consider referral to a vascular specialist (see sidebar Page 40). Absent pulses, calf claudication, absence of hair on the feet, altered temperature (a cold foot) and thin, bluish skin are suggestive of peripheral arterial disease.[6] A bounding, easily detected pulse in a warm, dry foot is suggestive of autonomic neuropathy, which causes abnormal arterio-venous shunting.

Best Practice Tip: Regular callus removal should be performed in people with diabetes and neuropathy. Calluses may hide underlying pressure ulcerations of the skin. It is recommended that patients at risk of diabetic foot complications are referred to a podiatrist for removal of calluses.

Classifying risk of ulceration[4]

Normal sensation, palpable pulses, no deformity	**Low current risk**
Evidence of neuropathy, absence of pedal pulse(s)	**Increased risk**
Evidence of neuropathy, absence of pedal pulse(s) and skin changes or deformity	**High risk**

Calculating ankle brachial index[7]

Equipment: Blood pressure cuff and hand-held Doppler machine

1. Take the blood pressure in the arm (brachial pressure)
2. Take the blood pressure in the ankle using the Doppler machine (ankle pressure)
3. Calculate ankle brachial index by dividing systolic ankle pressure by systolic brachial pressure e.g. ankle pressure is 120 mmHg and brachial pressure is 132 mmgHg, ankle brachial index is 120/132 = 0.9

Normal	0.9 – 1.2	Risk of vascular foot ulcer is small
Definite vascular disease	0.6 – 0.9	Risk of vascular ulcer moderate, depending on other risk factors
Severe vascular disease	Less than 0.6	Risk of vascular foot ulcer very high

Ankle brachial index may not be able to be reliably calculated in some people with diabetes as the arteries in the ankles may be calcified.

Treating "The Diabetic Foot"

Lesions and ulcers detected during a foot check should be initially treated and any pain managed. It is recommended that patients identified as being at increased risk of serious foot complications are then referred to a specialist multi-disciplinary team for further management and care.[3, 4]

Urgent referral to secondary care (within 24 hours) should be considered if:[4]

- An ulcer shows no signs of healing or becomes necrotic
- Significant swelling is present
- Discolouration of part or all of the foot is present
- There is suspicion of bone or joint involvement

Treatment of ulcers

Clean, debride and dress the wound

The wound may be cleaned, e.g. with saline, to remove surface bacteria and to allow assessment of swelling, redness and discharge.

Surgical (using a scalpel or tissue nippers), mechanical (using saline and gauze) or hydrogel debridement (applying a gel polymer dressing to the wound) can be used to remove non-viable or necrotic tissue, although this is not recommended in the primary care setting when the debridement area is extensive. Surgical debridement is not recommended when sensation to the foot is intact.[8, 9] There is limited evidence that hydrogel debridement increases the healing rate of ulcers compared to gauze dressings or standard care.[10] Hydrogel may also be preferable in the case of a painful ulcer.[9] Care must be taken to mask the edges of the wound, so surrounding tissue is not damaged.[10]

The ulcer should be kept clean and moist but free of excessive fluids.[9] There is no evidence that one type of dressing is superior to another for wound healing in diabetic foot ulcers. Dressings should be chosen based on their comfort and durability when worn inside footwear, their ability to absorb exudate without plugging the wound

Performing a test using a monofilament[3]

A test using a 10 g monofilament is the recommended method for assessing for neuropathy of the foot. Loss of protective sensation at any site on the foot indicates evidence of neuropathy, increasing the risk of ulceration and other complications.

Equipment: 10 g monofilament

Method:

1. Place the patient in a supine position with shoes and socks removed
2. Show the filament to the patient and bend it against their arm to illustrate that it is not painful
3. Ask the patient to close their eyes and to say "yes" when they feel the filament on their feet. Do not prompt the patient by asking "Did you feel that"?
4. Place the filament on one of the designated sites on the foot (Figure 1), press it against the skin until the patient indicates they can feel it, or a C shape is formed, and then lift it off. This should take approximately three seconds.
5. Repeat this sequence at each of the designated sites on the feet and record findings
6. Repeat again in the areas in which the patient did not indicate feeling the monofilament
7. If evidence of neuropathy is detected, further assessment is required

Tips:

- Avoid tapping the filament against the skin or using rapid movements
- Choose the sites on the foot at random and try not to test sites in a predictable pattern that will allow the patient to anticipate when and where the monofilament is likely to be positioned next
- Do not apply the filament directly on an ulcer, callous, scar or necrotic tissue. Apply the filament on near-by normal tissue.
- The filament should be cleaned after use with an alcohol swab or dilute bleach solution and returned to its case
- Filaments should not be used for more than ten patients in 24 hours, as they may buckle

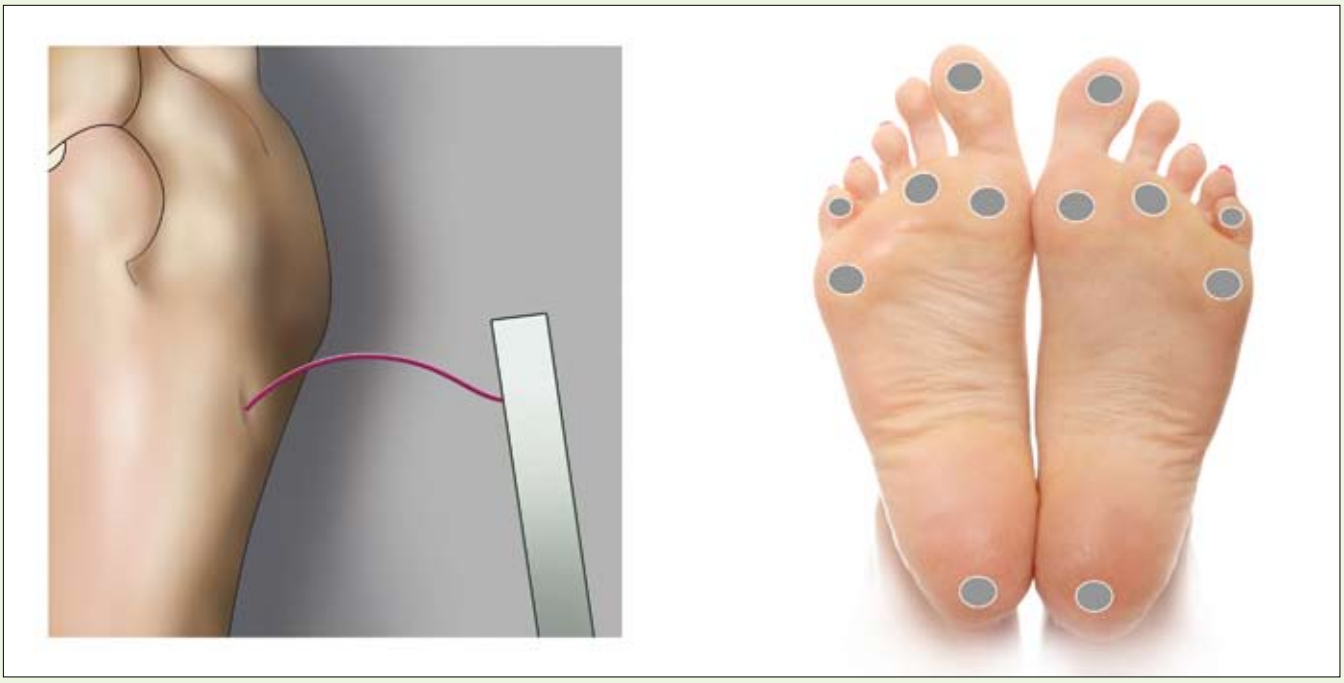

Figure 1: Monofilament bent to form a C shape. Recommended sites for cutaneous sensory pressure perception testing using a monofilament.[3]

Referral criteria for vascular review

Criteria for referral to a vascular surgeon for a patient with a diabetic foot complication includes the following:

- Foot lesion (ulcer, gangrene) or suggestion of rest pain with peripheral arterial disease
- Deteriorating ulcer with known peripheral arterial disease or absent pedal pulses
- Ankle Brachial Index <0.5 or absolute ankle pressure <50 mmHg
- New foot lesion with previously treated peripheral arterial disease
- Symptomatic intermittent claudication at <200 m
- Acute diabetic foot sepsis
- Osteomyelitis of forefoot or metatarsals
- Acute osteomyelitis

Osteomyelitis

Osteomyelitis is common in infected diabetic foot ulcers. Its presence greatly increases the risk of lower extremity amputation. A probe can be inserted into the wound to check for bone involvement (a probe-to-bone test). A non-healing ulcer, deep ulceration, extensive tissue loss, recurrent ulceration, previous osteomyelitis affecting the same bony region or a history of discharge of bony fragments from an ulcer raises the likelihood of osteomyelitis being present. Visible or palpable bone or joint structures make osteomyelitis a likely diagnosis. Referral to a multidisciplinary specialist team is strongly recommended.

and the ease with which they can be regularly removed for checking the wound.[4, 9]

If the wound does not appear to be infected, a long-term waterproof dressing can be applied and left in place for up to one week before review. If the wound shows signs of infection, a non-adherent dressing can be applied and reviewed every one to two days.

Off-load pressure from the foot

The central principle for healing any neuropathic ulcer is the reduction of pressure through pressure redistribution (off-loading) until healing occurs. This involves resting the foot and using therapeutic footwear. If adherence to treatment is problematic, some specialists may use a total contact cast to reduce pressure on the foot and allow more rapid healing.[9]

Graduated compression therapy (i.e. compression bandages or stockings) has an important role in healing and management of venous leg ulcers and mixed aetiology venous ulceration, in people with diabetes and longstanding venous incompetence. However, it does not usually have a role in healing neuropathic or arterial ulcerations associated with the diabetic foot and may in fact worsen the condition. Specialist advice is recommended before considering the use of graduated compression therapy in a person with diabetic foot complications.

Consider antibiotics

If the wound shows signs of infection e.g. erythema, oedema, foul odour or purulent discharge, antibiotic treatment is indicated, either orally or intravenously (IV).

Consider admission to hospital for IV antibiotics for patients with extensive infection or where osteomyelitis is suspected (see sidebar).

When treating the infection in the general practice setting, a broad-spectrum antibiotic such as amoxicillin clavulanate 500/125 mg, three times per day, for five to ten days, may be used (as the infection is most likely to be polymicrobial). Alternative agents are cefaclor or co-

trimoxazole plus metronidazole. Swabbing the wound for microbiological analysis is usually not necessary but can be helpful if the infection shows no sign of healing with the current antibiotic regimen.[3, 4]

Monitor, review and consider referral

Regular review of the patient is encouraged. An infected wound should be reviewed and re-dressed every one to two days. Note the size of the ulcer and whether it is decreasing.[9]

Check that the patient is following instructions for care and that they have removed pressure from the infected area. If the ulcer shows no signs of healing or if infection is still apparent after antibiotic treatment, then referral to a specialist team is strongly recommended.

Referral for vascular assessment is strongly recommended if limb ischaemia is present and compromising wound healing. This can be corrected through revascularisation procedures.

Sub-optimal treatment can have serious long-term consequences for the patient. Referral to a specialist multidisciplinary team for wound care and off-loading pressure can be considered with any diabetic foot complication to improve healing times and patient outcomes.

Treatment of painful neuropathy

Pain associated with neuropathy is a common feature of diabetic foot complications. Neuropathic pain may be characterised by altered pain sensation, numbness, burning or spontaneous pain.[12]

Treating neuropathic pain can be challenging and there is a lack of clear consensus as to which medicines to use and in what order.[12] Treatment should be tailored to individual circumstances and take into account factors such as the severity of pain, coping strategies and lifestyle/occupational restrictions, e.g. a requirement to operate heavy machinery would preclude using sedating medicines during the day.

Charcot's osteoarthropathy

Charcot's osteoarthropathy (or neuroarthropathy) is associated with severe peripheral neuropathy. It is a progressive condition characterised by collapse and destruction of joints, fractures and bone destruction. In people with severe diabetic neuropathy, Charcot's osteoarthropathy most commonly manifests as acute swelling and deformity of the foot (without open ulceration), leading to collapse of the pedal arch. This is a major risk factor for ulceration and subsequent amputation of the foot.[4, 11]

Acute Charcot's osteoarthropathy can be confused with cellulitis, acute gout, osteomyelitis and abscess. In a patient with a long duration of diabetes, a history of poor glycaemic control and peripheral neuropathy and no history of open ulceration, Charcot's osteoarthropathy should be considered first.[11]

People with suspected Charcot's osteoarthropathy should be referred immediately for assessment and x-ray. Management includes total contact casting and immobilisation of the joint. Bisphosphonate treatment is sometimes considered.[4, 11]

After beginning any medicine (or medicine regimen) for treating neuropathic pain, the following aspects should be regularly reviewed:[12]

- Pain control
- Adverse effects
- Mood
- Daily functioning
- Sleep patterns

Consider dose adjustment or adding or substituting another medicine if optimum control of these factors is not being achieved.[12]

First-line pain management

Paracetamol may be trialled as first-line management for neuropathic pain and may be continued throughout any regimen.

Second-line pain management

If paracetamol alone is not adequate for controlling pain, a tricyclic antidepressant (TCA) may be added to the regimen (or paracetamol substituted for a TCA).

Nortriptyline is the preferred TCA for neuropathic pain, due to fewer adverse effects than other TCAs. Initiate nortriptyline at 10 mg per day (usually taken at night) and titrate dose upwards until pain is controlled. The dose should not usually exceed 75 mg.[12]

Third-line pain management

If second-line pain management is insufficient, an anticonvulsant may be added to the treatment regimen, or the TCA substituted for an anticonvulsant. Referral to, or discussion with, a pain specialist can be considered.

Carbamazepine and sodium valproate are both effective for neuropathic pain. Gabapentin has also traditionally been used for neuropathic pain but recent evidence suggests that it has limited effectiveness for this indication.[13]

 For more information see "New evidence shows less benefit of gabapentin for neuropathic pain" Snippets, BPJ 28 (June, 2010).

Carbamazepine may be initiated at a dose of 100 mg per day. Increase the dose slowly until pain is controlled, to avoid adverse effects such as nausea, vomiting and dizziness. Regular monitoring is required.

Opioids such as methadone or oxycodone may have a limited place in the treatment of neuropathic pain but their use is not advised unless in consultation with a specialist in pain management.[12]

Adjuvants

Capsaicin cream and local anaesthetic gels may be trialled throughout a treatment regimen for neuropathic pain, They should not be applied to broken/ulcerated skin.

 For more information about treating neuropathic pain, including considerations for specific patient circumstances, see "Pharmacological management of neuropathic pain", BPJ 16 (Sept, 2008).

Preventing diabetic foot complications

The two main factors in preventing diabetic foot complications are:

1. Maintaining optimal control of risk factors
2. Educating about appropriate care of the feet

Optimal control of risk factors

The development of peripheral vascular disease and neuropathy, leading to foot complications, may be able to be avoided or delayed with optimal management of diabetes and cardiovascular risk factors. This includes:

- Maintaining good glycaemic control – establish an individualised HbA_{1c} target ("HbA_{1c} targets in people with type 2 diabetes" BPJ 30, Aug 2010)

- Managing hypertension – New Zealand cardiovascular guidelines recommend reducing blood pressure to < 130/80 mm Hg for people with diabetes, however this level may not be achievable for some people. In the presence of microalbuminuria or renal disease more aggressive control may be required to reduce blood pressure to < 125/75 mm Hg.[14]
- Reducing blood lipid levels – aim for a reduction towards the target level of total cholesterol < 4.0 mmol/L,[14] although this level may not always be achievable ( "An update on statins" BPJ 30, Aug 2010)
- Smoking cessation – provide advice and treatment options on how to quit
- Weight management – promote exercise and healthy diet

Educating about foot care

The three main aspects of foot care education have been identified as foot hygiene, awareness of fungal infections and appropriate actions required for skin injuries.[4] There is conflicting evidence on the effectiveness of educational interventions on reducing the occurrence of foot ulceration, and which methods are best.[15] Education is likely to be effective in the short-term, but messages must be periodically reinforced for longer-term behavioural change.[4]

Providing advice about foot care

The following points can be discussed with patients in regards to the care of their feet:[4]

- Clean and thoroughly dry feet (including between the toes) every day
- Moisturise areas of dry skin and apply sun-screen if feet are exposed to the sun
- Inspect feet every day for abrasions, blisters, ulcers, redness, swelling or calluses
- Inspect between the toes for any signs of fungal infection

Considerations for Māori and Pacific people with diabetic foot complications

Māori and Pacific people with diabetes are at high risk of diabetic foot disease.

For Māori, tapu and noa are key concepts that underpin many practices. It is important to keep things that are tapu (restricted) separate from things that are noa (unrestricted). In many cases these concepts or tikanga, align with good health and safety practice.

 **Best Practice Tip:** Become familiar with the basic principles of tapu and noa, and practical ways of respecting these concepts. For example:

- For many Māori, it is inappropriate for their feet to be placed on a pillow, which is also used for the head. Avoid propping feet up with a pillow during a foot examination or treatment.
- Māori may prefer their nail clippings and any other body parts (regardless of how minor it is perceived to be) to be returned to them for disposal - ask them.
- Many Māori remove their footwear before entering their house or marae. Encourage the use of slippers or socks to protect feet when inside, if outdoor shoes are considered unacceptable.

Referral criteria for podiatry services

Community diabetes specialist podiatrists hold contracts with their DHBs in most regions around New Zealand and undertake primary care podiatry screening, assessment and treatment for the management of diabetic foot complications.

Secondary care hospital-based podiatrists are employed in most hospitals and can receive referrals for the acute management of diabetes-related complications.

Contact your local DHB for details of funding for these services and referral criteria. In many areas, people with diabetes related foot complications are able to access fully-funded podiatry services including supply of customised therapeutic footwear and orthoses.

Ministry of Health criteria for podiatry referral for people with diabetes related foot complications[17]

At risk foot (criteria for referral to community-based podiatry services)	High risk foot (criteria for referral to secondary care-based podiatry services)
▪ Neuropathic ulceration ▪ A positive history of diabetic foot ulceration (and no current ulceration) ▪ Neuropathic foot with absence of protective sensation (patient cannot detect the 10 g monofilament at four or more testing sites) ▪ Biothesiometer threshold >25 V ▪ Change to circulation and/or sensation with other risk factors present (see below) ▪ Neuropathy, musculoskeletal deformity and pre-ulcerative lesion **Risk factors:** Long standing diabetes Elevated HbA_{1c} Visual impairment Hypertension Dyslipidaemia Impaired mobility Perception of risk Nephropathy Poor glycaemic control Smoking Obesity Social isolation Male> 40 years	▪ Past history of gangrene or amputation ▪ Peripheral vascular disease including: – Absent pedal pulses and a history of claudication – Ankle brachial index at 0.5–0.8 (indicating impaired arterial flow) ▪ Night pain ▪ Pre-ulcerated or ulcerated ischaemic lesion **URGENT referral to secondary care** ▪ Neuropathic or neuro-ischaemic ulcers that have not demonstrated significant measurable improvement (30–40%) within four weeks of treatment ▪ Ulcers presenting at > Grade 2 or indolent Grade 1 (graded by podiatrist) ▪ Cellulitis ▪ Systemic signs of infection ▪ Infection not responding to oral antibiotic therapy ▪ Radiological or clinical evidence of bone involvement including active Charcot's neuroarthropathy

- Keep toenails trimmed, do not use "corn remover", seek advice from a podiatrist about the treatment of corns or calluses
- Break in a new pair of shoes gradually, by first wearing for only an hour at a time
- Regularly inspect the inside of shoes for tears, sharp edges or foreign objects
- If neuropathy is present, extra vigilance is needed to avoid burns – check bath temperature, avoid hot water bottles, electric blankets or foot spas
- Seek medical attention if any changes to the foot, abrasions or injuries are detected or pain or numbness develop

Organisations such as Diabetes New Zealand have websites with downloadable patient information and resources that can be helpful to reinforce advice: **www.diabetes.org.nz**

Due to limited mobility or visual impairment, many people will be unable to adequately inspect and care for their feet. Discuss methods to help self-examination such as the use of a mirror or the possibility of a family member or carer being involved in regular foot care.

Selecting appropriate footwear

One of the most important aspects of preventing diabetic foot complications is wearing appropriate footwear. Patients should be advised to always wear well-fitting, cushioned footwear (including slippers) to protect their feet from injuries. Loose-fitting or open-toed footwear such as gumboots, jandals or sandals, and going barefoot should be avoided.

Patients (especially those at high risk) can be custom-fitted with specialised shoes and orthoses (insoles) by a podiatrist. Specialised shoes for people with diabetes are usually made with extra depth and room to accommodate foot deformities and orthoses. They have increased cushioning and reduce the pressure on certain parts of the foot, therefore reducing the potential for ulcers to occur.[16]

Non-customised, specialised shoes are available "off-the-shelf" and are generally the same price as cushioned, high-quality sports shoes, which are also an option. There is a lack of evidence of the superiority of custom-made therapeutic footwear to off-the-shelf varieties in reducing the occurrence of ulcers.[6] It appears that wearing a well-fitted, cushioned pair of shoes, at all appropriate times, is more important than the actual type of shoe.

Orthoses (specially made insoles) can provide cushioning and redistribution of pressure loading. They may be worn in specially designed or regular shoes.[16]

Socks and other hosiery should be well-fitted – neither too tight (leading to decreased circulation) nor too loose (leading to rubbing and abrasions). Padded hosiery may protect the feet, reduce plantar pressure and reduce calluses.[16] Socks made from a breathable fabric such as cotton are preferable to those made from other fabrics.[5]

ACKNOWLEDGMENT Thank you to **Associate Professor Geoff Braadvedt**, Physician and Endocrinologist, Department of Medicine, University of Auckland and **Angela Bayley**, Diabetes Specialist Podiatrist, Orthotics Centre, Wellington for expert guidance in developing this article.

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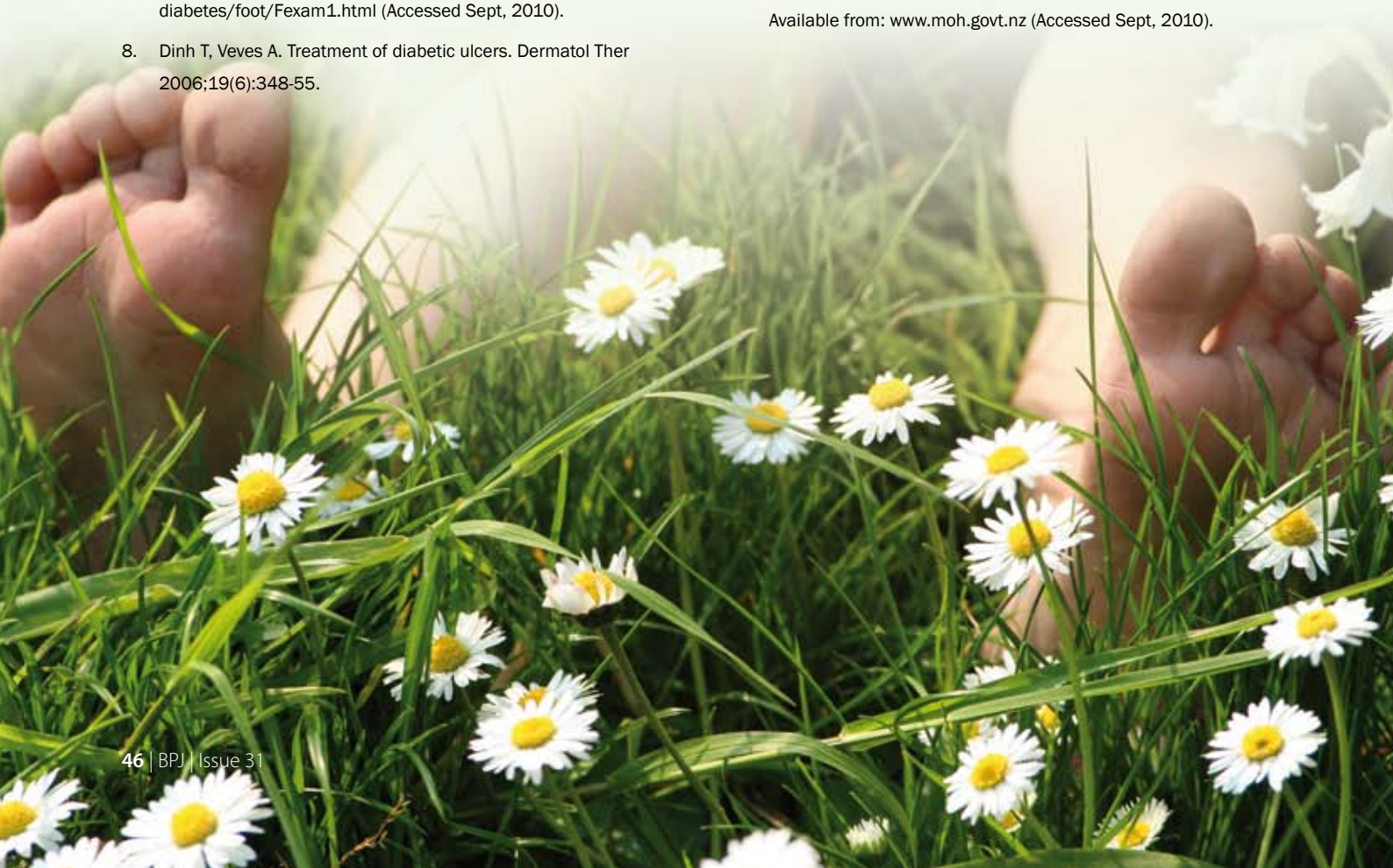

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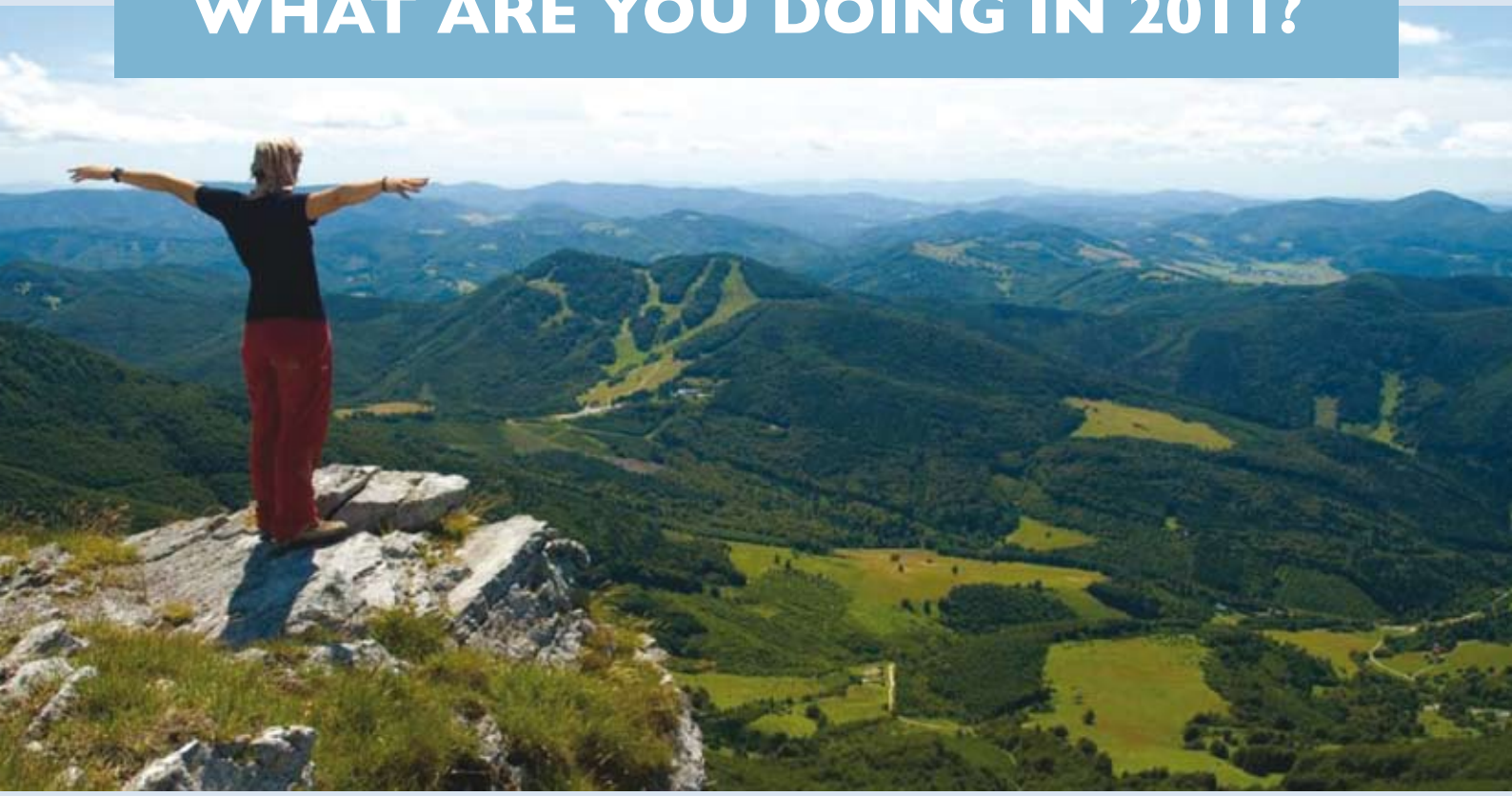

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Defining non-blanching rash

Dear bpac,

I am concerned about the recommendation in Best Practice Journal to refer a child with non-blanching rash immediately to hospital ("Identifying the risk of serious illness in children with fever, BPJ 29, July 2010).

I have seen many children presenting with a "non-blanching rash" and a lot of parental anxiety with it. The rashes I have seen may be due to eczema, skin infections and viral exanthems. Even pressure marks can be described as a non-blanching rash.

This term is too misleading and vague. Let's use the much more specific term haemorrhagic rash which GPs are quite capable of recognising and referring appropriately.

Dr Lynley Brown, GP
Gore

The NICE guideline which was used as the basis for this article is specifically aimed at recognition of serious illness in children with fever. The guidance states that a child with fever, who has a non-blanching rash, is classified as being at high risk of having a serious illness and it is recommended that they are referred to secondary care for further assessment.[1]

You are correct in stating that a non-blanching rash in this context is more accurately defined as a haemorrhagic rash. It is expected that clinical judgement would be used in interpreting this guidance and it is not intended that a child with a non-blanching rash and no signs of fever, e.g. as may be seen in eczema, would be referred immediately to secondary care.

Education about the signs of meningitis has increased awareness among parents of the potential importance of a non-blanching rash. Parents are encouraged to have their child assessed by a health professional if they have signs of a non-blanching rash, and in the majority of cases they can be reassured and appropriate advice and education given. GPs are likely to see large numbers of children with non-specific, non-blanching rashes, for every child who presents with symptoms and signs of serious illness.[2]

The difficulty for parents, and sometimes even for GPs, is in determining which of these children has a serious illness and which do not. A study in the UK reported that 11% of children who presented with a non-blanching rash had meningococcal infection and a further 5% were diagnosed with another serious illness such as Henoch Schonlein purpura, idiopathic thrombocytopenia and acute leukaemia.[3]

Any non-blanching rash in a child who is feverish and appears unwell is an important marker of serious illness and parents should continue to be encouraged to seek medical attention for their child. It is up to the clinician to interpret the significance of the rash in the context of other symptoms and decide whether further assessment is required

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Lung age for smoking cessation

Dear bpac,

I notice that you make reference to "lung age" in an article in Best Practice Journal ("Telling smokers their lung age increases their chance of quitting", BPJ 13 May 2008).

The idea is that it reflects damage done to the lung due to the smoking habit, and that confronting smokers with their "lung age" will increase quit rates. Dr. Paul Enright and I have reviewed this issue. First of all, the term "lung age" is a misnomer, as healthy lifelong non-smokers can have ludicrous lung ages. Secondly there is no convincing evidence from the literature that confronting smokers with their "lung age" increases quit rates in smokers. This is relevant information for the general public and the medical profession.

The above is reviewed in: Quanjer P, Enright P. Should we use 'lung age'? Prim Care Resp J 2010;[Epub ahead of print]. Available from: www.thepcrj.org/journ/aop/RHI-032-10.pdf

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The use of lung age as a way of encouraging people to quit smoking is indeed contentious. It is agreed that the supporting evidence is relatively weak, and that there are several factors that may confound the estimation of lung age.

The "snippet" in BPJ 13 was based on the study by Parkes et al[1] in which smokers underwent spirometry. The intervention group were given an estimate of their lung age, and the control group were given a simple spirometry reading. Smoking quit rates were higher in the intervention group compared with the control group. However, in the intervention group, quit rates were similar between those with "higher lung age" and those with "normal lung age". This suggests that factors other than telling people their lung age might have also influenced quit rates.

For some individuals, lung age might be a useful motivating strategy, in addition to other smoking cessation methods. However, it does appear that on a population basis, the use of "lung age" may not be helpful.

Reference:

1. Parkes G, Greenhalgh T, Griffin M, Dent R. Effect of smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. BMJ 2008;336(7644):598-600.

Iodine supplements for goitre?

Dear bpac,

I was interested to read in Best Practice Journal ("Iodine supplements now funded" BPJ 30, Aug, 2010) about the newly funded iodine supplement, NeuroKare, that the Ministry of Health has recommended be used in pregnancy and lactation. What I wish to know is whether I should be considering using these supplements in most of my non-pregnant patients (if we are assuming that our soils are iodine deficient)? Also, should I be using iodine supplements in my patients with mild goitre but normal thyroid function?

GP, South Island

Iodine has been recognised for some time now as being an important trace element. Due to its low content in local soil, it is difficult to achieve adequate levels of iodine in New Zealand. As a public heath measure, iodine was added to salt, and more recently iodised salt is now required to be used in most commercially available breads.

Iodine supplements could in theory be taken by non-pregnant patients who may be iodine deficient, but it would

be more appropriate to advise these patients to enhance their dietary iodine intake by the use of iodised salt, bread, seafood, etc, much in the same way as patients are generally advised to achieve their recommended vitamin C intake through diet rather than by supplementation.

Euthyroid goitre (simple, non-toxic goitre) is a non-cancerous hypertrophy of the thyroid without hyperthyroidism, hypothyroidism or inflammation. Except in severe iodine deficiency, the thyroid function is normal and patients are asymptomatic apart from an obviously enlarged, non-tender thyroid.[1] The diagnosis is made clinically along with normal thyroid function.

Euthyroid goitre is most frequently noted at puberty, during pregnancy and at menopause. The cause at these times is usually unclear. Known causes include intrinsic thyroid hormone production defects and, in iodine-deficient countries, ingestion of foods that contain substances that inhibit thyroid hormone synthesis such as raw brassicas e.g. broccoli, cauliflower or cabbage. Other causes include the use of drugs that can decrease the synthesis of thyroid hormone e.g. amiodarone, lithium.

Treatment of euthyroid goitre is directed at the underlying cause, but partial surgical removal may be required for very large goitres. Iodine supplements are not routinely recommended. Advice about adequate iodine intake through dietary measures is appropriate in the majority of cases.

N.B. Measurement of iodine levels to detect deficiency is not recommended.

Reference:

1. Merck Manual. Simple nontoxic goitre (euthyroid goitre). Available from: www.merck.com/mmpe/sec12/ch152/ch152i.html (Accessed Sept, 2010).

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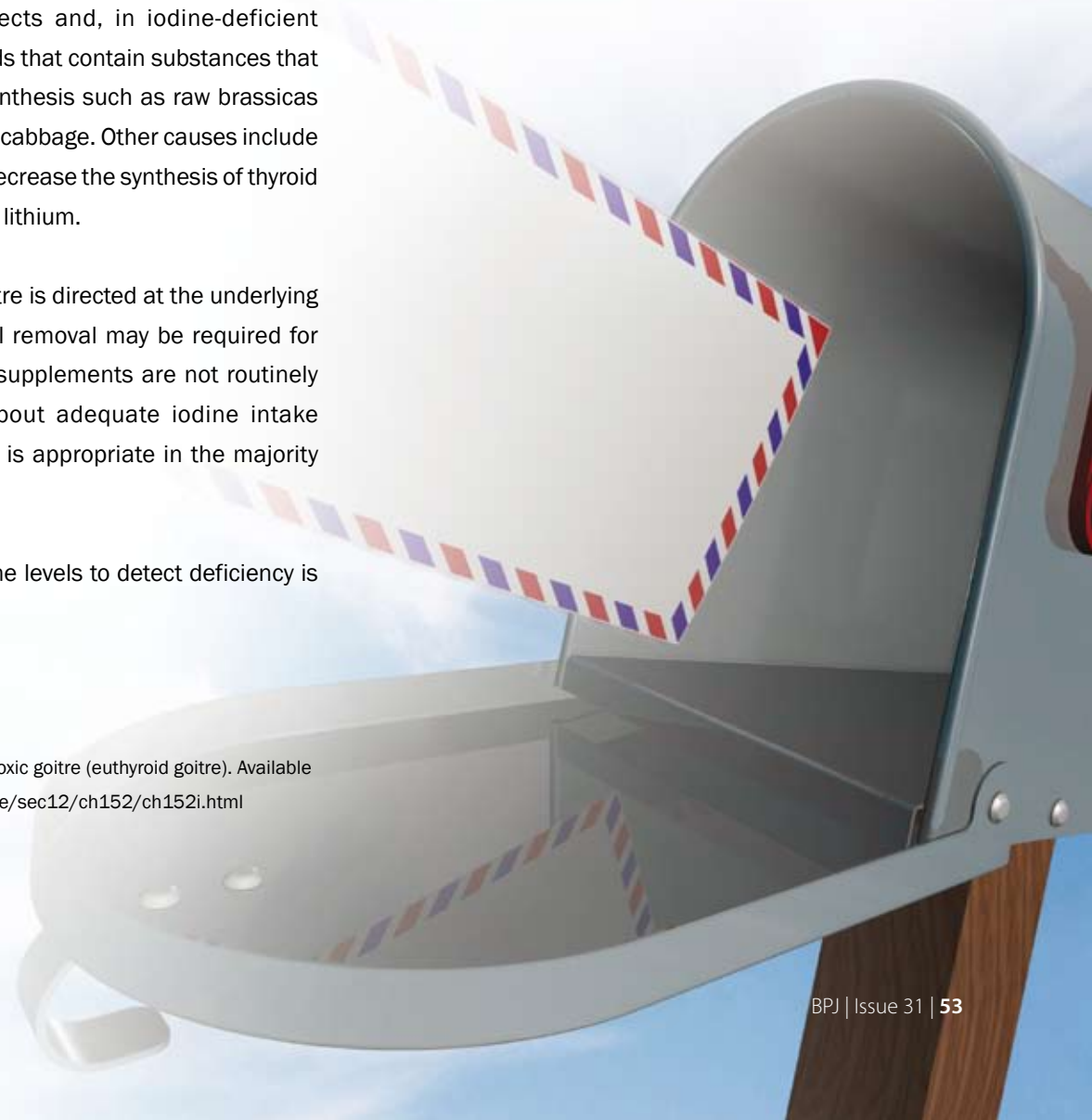

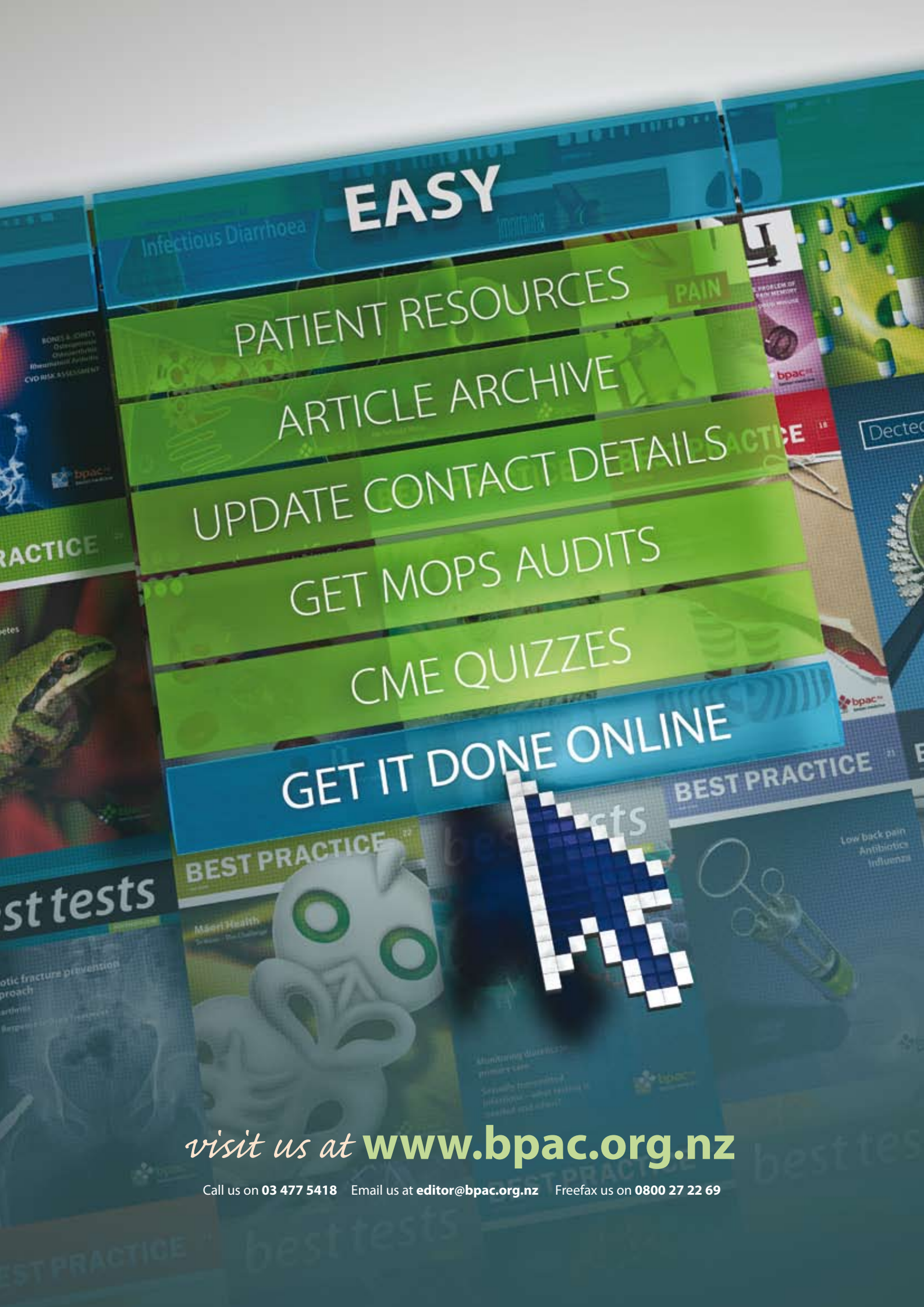
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