

# New Zealand **Primary Care Handbook 2012**

Cardiovascular risk assessment and diabetes screening

Cardiovascular risk factor management

Management of type 2 diabetes

Smoking cessation

Weight management

Stroke and transient ischaemic attack

Coronary heart disease

Heart failure

Prevention of infective endocarditis

Rheumatic fever



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## Statement of intent

Guideline handbooks are an important tool for evidenced-based practitioners. Handbooks both distil the contents of full guidelines and provide practical aids to the practitioner that may not be appropriate to include in the full guideline. While they represent a statement of best practice based on the latest available evidence and expert consensus (at the time of publishing), they are not intended to replace the health practitioner's judgment in each individual case.

Care decisions should consider the following:

- the individual's clinical state, age and comorbidities
- personal preferences and preferences of family/whānau
- current best practice based on the latest available research evidence.

## Development

The New Zealand Guidelines Group has managed the development of this publication for the Ministry of Health. The clinical content has been independently developed through processes managed by the New Zealand Guidelines Group, the New Zealand Stroke Foundation, the National Heart Foundation and the Clinical Trials Research Unit of Auckland University.

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# About the 2012 edition of the Handbook

## New content in the 2012 update

- **Management of type 2 diabetes**

New section that includes all content from *Guidance on the Management of Type 2 Diabetes* (2011) and content on the use of HbA1c in diagnosis

- **Weight management**

New section that includes summary content from *Clinical Guidelines for Weight Management in New Zealand Adults* (2009)

- **Stroke and transient ischaemic attack**

Updated section that includes new summary content from *New Zealand Clinical Guidelines for Stroke Management* (2010)

- **Heart failure**

Updated section that includes summary content from the updated *New Zealand Guideline for Management of Chronic Heart Failure* (2009)

- **Rheumatic fever**

Updated section developed for inclusion in this edition that includes content on sore throat management from a 2011 NZGG systematic review

*The New Zealand Primary Care Handbook 2012* is an updated edition of the original *New Zealand Cardiovascular Guidelines Handbook* published in 2005 (1st edition) and revised in 2009 (2nd edition). In this edition, new content is included on the following: management of type 2 diabetes, weight management, stroke and transient ischaemic attack, heart failure, and rheumatic fever. See the box above and individual sections for further detail. The section 'Atrial fibrillation and flutter', which appeared in the 2009 edition, is not repeated in this edition as the source guideline is out of date.

It is intended atrial fibrillation guidance will be updated in 2012. Other content in this Handbook is as per the 2009 edition, with limited editorial revisions to assist alignment with the new and updated sections. For details of the 2009 revision of the Handbook see Appendix A.

The Handbook provides summary guidance from the collection of guidelines and guidance documents listed below and is intended as a convenient ready-reference for primary care practitioners and allied health professionals. It is not intended to replace the health professional's judgment in each individual case.

The relevant source guidelines for content in this 2012 edition are listed below.

- *The Assessment and Management of Cardiovascular Risk* (2003)\*
- *Guidance on the Management of Type 2 Diabetes* (2011)\*
- *New Zealand Smoking Cessation Guidelines* (2007)<sup>†</sup>
- *Clinical Guidelines for Weight Management in New Zealand Adults* (2009)<sup>†</sup>
- *Cardiac Rehabilitation* (2002)\*
- *New Zealand Clinical Guidelines for Stroke Management* (2010)<sup>§</sup>
- *New Zealand Guideline for the Assessment and Management of People with Recent Transient Ischaemic Attack (TIA)* (2008)<sup>§</sup>
- *New Zealand Guidelines for Rheumatic Fever* (2006)<sup>†</sup>
- *Prevention of Infective Endocarditis associated with Dental and other Medical Interventions* (2008)<sup>†</sup>
- *New Zealand Guideline for Management of Chronic Heart Failure* (2009)<sup>†</sup>

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\* [www.nzgg.org.nz](http://www.nzgg.org.nz)

† [www.health.govt.nz](http://www.health.govt.nz)

‡ [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz)

§ [www.stroke.org.nz](http://www.stroke.org.nz)

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# 1 Cardiovascular risk assessment and diabetes screening



All treatment decisions should be based on an individual's **5-year absolute cardiovascular risk** (the likelihood of a cardiovascular event over 5 years)

This replaces decision-making based on individual risk factor levels.

By knowing the absolute risk, decisions can be made on prevention and treatment of cardiovascular disease (CVD). These include choices about appropriate lifestyle change, lipid-modifying and blood pressure lowering (BP lowering) medication, diabetes care, and medication after myocardial infarction (MI), stroke and other cardiovascular disease.

2012

Note that as well as higher CVD risk, people with diabetes face additional risks. Consult Chapter 4 *Management of type 2 diabetes* for assessment and management of these risks.

The overall goal is to reduce 5-year cardiovascular risk to **less than 15%**.

## New Zealand Cardiovascular Risk Charts

To calculate an individual's 5-year absolute cardiovascular risk use the New Zealand Cardiovascular Risk Charts (see Figure 1).



Risk factors determine the age at which risk assessment starts (see Table 1)



The charts are not used for certain high-risk groups (see Table 2)



Some people should be moved up one risk category (see Table 2)



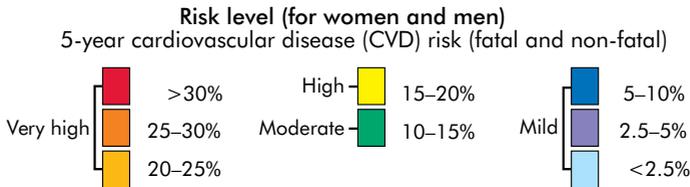
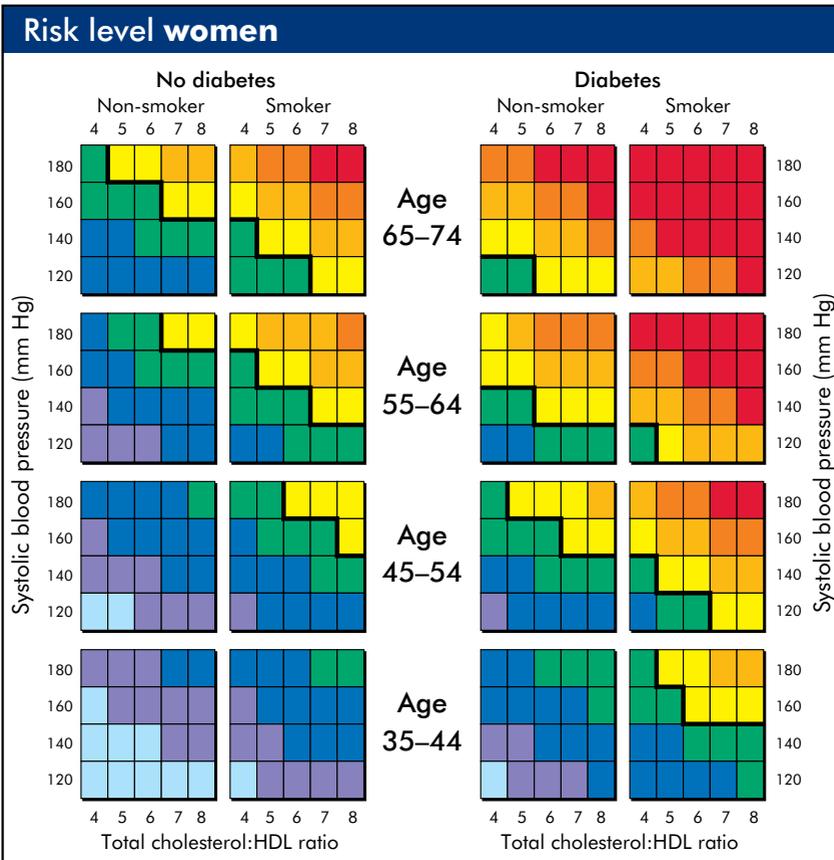
Include fasting blood tests as part of an assessment (see Table 3)



Follow-up intervals are determined by cardiovascular-risk calculation (see Table 4)

Figure 1

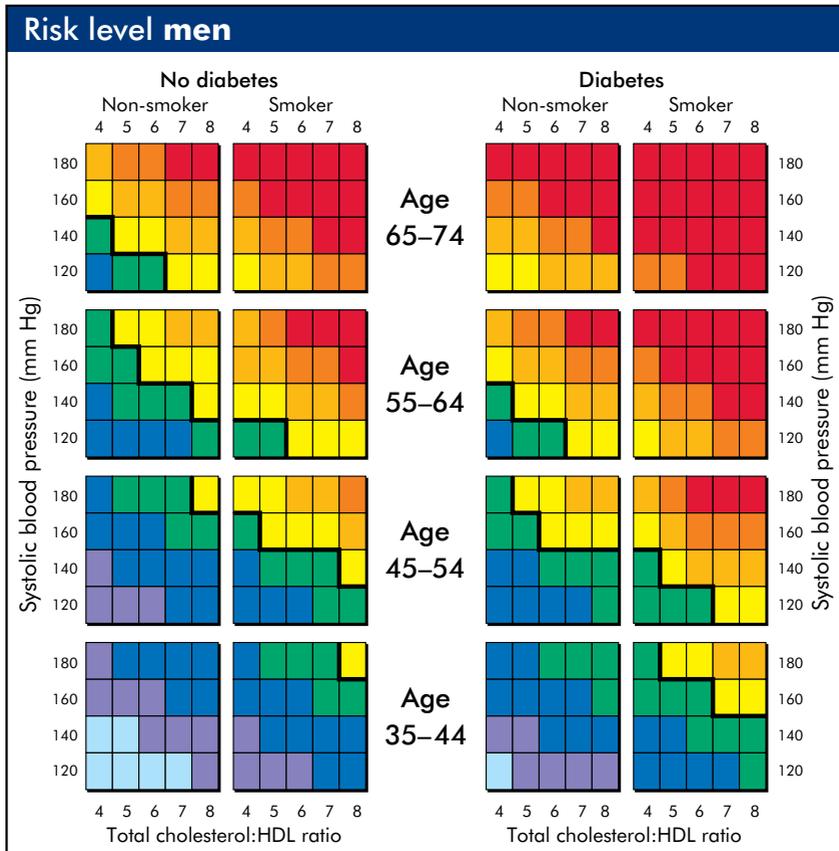
New Zealand Cardiovascular Risk Charts



**How to use the Charts**

- Identify the chart relating to the person's sex, diabetic status, smoking history and age
- Within the chart choose the cell nearest to the person's age, systolic blood pressure (SBP) and total cholesterol (TC) TC:HDL ratio. For example, the lower left cell contains all non-smokers without diabetes who are 35-44 years and have a TC:HDL ratio of less than 4.5 and a SBP of less than 130 mm Hg. People who fall exactly on a threshold between cells are placed in the cell indicating higher risk
- The risk charts now include values for SBP alone, as this is the most informative of conventionally measured blood pressure parameters for cardiovascular risk. Diastolic pressures may add some predictive power, especially at younger ages (eg, a diastolic pressure consistently > 100 mm Hg in a patient with SBP values between 140 and 170 mm Hg)

Certain groups may have CVD risk underestimated using these charts, see Table 2 (page 5) for recommended adjustments.



Risk level: 5-year CVD risk (fatal and non-fatal)	Benefits: NNT for 5 years to prevent one event (CVD events prevented per 100 people treated for 5 years)		
	1 intervention (25% risk reduction)	2 interventions (45% risk reduction)	3 interventions (55% risk reduction)
30%	13 (7.5 per 100)	7 (14 per 100)	6 (16 per 100)
20%	20 (5 per 100)	11 (9 per 100)	9 (11 per 100)
15%	27 (4 per 100)	15 (7 per 100)	12 (8 per 100)
10%	40 (2.5 per 100)	22 (4.5 per 100)	18 (5.5 per 100)
5%	80 (1.25 per 100)	44 (2.25 per 100)	36 (3 per 100)

#### NNT Number needed to treat

Based on the conservative estimate that each intervention: aspirin, BP treatment (lowering SBP by 10 mm Hg) or lipid modification (lowering LDL-C by 20%) reduces cardiovascular risk by about 25% over 5 years.

**Note:** Cardiovascular events are defined as myocardial infarction, new angina, ischaemic stroke, transient ischaemic attack (TIA), peripheral vascular disease, congestive heart failure and cardiovascular-related death.

**Adapted with permission from:** Rod Jackson, Head of the Section of Epidemiology and Biostatistics, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland.

**Table 1** The age to start cardiovascular disease risk assessment

Group	Men	Women
Asymptomatic people without known risk factors	Age 45 years	Age 55 years
Māori, Pacific peoples or Indo-Asian* peoples	Age 35 years	Age 45 years
People with other known cardiovascular risk factors or at high risk of developing diabetes <b>Family history risk factors</b> <ul style="list-style-type: none"> <li>• Diabetes in first-degree relative (parent, brother or sister)</li> <li>• Premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother &lt;55 years, mother or sister &lt;65 years)</li> </ul> <b>Personal history risk factors</b> <ul style="list-style-type: none"> <li>• People who smoke (or who have quit only in the last 12 months)</li> <li>• Gestational diabetes, polycystic ovary syndrome</li> <li>• Prior blood pressure (BP) <math>\geq 160/95</math> mm Hg, prior TC:HDL ratio <math>\geq 7</math></li> <li>• Prediabetes (see section 'Screening and diagnosis of type 2 diabetes' in Chapter 4)</li> <li>• BMI <math>\geq 30</math> or truncal obesity (waist circumference <math>\geq 100</math> cm in men or <math>\geq 90</math> cm in women)</li> <li>• eGFR<sup>†</sup> &lt;60 ml/min/1.73 m<sup>2</sup></li> </ul>	Age 35 years	Age 45 years
People with diabetes	Annually from the time of diagnosis	
* <b>Indo-Asian peoples</b> Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani, Tibetan † <b>eGFR</b> estimated glomerular filtration rate		

Risk assessment using a risk trajectory approach (see page 7) could be considered on a case-by-case basis for patients younger than the recommended ages, particularly where there is clinical concern regarding unfavourable risk factors.

Table 2	Estimating 5-year cardiovascular risk: when to use the New Zealand Cardiovascular Risk Charts*
Risk group	Estimating risk
Very high risk groups: 5-year risk assumed clinically >20%	<p><b>These people should not have their risk calculated using the New Zealand Cardiovascular Risk Charts as they will already have a very high risk due to their clinical condition</b></p> <ul style="list-style-type: none"> <li>• Previous CVD event: angina, MI, percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), transient ischaemic attack (TIA), ischaemic stroke, peripheral vascular disease</li> <li>• Some genetic lipid disorders: familial hypercholesterolaemia (FH), familial defective ApoB (FDB), familial combined dyslipidaemia (FCH)</li> <li>• Diabetes with overt nephropathy (albumin:creatinine ratio <math>\geq 30</math> mg/mmol OR urinary albumin <math>\geq 200</math> mg/L)</li> <li>• Diabetes with other renal disease causing renal impairment (eGFR <math>\leq 60</math> ml/min/1.73m<sup>2</sup>)</li> </ul>
Isolated elevated single risk factors: 5-year risk of >15%	<p>Calculate 5-year risk using the New Zealand Cardiovascular Risk Charts. When all risk factors are taken into account, the risk may be even higher than the assumed 5-year CVD risk of <math>\geq 15\%</math></p> <ul style="list-style-type: none"> <li>• TC <math>\geq 8</math> mmol/L</li> <li>• TC:HDL ratio <math>\geq 8</math></li> <li>• BP consistently <math>\geq 170/100</math></li> </ul>
People aged 35–74 years: calculate the 5-year CVD risk	<p>Calculate 5-year risk using the New Zealand Cardiovascular Risk Charts or an electronic decision-support tool (stand alone or incorporated into some practice software)</p> <p><b>These groups should be moved up one risk category (5%):<sup>†</sup></b></p> <ul style="list-style-type: none"> <li>• family history of premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother &lt;55 years, mother or sister &lt;65 years)</li> <li>• Māori, Pacific peoples or Indo-Asian<sup>‡</sup> peoples</li> <li>• diabetes with microalbuminuria OR for <math>\geq 10</math> years OR with HbA1c consistently <math>\geq 8\%</math> (64 mmol/mol)</li> </ul>

continued over...

**Table 2** continued...

Risk group	Estimating risk
<p>People aged &lt;35 years with known risk factors</p>	<p><b>All calculations outside the age ranges of the Framingham equation are approximations, but can be useful</b></p> <p><b>Aged under 35 years:</b> calculate the risk as if they were 35 years. The result can be used to guide clinical decision-making. Some risk factors in young people might require more intensive intervention or specialist referral</p> <ul style="list-style-type: none"> <li>• Low HDL &lt;0.7 mmol/L (because of the risk of a genetic lipid disorder – see Chapter 9 of the guideline <i>The Assessment and Management of Cardiovascular Risk</i>)</li> <li>• Known familial dyslipidaemias or suspected genetic lipid disorders</li> <li>• Type 1 diabetes, type 2 diabetes with microalbuminuria or type 2 diabetes of long duration (≥10 years)</li> </ul>
<p>People aged ≥75 years</p>	<p><b>Aged over 75 years:</b> calculate the risk as if they were 65–74 years</p> <p>An assessment of the balance between the risks and benefits of treatment is more difficult in older than in younger people. Older people gain a similar relative benefit from cholesterol lowering, but are more likely to benefit in absolute terms because of their much higher pretreatment cardiovascular risk. Smoking cessation is beneficial at any age</p> <p>A clinical judgment should take into account:</p> <ul style="list-style-type: none"> <li>• likely benefits and risks of treatment</li> <li>• life expectancy and comorbidities</li> <li>• personal values</li> </ul>
<p>* <b>Note that as well as higher CVD risk, people with diabetes face additional risks.</b> Consult Chapter 4 <i>Management of type 2 diabetes</i> for assessment and management of these risks.</p> <p>† Make the 5% adjustment <b>once</b> only for people with &gt;1 criterion.</p> <p>‡ <b>Indo-Asian peoples</b> Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani, Tibetan.</p>	

## Risk trajectory approach

Many younger patients have a low 5-year CVD risk despite having an unfavourable risk factor profile. When communicating risk to these patients it is recommended that practitioners follow the risk trajectory approach.

This involves not only showing the patient their current 5-year risk, but also their 5-year risk as they age, assuming no change in their risk factor profile (ie, their risk trajectory). In addition, the ideal risk trajectory for a patient of the same age, gender and diabetes status (ie, SBP = 120 mm Hg, TC:HDL = 4, non-smoker) should be shown to demonstrate the potential benefits of lifestyle modification.

Intermediate risk trajectories (eg, changing one risk factor) could also be shown. Risk trajectories can be derived directly from the New Zealand Cardiovascular Risk Charts.

### 2012 update: Your Heart Forecast

A purpose-built electronic tool has been developed jointly by the Heart Foundation, University of Auckland and Enigma. It can be downloaded from the Heart Foundation website:

[www.heartfoundation.org.nz/programmes-resources/health-professionals/your-heart-forecast](http://www.heartfoundation.org.nz/programmes-resources/health-professionals/your-heart-forecast)

Your Heart Forecast has been designed to help patients easily understand their individual risk of cardiovascular disease, overcoming many health literacy issues. It has been integrated into practice decision support software by some providers.

Table 3

### What to measure and record for cardiovascular risk assessment and diabetes screening

Everyone	History	<ul style="list-style-type: none"> <li>• Age</li> <li>• Gender</li> <li>• Ethnicity</li> <li>• Smoking status (if stopped smoking for &lt;12 months, assess as a smoker)</li> </ul>
	Family history	<ul style="list-style-type: none"> <li>• Premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother &lt;55 years, mother or sister &lt;65 years)</li> <li>• Type 2 diabetes</li> <li>• Genetic lipid disorder (see Appendix B)</li> </ul>
	Past medical history	<ul style="list-style-type: none"> <li>• Past history of CVD (MI, PCI, CABG, angina, ischaemic stroke, TIA, peripheral vascular disease [PVD])</li> <li>• Genetic lipid disorder (FH, FDB, FCH: see Appendix B)</li> <li>• Renal impairment</li> </ul>
	Measure	<ul style="list-style-type: none"> <li>• Average of two sitting BP measurements</li> <li>• Pulse</li> <li>• BMI, waist circumference</li> <li>• Fasting lipid profile*</li> <li>• HbA1c or fasting plasma glucose (see section 'Screening and diagnosis of type 2 diabetes' in Chapter 4)</li> </ul>
Diabetes	History and examination	<ul style="list-style-type: none"> <li>• Date of diagnosis</li> <li>• Type of diabetes (type 1, type 2, including type 2 on insulin, gestational diabetes)</li> <li>• HbA1c</li> <li>• Urine albumin: creatinine ratio (ACR)</li> <li>• eGFR<sup>†</sup> and history of renal disease</li> </ul>
Atrial fibrillation (AF), confirmed on electro-cardiogram (ECG)	History and examination	<ul style="list-style-type: none"> <li>• Echocardiogram (where possible)</li> <li>• Past history of stroke, TIA, heart failure, rheumatic or mitral valve disease</li> </ul>

\* When a fasting sample is not possible, measure non-fasting total cholesterol and HDL-cholesterol.

† Estimated glomerular filtration rate (eGFR).



Follow-up intervals are determined by cardiovascular risk calculation (see Table 4)

<b>Table 4</b> Frequency of cardiovascular risk assessment	
5-year risk <5%	Further risk assessment in 10 years
5-year risk 5–10%	Further risk assessment in 5 years
5-year risk 10–15%	Further risk assessment in 2 years
5-year risk $\geq$ 15%, diabetes, or on lipid or BP lowering medication	Annual risk assessment
People with diabetes,* those receiving medication or smoking cessation treatment or intensive lifestyle advice	May need individual risk factor measurements taken more frequently, eg, 3-monthly until controlled, then every 6 months
* For guidance on frequency of measurement of individual risk factors for people with diabetes see Chapter 4 <i>Management of type 2 diabetes</i> .	

# How to measure risk factors

## Lipids



Fasting lipid profile\* (TC, LDL-C, HDL-C, TC:HDL ratio and triglycerides) should be taken. A single TC:HDL ratio is used to calculate cardiovascular risk

\* When a fasting sample is not possible, a non-fasting TC:HDL ratio may be used for an initial calculation of cardiovascular risk.

Two lipid measurements should be taken prior to initiating drug treatment or intensive lifestyle treatment. If the total cholesterol level varies by more than 0.8 to 1.0 mmol/L in the two samples, a third sample should be taken and the average of the three samples should be used as the baseline measure.

A fasting sample is required for the measurement of triglycerides.

### Secondary causes of lipid abnormalities

The secondary causes of lipid abnormalities include diet and alcohol influences, hypothyroidism, diabetes, liver disease, nephrotic syndrome and steroid treatment.

A rise in triglycerides is seen in people with diabetes, people who are obese, or who have excessive alcohol consumption. Any identifiable cause should be treated prior to initiating lipid-lowering treatment. Markedly elevated triglycerides preclude the estimation of HDL and thus reliable risk assessment. A rise in cholesterol is normal in pregnancy and a cholesterol level should not be measured at this time.

### Genetic lipid disorders

Consider the possibility of a genetic lipid disorder if TC  $\geq 8$  mmol/L or if there is a family history of premature coronary heart disease. See Appendix B for definitions and management of genetic lipid disorders.

## Blood pressure



The average of two seated BP measurements is recommended for the initial risk assessment. This should be repeated on three separate occasions to obtain a baseline prior to the initiation of either intensive lifestyle modification or drug treatment

See Appendix C for recommended method of measuring BP. See Table 5 for cuff size to use when taking blood pressure.

<b>Table 5</b>		<b>Acceptable blood pressure cuff dimensions for arms of different sizes</b>	
<b>Cuff</b>	<b>Arm circumference range at midpoint (cm)</b>	<b>Bladder width (cm)</b>	<b>Bladder length (cm)</b>
Newborn	≤6	3	6
Infant	6–15	5	15
Child	16–21	8	21
Small adult	22–26	10	24
Adult	27–34	13	30
Large adult	35–44	16	38
Adult thigh	45–52	20	42

## Secondary causes of raised blood pressure

Secondary causes of raised BP include high alcohol intake, sleep apnoea, oestrogen and glucocorticoid administration, anti-inflammatory agents, cyclosporin and use of sympathomimetics.

Rarer causes that require further investigation in severe or resistant hypertension (especially in younger individuals) are renal disease, coarctation of the aorta, renal artery stenosis, pheochromocytoma, Cushing's syndrome and Conn's syndrome.

## Hyperglycaemia



Assessment for possible diabetes or prediabetes is an essential element of initial risk assessment. See Chapter 4 *Management of type 2 diabetes* for further details on screening and diagnosis of type 2 diabetes

## Smoking history



Current and past smoking habits should be recorded. For the purposes of CVD risk assessment, a non-smoker is defined as someone who has never smoked or has given up smoking and not smoked for 12 months

## Measures of weight and truncal obesity



Measure weight, height, waist circumference and calculate BMI ( $\text{kg}/\text{m}^2$ ) using Table 6

**normal weight** BMI of 18.5–24.9  $\text{kg}/\text{m}^2$

**overweight** BMI of 25.0–29.9  $\text{kg}/\text{m}^2$

**obese** BMI of 30  $\text{kg}/\text{m}^2$  or greater



BMI is not a direct measure of body fat. A range of indicators are necessary for screening and monitoring. BMI does not:

- distinguish between fat and lean mass
- distinguish between ethnic differences in the ratio of fat to lean muscle – individuals with similar BMIs but from different ethnic backgrounds may have different levels of fat tissue
- measure actual body fat or provide information about distribution of body fat



People of smaller stature and with a BMI  $<25 \text{ kg}/\text{m}^2$ , but with a large waist (indicating abdominal fatness) may still benefit from weight loss advice

2012

This content has been updated to align with *Clinical Guidelines for Weight Management in New Zealand Adults* (2009). See Chapter 5 *Weight management* for further content from this guideline.

**Table 6**

**Classification of weight in adults**

		Body mass index																															
		20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37														
Height in metres		Weight in kilograms																															
		1.50	1.55	1.60	1.65	1.70	1.75	1.80	1.85	1.90	1.95	45	47	50	52	54	56	59	61	63	65	68	71	74	76	79	82	84	87	89			
	1.50	45	47	50	52	54	56	59	61	63	65	68	71	74	76	79	82	84	87	89	61	63	65	68	71	74	76	79	82	84	87	89	
	1.55	48	51	53	55	58	60	63	65	67	70	72	75	77	79	82	84	87	89	64	67	69	72	75	78	81	84	87	90	92	95		
	1.60	51	54	56	59	61	64	67	69	72	74	77	79	82	85	87	90	92	95	67	69	72	75	78	81	84	87	90	93	95	98	101	
	1.65	54	57	60	63	65	68	71	74	76	79	82	84	87	90	93	95	98	101	70	74	77	81	84	87	90	93	95	98	101	104	107	
	1.70	58	61	64	67	69	72	75	78	81	84	87	90	93	95	98	101	104	107	74	77	80	83	86	89	92	95	98	101	104	107	110	113
	1.75	61	64	67	70	74	77	80	83	86	89	92	95	98	101	104	107	110	113	77	80	83	86	89	92	95	98	101	104	107	110	113	120
	1.80	65	68	71	75	78	81	84	88	91	94	97	100	104	107	110	113	117	120	80	84	88	91	94	97	100	104	107	110	113	117	120	127
	1.85	69	72	75	79	82	86	89	92	96	99	103	106	110	113	116	120	123	127	84	89	92	96	99	103	106	110	113	116	120	123	127	134
	1.90	72	76	79	83	87	90	94	98	101	105	108	112	116	119	123	126	130	134	88	91	95	99	103	107	110	114	118	122	126	130	134	141
	1.95	76	80	84	88	91	95	99	103	107	110	114	118	122	126	129	133	137	141	91	95	99	103	107	110	114	118	122	126	129	133	137	141
		Healthy					Overweight					Obese																					

See also Table 31 for body mass index and estimate of risk.

**How to measure waist circumference**

1. Ask the person to hold the end of the tape and to turn around.  
The tape should be horizontal and lie loosely against the skin.
2. Record waist circumference midway between the lower rib margin and the iliac crest to the nearest 1 cm.

## 2 Cardiovascular risk factor management

**2012** For guidance on management of blood pressure, microalbuminuria and glycaemic control in people with diabetes see Chapter 4 *Management of type 2 diabetes*.

### Goals and targets

	All treatment decisions should be based on an individual's 5-year absolute cardiovascular risk, not the level of individual risk factors
	Among people with a 5-year cardiovascular risk >15%, the aim of treatment is to lower cardiovascular risk to <15% (see Table 7)
	The order in which to start interventions should take into account individual risk factor profiles, potential side effects, other concurrent illness, compliance, personal preference and cost. It is appropriate to treat multiple risk factors simultaneously

Table 7 Goals for people without known cardiovascular disease	
CVD risk $\geq$ 15%	CVD risk <15%
Reduce 5-year cardiovascular risk to <15%	Reduce risk with lifestyle interventions
Recalculate risk at each review to determine current CVD risk	

The goal for everyone is to reduce 5-year cardiovascular risk.

An individual's risk factor levels should always be interpreted within the context of their calculated cardiovascular risk.

CVD risk goals can be more easily achieved by the simultaneous reduction in several risk factors (see Table 11, page 18).



Risk factors can be used as targets for people at high risk (see Table 8)

<b>Table 8</b>		<b>Optimal levels (targets) for people with known cardiovascular disease, or cardiovascular risk &gt;15% or diabetes</b>
		<b>Known cardiovascular disease or cardiovascular risk &gt;15% or diabetes*</b>
<b>Lipids</b>		
Total cholesterol		<4.0 mmol/L
LDL cholesterol		<2.0 mmol/L
HDL cholesterol		≥1.0 mmol/L
TC:HDL ratio		<4.0
Triglycerides		<1.7 mmol/L
<b>Blood pressure</b>		
BP		<130/80 mm Hg
<b>Glycaemic control in people with diabetes</b>		
HbA1c		50–55 mmol/mol or as individually agreed
<b>Smoking cessation</b>		
Smoking cessation should be strongly and repeatedly recommended at any level of CVD risk. All people who smoke should be advised to quit and offered treatment to help them stop completely. Reducing cigarette consumption is not a recommended treatment strategy		
* Content on diabetes has been updated to align with 2011 guidance on the management of people with type 2 diabetes (see Chapter 4 <i>Management of type 2 diabetes</i> ).		



Graded lifestyle advice is appropriate for everyone (see Table 9)

Table 9 Recommended lifestyle interventions (diet, physical activity, weight management) based on cardiovascular risk assessment	
5-year CVD risk	Intervention
<ul style="list-style-type: none"> <li>• Calculated &gt;20%</li> <li>• Cardiovascular disease</li> <li>• Genetic lipid disorders</li> <li>• Diabetes*</li> </ul>	Intensive lifestyle interventions (see page 24)
• Calculated 10–20%	Specific lifestyle interventions (see page 20)
• Calculated <10%	General lifestyle advice (see page 19)
* See Chapter 4 <i>Management of type 2 diabetes</i> for guidance on management for people with type 2 diabetes.	



Drug therapy is indicated for people with CVD risk  $\geq 15\%$  (see Table 10)

Table 10 Recommended drug interventions based on cardiovascular risk assessment	
5-year CVD risk	Intervention
Clinically >20%	Start low dose aspirin, unless contraindicated, and other drugs as appropriate to the condition (see Tables 26, 27 and see also Chapter 6 <i>Stroke and transient ischaemic attack</i> )
Calculated >20%	Start low dose aspirin, unless contraindicated, lipid modification and BP lowering simultaneously with intensive lifestyle advice
Calculated 15–20%	Start drug therapy after 3–6 months of lifestyle advice (if the calculated CVD risk is still > 15%)
CVD risk > 15%	Start drug therapy for persistently elevated isolated risk factors (TC $\geq 8$ mmol/L or TC:HDL ratio $\geq 8$ or BP $\geq 170/100$ )
All levels of CVD risk	<p>Smoking cessation drug therapy (nicotine replacement therapy, varenicline, bupropion, or nortriptyline) should be recommended to all smokers who wish to stop regardless of their level of CVD risk</p> <p><sup>2012</sup> Weight loss drug intervention (orlistat) can be considered for people with a BMI of 30 kg/m<sup>2</sup> or greater if other methods of weight loss have failed</p> <p>This statement has been updated to align with <i>Clinical Guidelines for Weight Management in New Zealand Adults</i> (2009). See Chapter 5 <i>Weight management</i> for further content from this guideline</p>



The higher an individual's absolute risk of a cardiovascular event the more aggressive the management should be (see Table 11)

Table 11

## The recommended interventions, goals and follow-up based on cardiovascular risk assessment

Cardiovascular risk	Lifestyle	Drug therapy	Treatment goals	Follow-up
CVD risk clinically determined* >20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, and physical activity  Lifestyle advice should be given simultaneously with drug treatment	Aspirin, if not contra-indicated, a beta-blocker, statin and an ACE inhibitor (after MI) or aspirin, statin and a new or increased dose of a BP lowering agent (after stroke)  Treatment for smoking cessation†	Efforts should be made to reach optimal risk factor levels	CVD risk assessments at least annually  Risk factor monitoring every 3–6 months
CVD risk calculated >20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, and physical activity  Lifestyle advice should be given simultaneously with drug treatment	Aspirin and drug treatment of all modifiable risk factors – BP lowering, lipid modification, glycaemic control (in people with diabetes)  Treatment for smoking cessation†	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (on recalculating risk)	CVD risk assessments at least annually  Risk factor monitoring every 3–6 months
15–20%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, and physical activity  This lifestyle advice should be given by the primary health care team for 3–6 months prior to initiating drug treatment	Aspirin and drug treatment of all modifiable risk factors – BP lowering lipid modification glycaemic control (in people with diabetes)  Treatment for smoking cessation†  Drug therapy indicated simultaneously with lifestyle advice for people with isolated high risk factor levels‡	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	CVD risk assessments at least annually  Risk factor monitoring every 3–6 months
10–15%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, and physical activity  This lifestyle advice should be given by the primary health care team	Treatment for smoking cessation†  Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further CVD risk assessment in 2 years
<10%	General lifestyle advice on a cardioprotective dietary pattern, and physical activity	Treatment for smoking cessation†  Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further CVD risk assessment in 5 or 10 years (see Table 4, page 9)
<p>* People who have had a previous cardiovascular event (angina, MI, PCI, coronary artery bypass graft, TIA, ischaemic stroke or peripheral vascular disease) OR people with certain genetic lipid disorders (FH, FDB, FCH) OR people with diabetes and overt diabetic nephropathy OR people with diabetes and renal disease.</p> <p>† Smoking cessation treatment should combine pharmacotherapy and behavioural support.</p> <p>‡ People with isolated high risk-factor levels, either total cholesterol <math>\geq 8</math> mmol/L or TC:HDL ratio <math>\geq 8</math> or blood pressure <math>\geq 170/100</math> mm Hg, should have these risk factors treated and their risk recalculated.</p>				

## General lifestyle interventions



Offer everyone advice promoting 'healthy heart' foods and a smoke-free, active lifestyle (see Table 12)

Table 12	General lifestyle advice for people at 5-year cardiovascular risk <10%
The Heart Foundation's 9 steps to eating for a healthy heart	<ol style="list-style-type: none"> <li>1. Enjoy three meals each day, select from dishes that include plant foods and fish and avoid dairy fat, meat fat or deep fried foods</li> <li>2. Choose fruits and/or vegetables at every meal and most snacks</li> <li>3. Select whole grains, whole grain breads, or high fibre breakfast cereals in place of white bread and low fibre varieties at most meals and snacks</li> <li>4. Include fish,* or legumes (eg, peas, beans or soy products) or a small serving of lean meat or skinned poultry, at one or two meals each day * Fish oil supplements, 1 g/day EPA and DHA combined, are recommended for people at increased CVD risk who do not eat oily fish</li> <li>5. Choose low fat milk, low fat milk products or replace with soy products</li> <li>6. Use small amounts of oil, margarine, nuts or seeds</li> <li>7. Drink plenty of fluids each day, particularly water, and limit sugar-sweetened drinks and alcohol</li> <li>8. Use only small amounts of total fats and oils, sugar and salt when cooking and preparing meals, snacks or drinks. Choose ready-prepared foods low in these ingredients</li> <li>9. Mostly avoid or rarely include butter, deep-fried and fatty foods, and only occasionally choose sweet bakery products</li> </ol>
Physical activity	<p>A minimum of 30 minutes of moderate intensity physical activity (eg, brisk walking) on most days of the week. People who are already doing this should do more activity of higher intensity, if they can. For people with time constraints this physical activity may be accumulated in bouts of 8 to 10 minutes</p> <p>Push Play – <a href="http://www.sparc.org.nz/en-nz/communities-and-clubs/Push-Play">www.sparc.org.nz/en-nz/communities-and-clubs/Push-Play</a></p>
Healthy weight	<ul style="list-style-type: none"> <li>• BMI &lt;25</li> <li>• Waist circumference &lt;100 cm in men or &lt;90 cm in women</li> </ul>
Quit smoking	<p>Quitting smoking has major and immediate health benefits for smokers of all ages and their families</p> <p>Smoking cessation should be strongly and repeatedly recommended at any level of CVD risk. All people who smoke should be advised to quit and offered treatment to help them stop completely. Details of treatments for smoking cessation are given in Tables 17–21 (pages 25–31)</p>

## Specific lifestyle interventions



Everyone with a 5-year cardiovascular risk between 10% and 20% should receive specific lifestyle advice from their primary health care team. This advice should be followed for 3 to 6 months prior to considering drug treatment, and continued for life



Smoking cessation should be strongly and repeatedly recommended at any level of CVD risk. All people who smoke should be advised to quit and offered treatment to help them stop completely



The *Clinical Guidelines for Weight Management in New Zealand Adults* (2009) recommends a stepped approach to assisting people to achieve and maintain a healthy weight. See Chapter 5 *Weight management* for details of this approach, summarised in Figure 7. For all people with a BMI  $\geq 25$ , the guideline recommends a combination of changes in food/nutrition, physical activity, and behavioural strategies to support these changes (see Tables 13 and 14)



An assessment of the duration, frequency, intensity and type of physical activity should be made. People who maintain a duration of activity level 3 (see Table 13) at 3 to 6 METs intensity (see Appendix D) are meeting the minimum requirement for health. More intense activity for longer should be encouraged



Specific lifestyle interventions are based on a behavioural approach to counselling. They aim to help people acquire the skills and motivation to alter eating patterns or physical activity habits. Techniques used include: self-monitoring, training to overcome common barriers, goal setting, providing guidance in shopping and food preparation, role playing, and arranging support or referral (see Tables 14 and 15)

	Level	Description
<b>Inactive</b>	1. Sedentary	People who have not taken part in sport or active leisure in the last 4 weeks
	2. Relatively inactive	People who have done some sport and active leisure in the last 4 weeks (but not necessarily in the last 7 days) and usually take part in <2.5 hours of sport and active leisure per week
<b>Active</b>	3. Relatively active	People who usually take part in 2.5–5 hours of sport and active leisure per week
	4. Highly active	People who usually take part in >5 hours of sport and active leisure per week

Aim for a minimum of 30 minutes of moderate-intensity physical activity on most days of the week.



Use motivational interviewing to establish goals appropriate for the person's readiness to change

Risk factor	Assessment and advice
Nutrition	<ul style="list-style-type: none"> <li>Assess general dietary habits against the National Heart Foundation's 9 steps to eating for a healthy heart (Table 12)</li> <li>Complete a lifestyle assessment diary</li> <li>Quantify intake and offer advice on the cardioprotective dietary pattern table (Appendix E)</li> </ul>
Physical activity	<ul style="list-style-type: none"> <li>Assess the current level of physical activity: duration and frequency (Table 13), intensity and type (Appendix D). Sports and leisure activities with energy expenditure of 3–6 METs meet the definition of 'moderate physical activity'</li> <li>Complete a lifestyle assessment diary</li> <li>The minimum goal is 30 minutes (level 3) of moderate intensity (3–6 METs) physical activity on most days of the week. For people with time constraints this physical activity may be accumulated in bouts of 8–10 minutes</li> <li>People who are already active at level 3 should be encouraged to do physical activity of higher intensity or for longer (aim for <math>\geq 6</math> METS or level 4)</li> <li>Consider issuing a green prescription/referring to a local sports trust</li> </ul>

continued over...

**Table 14** continued...

Risk factor	Assessment and advice
Weight	<ul style="list-style-type: none"> <li>• Set achievable goals, prevent weight gain, achieve and sustain moderate weight loss (5–10%) where appropriate and increase physical fitness</li> <li>• Discourage the use of weight loss programmes that promote the exclusion of food groups from the cardioprotective dietary pattern or that increase saturated fatty acid intake</li> <li>• Reduce foods rich in fats and oils, particularly saturated fat-rich foods and deep-fried products</li> <li>• Reduce white flour products and partially replace with whole grain products</li> <li>• Reduce foods and drinks rich in added sugars (bakery and confectionery items)</li> <li>• Ensure nutritional adequacy and cardiovascular protection</li> <li>• Consider the metabolic profile and other goals (including glycaemic, LDL-C, HDL-C, triglyceride levels and BP)</li> </ul> <p data-bbox="255 678 958 756"> <span style="border: 1px solid black; padding: 2px;">2012</span> The <i>Clinical Guidelines for Weight Management in New Zealand Adults</i> (2009) recommends a 4-step approach for achieving and maintaining healthy weight         </p> <ol style="list-style-type: none"> <li>1. Engage and raise awareness</li> <li>2. Identify the need and context for action</li> <li>3. Determine options for action – largely based on BMI</li> <li>4. Arrange ongoing contact and support</li> </ol> <p data-bbox="306 911 897 938">See Chapter 5 <i>Weight management</i> for details of this approach</p>
Smoking	<ul style="list-style-type: none"> <li>• Ask about and document smoking status prominently in medical record</li> <li>• Give brief advice to stop smoking. Strongly and repeatedly encourage person and family to stop smoking (this in itself is an effective intervention NNT = 40)</li> <li>• Offer cessation treatment to all smokers and provide treatment to those who want to stop (details of smoking cessation treatments are given in Tables 17–21, pages 25–31)</li> </ul>

<b>Table 15 Specific lifestyle changes to modify biomedical risk factors</b>	
<b>Risk factor</b>	<b>Assessment and advice</b>
Lipid modification	<ul style="list-style-type: none"> <li>• Adopt a cardioprotective dietary pattern (Appendix E)</li> <li>• Consider adding plant sterol or stanol-fortified spreads</li> <li>• Eat oily fish regularly</li> <li>• Choose foods which are low in saturated fatty acids, transunsaturated fat and dietary cholesterol</li> </ul>
BP lowering	<ul style="list-style-type: none"> <li>• Adopt a cardioprotective dietary pattern (Appendix E)</li> <li>• Reduce excessive alcohol intake (no more than 3 standard drinks/day for men or 2 standard drinks/day for women)</li> <li>• Reduce sodium intake to no more than 2 g/day (6 g sodium chloride)</li> </ul>

## Intensive lifestyle interventions



Intensive lifestyle advice is recommended for people with 5-year CVD risk >20% and some other high risk groups (see Table 16)



Intensive intervention usually requires referral; it assumes a quantitative assessment by a health professional specifically trained in the lifestyle area with arranged follow-up over a period of time. Intensive dietary advice should be given in individual or group sessions with a dietitian

**Table 16** Intensive lifestyle advice and referral guidelines for some high-risk groups

### MI, angina, after CABG or PCI

- Refer to a comprehensive cardiac rehabilitation programme that includes exercise training
- Fish oil supplements, 1 g/day EPA and DHA combined, may be offered post-MI
- Individuals with a history of CVD should consult their doctor before they undertake vigorous physical activity. Vigorous activity is generally not encouraged in people with impaired left ventricular function, severe coronary artery disease, recent MI, significant ventricular arrhythmias or stenotic valve disease
- Physical activity for people with coronary heart disease should begin at a low intensity and gradually increase over several weeks

### Ischaemic stroke or TIA

- Refer to organised stroke services

### Diabetes

- Refer to a dietitian and diabetes nurse specialist

### Genetic lipid disorders

- Refer to a specialist clinic for family tracing

### Tobacco use

- Provide advice and medication to aid cessation (see Tables 17–22, pages 25–32)
- Refer to smoking cessation treatment provider (eg, Quitline, Aukati Kai Paipa, local provider [www.smokefreecontacts.org.nz](http://www.smokefreecontacts.org.nz))

## Smoking cessation interventions

	Smoking cessation has major and immediate health benefits for all smokers
	Smoking cessation treatment should <b>follow the ABC approach</b> (see Table 17)
	The provision of pharmacotherapy to smokers with CVD risk is highly indicated (see Tables 18–20)
	The ABC approach should be repeated at follow-up visits to provide further assistance and to ensure that repeated quit attempts are made

Table 17	The ABC of smoking cessation
<p>The <i>New Zealand Smoking Cessation Guidelines</i> (2007)* recommends the use of ABC as a memory aid for smoking cessation interventions:</p> <p><b>A</b> is for asking all people if they smoke</p> <p><b>B</b> is for giving brief advice to stop smoking</p> <p><b>C</b> is for cessation support, which should be offered to all smokers who have an interest in stopping</p>	
<b>A</b>	<p><b>Ask about and document smoking status</b></p> <ul style="list-style-type: none"> <li>• All patients should have their smoking status documented in their clinical record as a vital sign</li> <li>• Smoking status should be updated regularly</li> </ul>
<b>B</b>	<p><b>Brief advice to quit</b></p> <ul style="list-style-type: none"> <li>• One of the most important interventions a health professional can deliver</li> <li>• Brief advice to quit roughly doubles the chances of long-term quitting</li> <li>• It can be delivered in under a minute</li> <li>• Brief advice should:               <ul style="list-style-type: none"> <li>– contain a clear message to stop smoking completely (do not advise to ‘just cut down’)</li> <li>– be linked to a current illness if appropriate (eg, ‘stopping smoking will reduce your risk of having a heart attack’)</li> <li>– be given to all smokers regardless of whether they want to quit or not (assessment of the stage of behavioural change is not necessary)</li> </ul> </li> <li>• It can be acknowledged that stopping smoking can be difficult and that some people try several times before they succeed. However, a positive message should be given (eg, ‘there are treatments I can give you that will make quitting easier and increase the chances of you stopping for good’)</li> </ul>

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Table 17 continued...

**C Cessation support**

Following advice to stop smoking, help to stop smoking should be offered including:

- referral for support
  - **Quitline:** a national telephone support line. Tel: 0800 778 778; Website: [www.quit.org.nz](http://www.quit.org.nz)
  - **Aukati Kai Paipa:** a smoking cessation service provided by Māori organisations for Māori who smoke. Tel: (09) 638 5800 Website: [www.tehotumanawa.org.nz](http://www.tehotumanawa.org.nz)
  - **Local smoking cessation provider:** for people who have been trained to deliver behavioural support and can provide nicotine replacement therapy via QuitCards. See [www.smokefreecontacts.org.nz](http://www.smokefreecontacts.org.nz)
- provision of a **smoking cessation medicine** (see Tables 18–20, pages 27–31). These work by alleviating symptoms of nicotine withdrawal (eg, craving, irritability, poor concentration)

\* The full guideline is available at [www.health.govt.nz/moh.nsf/indexmh/nz-smoking-cessation-guidelines](http://www.health.govt.nz/moh.nsf/indexmh/nz-smoking-cessation-guidelines)

## Advise to stop completely

**Cutting down** on the number of cigarettes smoked does not lead to significant health benefits. This is because smokers typically compensate by smoking the fewer cigarettes more intensively (eg, taking larger puffs, holding the smoke in for longer, smoking more of the cigarette). Switching to 'low tar' or 'light/mild' cigarettes has no health benefits for the same reason. The **best advice** you can give someone who smokes is to **stop completely**.

## Assess nicotine dependence

Measuring the degree of nicotine dependence can help identify those who would benefit from extra assistance to stop smoking. To assess the level of dependence ask: '**How soon after you wake up do you usually have your first cigarette?**'

If the person smokes within 30 minutes of waking, then they have a higher degree of nicotine dependence and are likely to benefit from more intensive smoking cessation treatments, particularly those utilising medications (see Tables 18–20).

Table 18 Nicotine replacement therapy	
<p><b>Use of NRT</b></p> <ul style="list-style-type: none"> <li>• Provides some of the nicotine a smoker would have otherwise got from cigarettes</li> <li>• Roughly doubles the chances of quitting long-term compared with placebo</li> <li>• Nicotine <b>patches, gum, and lozenges</b> are subsidised and available on prescription and via the QuitCard Scheme</li> <li>• Provide or refer for behavioural support and follow-up to increase likelihood of success</li> <li>• The choice of NRT product can be guided by individual preference</li> <li>• NRT should be used for at least 8 weeks. People who need NRT for longer than 8 weeks (eg, people who are highly dependent) can continue to use NRT</li> <li>• Combining two NRT products (eg, patch and gum) increases abstinence rates and is safe</li> </ul>	
<p><b>Patches</b> 21 mg/24 hr</p>	<ul style="list-style-type: none"> <li>• Patches come in full, medium and low strength. People should be commenced on the full-strength patch. The medium and low strengths are only used for weaning (weaning is not strictly necessary)</li> <li>• The advantages of patches are that they are very simple to use and people generally use them reliably as instructed</li> <li>• Patches are applied to a clean, dry, hairless area of skin and left on for a day. A new patch should be applied to a different area each day</li> <li>• Skin irritation is the most common side effect</li> </ul>
<p><b>Gum</b> 2 mg and 4 mg</p>	<ul style="list-style-type: none"> <li>• People who are highly dependent should use 4 mg gum</li> <li>• Each piece should be chewed slowly to release the nicotine, and a hot peppery taste will be experienced. The gum should then be 'parked' between the cheek and gums so that the nicotine can be absorbed. After a few minutes, the gum can be chewed again, then parked and the process repeated, for 20–30 minutes</li> <li>• People should aim to use between 10 and 15 pieces of gum a day (instruct people to use about one piece of gum per hour)</li> <li>• An initial unpleasant taste is common. People can be reassured that they will become tolerant of this taste after a short period (usually a couple of days)</li> <li>• Incorrect use of gum (and the other oral products listed in this table), for example, chewing gum too vigorously, usually results in more nicotine being swallowed. This is not hazardous but means that less nicotine is absorbed and may cause local irritation and hiccups</li> </ul>

continued over...

**Table 18** continued...

<p><b>Lozenge</b> 1 mg and 2 mg</p>	<ul style="list-style-type: none"> <li>• People who are highly dependent should use the higher dose lozenge</li> <li>• Use one lozenge per hour</li> </ul>
<p><b>Sublingual tablet</b> 2 mg</p>	<ul style="list-style-type: none"> <li>• These are placed under the tongue where they are left to dissolve</li> <li>• They should be used on an hourly basis</li> </ul>
<p><b>Inhaler</b></p>	<ul style="list-style-type: none"> <li>• The inhaler is a small plastic tube containing a replaceable nicotine cartridge</li> <li>• The user should puff on the inhaler for 20 minutes each hour. After four 20-minute puffing sessions, the cartridge should be changed</li> </ul>
<p><b>Exchange card programme (QuitCards)</b></p>	<ul style="list-style-type: none"> <li>• Provides a supply of nicotine patches (24 hr only), gum and lozenges to any smoker</li> <li>• Cost: \$3 per item on each card</li> <li>• Available via QuitCard providers, Quitline (0800 778 778), Quit Group website (<a href="http://www.quit.org.nz">www.quit.org.nz</a>)</li> </ul> <p>Registered Healthcare Professionals can become QuitCard providers by completing a brief online training module available at <a href="http://www.smokingcessation.abc.org.nz">www.smokingcessation.abc.org.nz</a></p>



Provide or refer for behavioral support and follow-up to increase likelihood of success

Table 19	Non-nicotine pharmacotherapies for smoking cessation
Varenicline (Champix)	<p><b>2012 Update</b></p> <ul style="list-style-type: none"> <li>• This medicine was designed specifically for smoking cessation. It acts on nicotinic acetylcholine receptors to reduce the severity of nicotine withdrawal symptoms</li> <li>• It at least doubles the chances of quitting long-term compared with placebo</li> <li>• It is not recommended for use in children under the age of 18 years and women who are pregnant or breastfeeding</li> <li>• There are no clinically-significant drug interactions to consider</li> <li>• People need to commence varenicline one week prior to their quit date. The dosage is as follows: days 1–3: 0.5 mg once daily; days 4–7: 0.5 mg twice daily; days 8 to end of treatment (12 weeks): 1 mg twice daily</li> <li>• Lower dose regimens also assist smoking cessation and reduce the incidence of adverse events (Cahill et al 2011)</li> <li>• The most common adverse effect is nausea</li> <li>• Possible links with serious adverse events including depressed mood, agitation, and suicidal thoughts have been reported but have not been substantiated to date (Cahill et al 2011). These safety concerns are being monitored</li> <li>• There may be a small, increased risk of cardiovascular adverse events (including angina, nonfatal MI, need for coronary revascularisation, and new diagnosis of PVD or admission for a procedure for the treatment of PVD) in patients with established cardiovascular disease (FDA, accessed 2011). The absolute risk of cardiovascular adverse events with varenicline in relation to its efficacy is small (Singh et al 2011). The benefits of varenicline should be weighed against the potential risks of its use. Patients should be advised to seek medical attention if they experience new or worsening symptoms of cardiovascular disease while taking varenicline</li> <li>• Varenicline is subsidised for those meeting the criteria (Special Authority currently applies)</li> </ul> <p>This information on varenicline has been updated due to safety concerns about the use of this drug. Updated content is drawn from the following: Cahill K, et al. <i>Cochrane Database of Systematic Reviews</i> 2011; <a href="http://www.fda.gov/Drugs/DrugSafety/ucm264436.htm">www.fda.gov/Drugs/DrugSafety/ucm264436.htm</a> (accessed September 2011); Singh S, et al. <i>Canadian Medical Association Journal</i> 2011.</p>

continued over...

Table 19 continued...

Bupropion (Zyban)	<ul style="list-style-type: none"><li>• Bupropion is an atypical antidepressant that also increases the chances of stopping smoking long-term (approximately doubles the chances compared with placebo)</li><li>• It is a prescription-only medicine and is not subsidised</li><li>• There are a number of contraindications and cautions (see <i>New Zealand Smoking Cessation Guidelines (2007)</i>, Appendix 5) that need to be taken into account when deciding to use this medicine. There are also some drug interactions that should be considered</li><li>• People need to start this medicine a week before their quit day. The dosage is as follows: days 1–3: one tablet (150 mg) daily; from day 4: one tablet twice a day, keeping at least 8 hours between each dose. A total course of 120 tablets should be prescribed</li><li>• Adverse effects include dry mouth, headache, and there is a small seizure risk</li></ul>
Nortriptyline	<ul style="list-style-type: none"><li>• This tricyclic antidepressant has also been found to approximately double the chances of long-term abstinence compared with placebo</li><li>• The advantage of this medicine is that it is inexpensive, but it can be difficult to use for smoking cessation since it has to be started a few weeks before quitting</li><li>• The treatment regimen is as follows: initially 25 mg/day, beginning 10–28 days before quit date; increase gradually to 75–100 mg/day over 10 days–5 weeks; continue for a total of 12 weeks. The dose should be tapered at the end of treatment to avoid withdrawal symptoms that may occur if it is stopped abruptly</li><li>• Adverse effects, such as dry mouth and sedation, are common</li></ul>



Current evidence shows NRT to be safe in people with cardiovascular disease (see Table 20)

<b>Table 20 Cardiovascular disease and smoking cessation therapies</b>	
NRT	<ul style="list-style-type: none"> <li>• NRT can be provided to people with cardiovascular disease; dosage adjustment is required</li> <li>• Where people have suffered a serious cardiovascular event (eg, a myocardial infarction or stroke) in the past 2 weeks or have poorly controlled disease, treatment should be discussed with a physician. Oral NRT products are recommended (rather than longer-acting patches) for these patients</li> </ul>
Varenicline	<ul style="list-style-type: none"> <li>• Suitable treatment, if appropriate</li> <li>• There are no data regarding use of varenicline in people with acute CVD</li> </ul>
Bupropion	<ul style="list-style-type: none"> <li>• Suitable treatment, if appropriate</li> </ul>
Nortriptyline	<ul style="list-style-type: none"> <li>• Contraindicated in acute recovery phase after MI</li> </ul>



Quitting at any point in pregnancy can be beneficial for the foetus and mother (see Table 21)

<b>Table 21 Smoking cessation in pregnancy and breastfeeding</b>	
NRT in pregnancy	<ul style="list-style-type: none"> <li>• Manufacturers do not recommend NRT; however, NRT is safer than smoking</li> <li>• Pregnant women can use NRT after they have been informed of and have weighed up the risks and benefits. Intermittent NRT (for example, gum, inhaler, microtab and lozenge) should be used in preference to patches</li> <li>• NRT may be used in women who are breastfeeding. More detailed information can be found in the <i>New Zealand Smoking Cessation Guidelines (2007)</i></li> </ul>
Other medications	<ul style="list-style-type: none"> <li>• Varenicline, bupropion and nortriptyline are NOT recommended for use in pregnant women for smoking cessation</li> </ul>



Minor weight gain is common when people stop smoking (see Table 22)

**Table 22** Smoking cessation and weight gain

Weight gain	<ul style="list-style-type: none"><li>• On average, people can expect to gain 4–5 kg in the first year of abstinence</li><li>• Although this is a significant gain, the benefits of stopping smoking outweigh the health risks of the additional weight gain</li><li>• ‘Dieting’ at the same time as stopping smoking can increase urges to smoke and so may increase the risk of relapse. People should concentrate on achieving and maintaining abstinence from smoking first and then tackle the issue of weight gain</li><li>• For smokers concerned with weight gain, consider bupropion or NRT, in particular gum, which has been shown to delay weight gain after quitting</li></ul>
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## Complementary and alternative therapies



Clinicians should enquire about the use of alternative and complementary medicines when assessing cardiovascular risk or prescribing medication (see Table 23)

Table 23 Adverse effects of some complementary or alternative medicines	
Complementary or alternative medicine	Effect
Feverfew, garlic, <i>Ginkgo biloba</i> , ginger, ginseng	May alter bleeding time and should not be used concomitantly with warfarin
St John's wort	Reduces serum digoxin levels and can enhance warfarin metabolism
Some herbs (eg, karela and ginseng)	May affect blood glucose levels and should not be used in people with diabetes
Beta-carotene, vitamin C and vitamin E	RCT* evidence shows that vitamin supplementation with these antioxidant vitamins does not reduce cardiovascular risk  A meta-analysis has shown that beta-carotene led to a small but significant increase in all-cause mortality and a slight increase in cardiovascular death
<p><b>Note:</b> There is <b>insufficient evidence</b> to recommend the following complementary and alternative therapies for the treatment or prevention of CVD: herbal medicines/botanicals (garlic, <i>Ginkgo biloba</i>, rosemary, horse-chestnut seeds, xin bao), acupuncture, chelation therapy, traditional Chinese medicine, aromatherapy, homeopathy, hypnosis, meditation, yoga, tai chi, intercessory prayer, Strauss heart drops.</p> <p>* Randomised controlled trials</p>	

## Lipid modification



Lipid levels (TC from about 4–8 mmol/L) in people without CVD should be interpreted in the context of their cardiovascular risk (see Table 24). There is no normal or ideal lipid level. Risk factors can be viewed as treatment targets for people at high risk (see Table 25)

<b>Table 24 Clinical scenario and intervention recommendations</b>	
TC 4–8 mmol/L	<ul style="list-style-type: none"> <li>All decisions to treat should be based on the individual's cardiovascular risk</li> </ul>
Isolated risk factors: TC $\geq$ 8 mmol/L or TC:HDL ratio $\geq$ 8	<ul style="list-style-type: none"> <li>Assume 5-year CVD risk to be at least 15%</li> <li>Calculate risk using the charts as CVD risk may be higher than this</li> <li>Commence specific individualised lifestyle advice, smoking cessation treatment, aspirin, lipid-modifying therapy, +/- BP lowering therapy</li> </ul>
CVD risk >20% clinically*	<ul style="list-style-type: none"> <li>Commence statin (unless contraindicated) simultaneously with intensive lifestyle advice, smoking cessation treatment, aspirin, and other appropriate medication</li> </ul>
Calculated CVD risk >20%	<ul style="list-style-type: none"> <li>Aim is to reduce 5-year CVD risk to &lt;15%, which can be more easily achieved by reduction of all modifiable risk factors</li> <li>Commence intensive lifestyle advice, smoking cessation treatment, simultaneously with aspirin and drug treatment of all modifiable risk factors</li> </ul>
Calculated CVD risk 15–20%	<ul style="list-style-type: none"> <li>Aim is to reduce 5-year CVD risk to &lt;15%, which can be more easily achieved by simultaneous reduction of all modifiable risk factors</li> <li>Smoking cessation treatment</li> <li>Commence specific individualised lifestyle advice for 3–6 months before considering aspirin, lipid-modifying or BP lowering therapy</li> </ul>
Calculated CVD risk 10–15%	<ul style="list-style-type: none"> <li>Smoking cessation treatment</li> <li>Specific individualised lifestyle advice on a cardioprotective dietary pattern and physical activity</li> </ul>
Calculated CVD risk <10%	<ul style="list-style-type: none"> <li>Smoking cessation treatment</li> <li>General lifestyle advice, including dietary advice on a cardioprotective diet, physical activity</li> </ul>
<p>* Previous CVD event (angina, MI, PCI, CABG, TIA, ischaemic stroke or peripheral vascular disease) OR people with certain genetic lipid disorders (FH, FDB, FCH) OR diabetes and overt diabetic nephropathy OR diabetes and renal disease.</p>	

<b>Table 25 Optimal lipid levels (targets) for people with known cardiovascular disease, or cardiovascular risk &gt;15% or diabetes</b>	
<b>Known cardiovascular disease or cardiovascular risk &gt;15% or diabetes</b>	
Total cholesterol	<4.0 mmol/L
LDL cholesterol	<2.0 mmol/L
HDL cholesterol	≥1.0 mmol/L
Triglycerides	<1.7 mmol/L

	Before starting medication, it is important to consider and exclude a treatable primary cause for a dyslipidaemia. Such causes include diet and alcohol influences, hypothyroidism, diabetes, liver disease, nephrotic syndrome and steroid treatment
	For people with known cardiovascular disease and those at high cardiovascular risk, statin treatment is recommended
	Recommended starting doses for statin treatment: <ul style="list-style-type: none"> <li>• for people with known CVD or CVD risk &gt;20%, simvastatin 40 mg</li> <li>• for people with 5-year CVD risk 15–20% if initiating drug therapy, simvastatin 20 mg and titrate if needed</li> </ul>
	LDL-C is the primary indicator of optimum lipid management for CVD risk. HDL-C and triglycerides are secondary indicators
	Monitoring of lipids every 3 months until treatment is stable and then every 6 months is recommended
	If LDL-C targets are not met, options include increasing simvastatin to 80 mg, substituting atorvastatin or combining simvastatin with nicotinic acid or ezetimibe
	In all cases, lifestyle measures (diet and physical activity) should continue to be encouraged after initiation of drug treatment

## Statin safety monitoring

- Monitoring of liver function tests with statin use is not considered necessary as the risk of liver toxicity appears negligible
- Monitoring of creatine kinase (CK) is not required in those who are asymptomatic. CK should be checked for unexplained muscle pain, tenderness or weakness. The risk of myopathy is usually dose-related and is increased in the elderly, and with combination treatments.
  - For muscle pain without CK rise, dose reduction or discontinuation may be required
  - With CK rise 3–10x normal with symptoms, dose reduction or discontinuation with regular weekly monitoring of symptoms and CK is appropriate
  - With CK rise >10x normal with symptoms, discontinue statin immediately

## Specific lipid profiles and treatments

### Predominant hypercholesterolaemia

Statins are first line treatment and can be used in combination with ezetimibe, nicotinic acid or resins to lower TC and LDL-C. Nicotinic acid or possibly fibrates may be considered if low HDL-C (<1.0 mmol/L) persists on statin treatment. People with a very low HDL-C (<0.7 mmol/L) may need specialist review.

### Predominant hypertriglyceridaemia and low HDL-C

Before using medications, it is important to identify lifestyle relating factors (eg, diet, alcohol, obesity) or any primary cause (eg, diabetes) which may be exacerbating lipid abnormalities. Correcting these factors may make drug treatment unnecessary. Nicotinic acid, acipimox or fibrates are the most appropriate options to consider. Statins are not usually effective if triglycerides are markedly elevated (>5 mmol/L).

### Combined dyslipidaemia

Lifestyle factors may be significant. Consider treatment with a statin and nicotinic acid or a fibrate in people with moderate to marked elevation of LDL-C and triglycerides. Because of the increased risk of myopathy with combinations (particularly with gemfibrozil), special care should be taken to inform and monitor people on combination treatment.

## Blood pressure lowering



Within the BP range 115/70 to 170/100 mm Hg, all decisions to treat should be based on the individual's cardiovascular risk

Everyone with a BP  $\geq$ 170/100 mm Hg should have drug treatment and specific lifestyle advice to lower risk factor levels. If they smoke they should be strongly advised to stop and offered smoking cessation treatment.

Most of the treatment benefit is achieved by reaching the following BP levels:

- <140/85 mm Hg in people without clinical CVD
- <130/80 mm Hg in people with diabetes or CVD
- <130/80 mm Hg in people with chronic kidney disease and significant albuminuria (urine protein/creatinine >100 mg/mmol).

Limit alcohol and salt consumption and recommend a cardioprotective dietary pattern (see Appendix E) as an integral part of BP management.

### Choice of blood pressure-lowering medication

- The conventional antihypertensive medications used (thiazide diuretics, beta-blockers, ACE inhibitors or A2 receptor blockers and calcium channel blockers) have similar efficacy in lowering BP, with the exception of beta-blockers, which appear to be less effective. This is also reflected in outcome studies which indicate that beta-blockers be reserved for those with specific indications or when the other three main classes have proved inadequate in achieving BP control
- A low dose thiazide diuretic remains an acceptable option for first-line therapy in many people without contraindications or indications for one of the other treatment options
- Beta-blockers and thiazide diuretics may be associated with a higher future incidence of new onset diabetes but the clinical impact of this is uncertain
- More than one drug is frequently required to lower BP to optimum levels. When combining antihypertensive agents, the addition of a beta-blocker to an ACE inhibitor or A2 receptor blocker may be less effective than other combinations. The same applies to the addition of a calcium channel blocker to a diuretic. However, in resistant cases, these combinations

may still be useful. As a rule, the combination of verapamil and a beta-blocker should be avoided and other combinations may have additional risks in particular patient groups

- Low dose combination therapies can maximise effectiveness and help minimise side effects

## After myocardial infarction

- Beta-blockers reduce total mortality, cardiovascular mortality and morbidity
- Treat **all** people post-MI with a beta-blocker (eg, metoprolol, propranolol or timolol). Consider adding an ACE inhibitor long-term (regardless of BP level) **especially if any significant left ventricular impairment**
- Give intensive lifestyle advice and other appropriate medication, such as aspirin and a statin

## After stroke or transient ischaemic attack

This content on stroke and transient ischaemic attack has been updated for this 2011 edition. See Chapter 6 *Stroke and transient ischaemic attack* for details of the source guidelines.

### Acute blood pressure lowering therapy

- If blood pressure is extremely high (eg, for ischaemic stroke BP >220/120; for intracerebral haemorrhage BP >180/100 mm Hg) antihypertensive therapy can be started or increased, but blood pressure should be cautiously reduced (eg, by no more than 10–20%) and the patient monitored for signs of neurological deterioration
- In acute primary intracerebral haemorrhage, medication (that could include intravenous treatment) can be used to maintain a blood pressure below 180 mm Hg systolic (mean arterial pressure of 130 mm Hg) if severe hypertension is observed over several repeated measures within the first 24 to 48 hours of stroke onset
- Pre-existing antihypertensive therapy can be continued (orally or via nasogastric tube) provided there is no symptomatic hypotension or other reason to withhold treatment

## Secondary prevention post-acute ischaemic stroke or TIA

- Long-term antiplatelet therapy should be prescribed to all people with ischaemic stroke or TIA who are not prescribed anticoagulation therapy
- Anticoagulation therapy for long-term secondary prevention should be used in all people with ischaemic stroke or TIA who have atrial fibrillation or cardioembolic stroke and no contraindication
- All patients after stroke or TIA, whether normotensive or hypertensive, should receive blood pressure lowering therapy for secondary prevention, unless contraindicated by symptomatic hypotension
- The most direct evidence of benefit is for the use of an ACE inhibitor (alone or in combination with a diuretic); however, different agents have generally been found to be effective in lowering BP, with the exception of beta-blockers
- New blood pressure lowering therapy should commence prior to discharge for those with stroke or TIA, or soon after TIA if the patient is not admitted
- Cautious introduction of BP lowering medication may be required in older people with frailty, due to risk of complications, such as symptomatic hypotension

## Lifestyle modifications

Every person with stroke should be assessed and informed of their risk factors for a further stroke and possible strategies to modify identified risk factors. Interventions for risk factors include:

- smoking cessation
- improving diet
- increasing regular exercise.

## People aged 75 years and over

- Cardiovascular risk increases with age. These people have a greater potential to benefit from treatment and this has been confirmed in randomised trials
- People aged 75 years and over with isolated raised systolic hypertension (SBP 160 mm Hg, DBP <90 mm Hg) have an increased risk of ischaemic stroke and BP should be managed aggressively

- Older people generally tolerate BP lowering medication as well as younger age groups
- Low dose thiazide diuretics and calcium channel blockers may be more effective initial choices in this group
- Beta-blockers and ACE inhibitors can be used in this group of people as additional agents
- Postural hypotension is common in the elderly, especially those on drug treatment, and alpha blockers should only be used with great caution in this patient group
- All people who smoke should be strongly advised to stop and offered smoking cessation treatment. There are benefits from stopping smoking at any age

## Diabetes

- BP target for **all** people with diabetes is <130/80 mm Hg
- All people who smoke should be offered ABC smoking cessation advice

This content has been updated to align with *Guidance on the Management of Type 2 Diabetes* (2011). See Chapter 4 *Management of type 2 diabetes* for guidance on management of raised blood pressure and microalbuminuria for people with type 2 diabetes.

## Chronic kidney disease

- Aggressive management of blood pressure (target BP levels <130/180 mm Hg) is advised. Combination of an ACE inhibitor and A2 receptor blocker is not currently supported by outcome evidence

## Long-term antiplatelet therapy

-  Aspirin reduces the risk of a cardiovascular event by about 25% over 5 years
-  The decision to use aspirin should be based on a balance of the risks and benefits for each person taking into account their absolute risk of an event (see Table 26)

Table 26 Indications for long-term aspirin use	
5-year CVD risk	Recommendation
Risk >20% clinically*	After angina or MI commence low dose aspirin (75–150 mg), a beta-blocker, a statin and an ACE inhibitor  After ischaemic stroke or TIA commence low dose aspirin and a statin. Start or increase doses of BP lowering drugs (two usually required)
Risk calculated >15%	Commence low dose aspirin (75–150 mg/day) unless contraindicated
Risk assumed to be >15%: isolated high-risk factors <ul style="list-style-type: none"> <li>• TC ≥8 mmol/L</li> <li>• TC:HDL ratio ≥8</li> <li>• BP ≥170/100 mm Hg</li> </ul>	Low dose aspirin is as effective as higher daily doses and may be associated with less bleeding
No clinical CVD and calculated 5-year CVD risk <15%	The risk of a significant bleed or major haemorrhage <b>outweighs</b> the benefits of aspirin for the prevention of CVD. Other indications may exist
* See Table 2 for a definition of people at >20% CVD risk clinically.	

## Aspirin contraindications

Aspirin allergies/intolerance, active peptic ulceration, uncontrolled BP and other major bleeding risks.

## Adverse effects

Haemorrhage is the most serious side effect, particularly intracranial haemorrhage.

- Intracranial haemorrhage: absolute excess risk of about 2/1000 people treated per year
- Extracranial haemorrhage: absolute excess risk of about 1 to 2/1000 people treated per year. Most extracranial haemorrhages are non-fatal
- Upper gastrointestinal bleeding/perforation: regular aspirin at doses <300 mg/day is associated with about a two-fold increased risk

## Aspirin alternatives

Clopidogrel (75 mg/day) is at least as effective and as safe as aspirin and is an alternative for people with an aspirin contraindication or intolerance.

Combination treatment with modified-release dipyridamole and aspirin can be used for prevention of non-fatal stroke for patients at high risk of cerebral ischaemic events, including those who have symptomatic cerebral ischaemia while treated with aspirin alone.

Monotherapy with modified-release dipyridamole is recommended for prevention of non-fatal stroke if aspirin is contraindicated and clopidogrel is unavailable.

### 3 Angina and myocardial infarction: long-term therapy

	Comprehensive cardiac rehabilitation should be considered in all people after MI, CABG or PCI
	Most therapies will have been started in hospital. Some people, on review in primary care, will require initiation or dose adjustment
	All people post-MI or angina should be on aspirin, a statin, and a beta-blocker and considered for an ACE inhibitor, unless contraindicated (see Table 27)
	All people who smoke should be strongly and repeatedly advised to stop and offered smoking cessation treatment

<b>Table 27</b>	
<b>Recommended medications after myocardial infarction or angina</b>	
<b>Drug</b>	<b>Recommendation</b>
<b>Aspirin</b>	Aspirin 75–150 mg should be given routinely and continued for life. These doses are at least as effective as higher doses
<b>Clopidogrel</b>	Clopidogrel (75 mg/day) is an effective alternative to aspirin for people with contraindications to aspirin or those who are intolerant of aspirin
<b>Warfarin</b>	<p>Warfarin should be prescribed for high-risk MI survivors including those with:</p> <ul style="list-style-type: none"> <li>• atrial fibrillation or paroxysmal atrial fibrillation</li> <li>• a large left ventricular aneurysm</li> <li>• thrombus demonstrated in the left ventricle at the infarction site by echocardiography</li> <li>• systemic embolism</li> </ul> <p>Consider warfarin in people who cannot be given antiplatelet agents after MI</p> <p>The target INR should be 2.5 (range 2.0–3.0)</p>

continued over...

**Table 27** continued...

<p><b>Beta-blockers</b></p>	<p>Beta-blockers (eg, metoprolol, timolol, propranolol) should be considered for everyone following MI unless contraindicated</p> <p>Beta-blockers are also recommended in those with left ventricular dysfunction and heart failure (see Figure 9)</p> <ul style="list-style-type: none"> <li>• The initial dose of beta-blockers may be low and the dose may then be slowly titrated</li> <li>• Beta-blockers given at night may reduce the risks of postural hypotension and alleviate symptoms of tiredness and lethargy</li> <li>• Before discontinuing beta-blockers because of side effects, a lower dose or alternative beta-blocker should be tried</li> <li>• If full doses of a beta-blocker and ACE inhibitor are not tolerated, moderate doses of both are preferable to a high-dose of a single agent</li> </ul>
<p><b>ACE inhibitors</b></p>	<p>An ACE inhibitor should be considered for everyone after MI</p> <p>Treatment should be started early and continued, especially in those with anterior infarction, LV dysfunction or heart failure</p>
<p><b>Statins</b></p>	<p>A statin equivalent to simvastatin 20–40 mg daily should be started after MI</p>
<p><b>Calcium channel blockers</b></p>	<p>Rate-limiting non-dihydropyridine calcium channel blockers (verapamil and diltiazem) may be considered for people with normal ventricular function where beta-blockers are contraindicated and treatment is required for concurrent angina or hypertension</p>
<p><b>Nitrates</b></p>	<p>Nitrates can be used after MI for controlling symptoms of angina, but are not indicated for reducing the risk of further events</p>
<p><b>Smoking cessation treatments</b></p>	<p>Nicotine replacement therapy can be used after MI. A risk-benefit assessment is normally indicated. Smoking after MI represents a much greater risk than nicotine from NRT. If NRT is used, it is recommended that oral short-acting products (eg, gum or lozenges) be used in preference to patches in the immediate post-acute period (see Table 18, page 27)</p>

Antiarrhythmic therapy, apart from beta-blockers, is not recommended for routine use after MI.

Combined hormone replacement therapy (HRT) should not be used for the prevention of coronary heart disease or after a cardiovascular event.

# 4 Management of type 2 diabetes

This chapter contains the content of *Guidance on the Management of Type 2 Diabetes* (2011). See the full guidance document for information on the development of this guidance at [www.nzgg.org.nz](http://www.nzgg.org.nz) – search on title. Additional content on screening and diagnosis of type 2 diabetes is also included.\*

This guidance addresses four priority areas in the management of type 2 diabetes:

- diagnosis of type 2 diabetes\*
- early identification of patients at high risk of diabetes-related complications
- better management of raised blood pressure and microalbuminuria
- improved glycaemic control (including insulin initiation).

\* Taken from the New Zealand Society for the Study of Diabetes (NZSSD) *Position Statement on the diagnosis of, and screening for, type 2 diabetes* (September, 2011). The position statement is available at [www.nzssd.org.nz](http://www.nzssd.org.nz)

## Screening and diagnosis of type 2 diabetes

### About diagnosis and use of HbA1c

In the absence of symptoms and/or markedly raised blood glucose levels, the diagnosis of diabetes has been based on measures of glycaemia that are associated with an increased risk of its specific microvascular complications (in particular, retinopathy). The precise criteria have always been determined by consensus among experts, based principally on several large observational cohort studies. The criteria have been somewhat modified over time as more high quality data have become available. More recently, several international diabetes organisations including the New Zealand Society for the Study of Diabetes (NZSSD) have issued consensus statements supporting the use of glycated haemoglobin (HbA1c) rather than blood glucose alone for the diagnosis of diabetes (and prediabetes).

NZGG notes that none of the international statements set out a strongly systematic method in the identification and critical appraisal of published research. As at December 2011, neither NZGG nor NZSSD have been funded to undertake a systematic review.

Nonetheless, HbA1c has low biological variability across patients and over time, there is a body of data relating HbA1c to microvascular outcomes, and standardisation in the measurement of HbA1c has improved. HbA1c is also more convenient for patients and practitioners since it avoids the need for fasting measurements and glucose tolerance testing.

This content on screening and diagnosis of diabetes is taken from the NZSSD *Position Statement on the diagnosis of, and screening for, type 2 diabetes* (September, 2011). The position statement is available at [www.nzssd.org.nz](http://www.nzssd.org.nz)

**The guidance does not apply to pregnant women and gestational diabetes.**

## Screening and diagnosis of type 2 diabetes continued...

	An HbA1c is the NZSSD-recommended diagnostic screening test for diagnosing diabetes. It should be measured by an accredited laboratory. Point-of-care assays are not sufficiently accurate for use in diagnosis
	If it is not possible to measure HbA1c or there are concerns about its validity, then a fasting plasma glucose is recommended
	HbA1c can be misleading in some circumstances (eg, falsely low in patients with increased red blood cell turnover or post blood transfusion, falsely high in some haemoglobinopathies, some ethnic differences in rate of Hb glycation)
	An oral glucose tolerance test (OGTT) should be used where there is uncertainty about the validity of HbA1c measures in specific patients (eg, in the presence of haemoglobinopathy or abnormal red cell turnover) or where there are special clinical reasons

<b>Table 28</b> Who should be screened for type 2 diabetes?	
People undergoing cardiovascular risk assessment	Table 1 specifies people requiring risk assessment and the age at which risk assessment should start
Other selected adults over 25 years	NZSSD recommends opportunistic screening for a person: <ul style="list-style-type: none"> <li>• with ischaemic heart disease (angina or myocardial infarction), cerebrovascular disease or peripheral vascular disease</li> <li>• on long-term steroid or antipsychotic treatment</li> </ul>
Obese children and young adults (BMI $\geq 30$ kg/m <sup>2</sup> or BMI $\geq 27$ kg/m <sup>2</sup> for Indo-Asian* peoples)	NZSSD recommends screening if: <ul style="list-style-type: none"> <li>• there is a family history of early onset type 2 diabetes; or</li> <li>• they are of Māori, Pacific or Indo-Asian* ethnicity</li> </ul>
* <b>Indo-Asian</b> Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani, Tibetan.	

Table 29

What to do following a screening test for type 2 diabetes

Result	Action	Why
<b>Symptomatic</b>		
<b>HbA1c <math>\geq 50</math> mmol/mol</b> <b>and</b> , if measured Fasting plasma glucose $\geq 7.0$ mmol/L <b>Or</b> Random plasma glucose $\geq 11.1$ mmol/L	No further tests required	Diabetes is confirmed
<b>Asymptomatic</b>		
<b>HbA1c <math>\geq 50</math> mmol/mol</b> <b>and</b> , if measured Fasting plasma glucose $\geq 7.0$ mmol/L <b>Or</b> Random plasma glucose $\geq 11.1$ mmol/L	Repeat HbA1c or a fasting plasma glucose	Two results above the diagnostic cut-offs, on separate occasions are required for the diagnosis of diabetes*
<b>HbA1c 41–49 mmol/mol</b> <b>and</b> , if measured Fasting plasma glucose 6.1–6.9 mmol/L	Advise on diet and lifestyle modification. If over 35 years, a full cardiovascular risk assessment and appropriate management is indicated  Repeat the test after 6–12 months	Results indicate 'prediabetes' or impaired fasting glucose*
<b>HbA1c <math>\leq 40</math> mmol/mol</b> <b>and</b> , if measured Fasting plasma glucose $\leq 6.0$ mmol/L	Retest at the next cardiovascular risk reassessment interval	This result is normal
* When HbA1c and fasting plasma glucose are discordant with regard to diagnosis of diabetes, repeat testing at an interval of 3–6 months is recommended. The test that is above the diagnostic cut point should be repeated – if the second test remains above the diagnostic threshold then diabetes is confirmed. If the second result is discordant with the first, then subsequent repeat testing at intervals of 3–6 months is recommended. Patients with discordant results are likely to have test results near the diagnostic threshold.		

Content on glucose testing, drawn from the 2009 edition of the *New Zealand Cardiovascular Guidelines Handbook*, is detailed in Appendix F.

NZSSD position statement advises that where glucose-based testing is used, the diagnostic criteria remain unchanged.

## Early identification of patients at high risk of diabetes-related complications

Determining level of risk for macrovascular and microvascular complications is a key component of treatment planning and target setting for each individual with type 2 diabetes.

- The risk of complications varies greatly across the diabetic population
- The aim is prevention of complications, especially targeting those at high risk
- Patients with **existing** complications (eg, foot, eye, kidney or cardiovascular disease) are in a high-risk category and should be managed intensively

See Figure 2 for information to assist identification of people with diabetes at high risk of diabetes-related complications. A cardiovascular risk assessment is also recommended to inform clinical management decisions.

For specific risk factors for foot complications see the box: Identifying high risk feet.

**Figure 2****Determining level of risk for diabetes-related complications****Low risk**

- HbA1c 50–55 mmol/mol\*
- BP <130/80 mm Hg\*
- ACR <2.5 mg/mmol in men or <3.5 mg/mmol in women
- eGFR  $\geq 60$  ml/min/1.73m<sup>2</sup>\*
- Lipids: triglycerides <1.7 mmol/L, total cholesterol <4.0 mmol/L
- Non-smoker
- Attends at least 6-monthly review of HbA1c and blood pressure; annual review of lipids, ACR, eGFR and foot check. Two-yearly retinal screening

**Moderate to high risk****High risk = 3 or more risk factors****Moderate risk = 2 risk factors**

- HbA1c > 55 mmol/mol. \* Risk increases incrementally with increasing HbA1c
- BP  $\geq 130/80$  mm Hg\*
- ACR  $\geq 2.5$  mg/mmol men, or  $\geq 3.5$  mg/mmol in women
- eGFR <60 ml/min/1.73m<sup>2</sup>\*
- Lipids: triglycerides  $\geq 1.7$  mmol/L, total cholesterol  $\geq 4.0$  mmol/L
- Current smoker
- Ethnicity (Māori, Pacific Islander, South Asian)
- Moderate retinopathy (R3), mild maculopathy (M3) – in either eye
- More than one year since diabetes last reviewed or poor adherence or attendance

\* Consider patient age. In younger people, tighter control should be considered given their higher lifetime risk of diabetes-related complications. Evidence suggests that a blood pressure target <120 mm Hg may be harmful. Care should be taken to estimate likely treatment response for patients when BP approaches the target of <130 mm Hg.

**Increasing risk for diabetes-related complications****Existing complications**

These place the person at **high risk** of developing more severe and/or additional complications:

- previous cardiac event or stroke/TIA
- eGFR <45 ml/min/1.73m<sup>2</sup> and/or ACR >30 mg/mmol
- severe retinopathy (R4), moderate maculopathy (M4) – in either eye
- previous amputation/ulceration
- peripheral arterial disease or previous leg vascular surgery

The aim of this chart is to assist the identification of people with diabetes at moderate to high risk of diabetes-related complications with a view to more intensive intervention and follow-up. The content of the chart is evidence-based. The quantification of risk reflects the consensus of the Diabetes Advisory Group convened by the New Zealand Guidelines Group.

### Figure 3 Management of people at moderate to high risk of diabetes-related complications

Urgent and intensive management is indicated to improve modifiable risk factors.

More frequent follow-up is recommended during treatment changes or if the parameter is much higher than target.

Management plan decisions should take into account patient preference, likely patient adherence and resource availability.

#### Lifestyle advice

- Offer evidence-based dietary advice including *achievable* goals (note 1). Dietitian advice should be sought if available
- Offer evidence-based advice on exercise including *achievable* goals (note 1)
- Offer ABC smoking cessation advice (note 2)

#### Medication adjustment/intensification

- Improve glycaemic control\* with adjustment of oral medication +/- insulin  
\* Refer to figure 5
- Control blood pressure\*\* through medication adjustment  
\*\* Refer to figure 4
- Improve lipid control with the use of statins (note 3)

#### Ongoing clinical review

- Monitor blood pressure, HbA1c and eGFR 3 monthly
- Monitor ACR 6 monthly (note 4)
- Review annually: weight, peripheral neurovascular status, cardiovascular status (clinical examination and cardiovascular risk calculation), feet. Review feet 3 monthly if at high risk for foot complications
- Screen retina 2 yearly as a minimum, at least annually if diabetic retinopathy present
- Seek specialist advice for newly-diagnosed complications or treatment resistance

#### Practice management

- Access long-term conditions funding to develop a wellness plan and promote regular follow-up
- Review nurse responsibilities and role in regular monitoring
- Set up computerised reminders to recall patients if these are not already in place
- Monitor patient risk profiles using a practice diabetes register

**Note 1.** See Chapter 5 *Weight management*.

**Note 2.** See section 'Smoking cessation interventions' in Chapter 2.

**Note 3.** See section 'Lipid modification' in Chapter 2.

**Note 4.** Unless eGFR  $<60\text{ml}/\text{min}/1.73\text{m}^2$  or frank proteinuria (24 h urine  $>1\text{ g}$  per day or urine protein creatinine ratio  $>100\text{ mg}/\text{mmol}$ ).

## Identifying high risk feet

Risk factors for diabetic foot disease include:

- peripheral vascular disease (PVD)\*
- peripheral neuropathy
- previous amputation
- previous ulceration
- presence of callus
- joint deformity
- visual/mobility problems.

\* Risk factors for PVD are smoking, hypertension and hypercholesterolaemia. The cumulative effect of these risk factors for PVD is considered to be at least additive.

Appropriate footwear is recognised in the literature as an important part of management to prevent diabetic foot disease.

## Approach to setting treatment targets

- Setting treatment targets is an important component of diabetes management for all patients
- Targets given for specific parameters are based on best available evidence but should be appropriate for the individual patient

### Treatment targets

Treatment targets to address risk factors:

- should be appropriate for and agreed with the individual patient
- glycaemic control target: HbA1c 50–55 mmol/mol  
or as individually agreed
- blood pressure target: <130/80 mm Hg. Evidence suggests a BP target <120 mm Hg may be harmful. Care should be taken to estimate likely treatment response for patients when BP approaches the target of <130 mm Hg
- lipids target: triglycerides <1.7 mmol/L; total cholesterol <4.0 mmol/L.

For lipid management including the guidance on the use of statins see section 'Lipid modification' in Chapter 2.

## Better management of raised blood pressure and microalbuminuria

See Figure 4 for a summary algorithm of recommended management of raised blood pressure and microalbuminuria for people with type 2 diabetes.

### Achieving target blood pressure

- Target systolic blood pressure is  $<130$  mm Hg and target diastolic blood pressure is  $\leq 80$  mm Hg
- Hypertension should be treated aggressively with lifestyle modification including dietary salt restriction and drug therapy
- 'Aggressively' should be interpreted as the initiation and intensification of lifestyle and pharmacological therapy not a recommendation to attempt to lower systolic blood pressure well below 130 mm Hg
- Evidence from the Accord Study Group in 2010 indicates a greater frequency of serious adverse effects where the systolic blood pressure target is  $<120$  mm Hg
- The recommended blood pressure target may not be appropriate for specific patients and should not be pursued in patients with a short life expectancy or who are at significant risk of hypotension

See Appendix C for the recommended method of blood pressure measurement.

## Microalbuminuria: monitoring and management

- Microalbuminuria is the earliest sign of diabetic kidney disease and should be treated promptly if identified
- Younger people with type 2 diabetes have a higher lifetime risk of renal complications
- Annual screening for microalbuminuria using albumin:creatinine ratio (ACR) measurement is recommended. More frequent monitoring of renal status is indicated for Māori, Pacific Island and South Asian peoples
- Those at moderate to high risk of diabetes-related complications (see Figure 3) should have their ACR measured 6 monthly
- Patients with confirmed microalbuminuria should be treated with an ACE inhibitor or angiotensin 2 receptor blocker (ARB) whether or not hypertension is present
- Combination ACE inhibitor and ARB therapy should not be used without recommendation of a diabetes or renal specialist
- Use of loop diuretics instead of or in combination with thiazide diuretics is considered appropriate for patients with significant renal impairment (eGFR <45 ml/min/1.73m<sup>2</sup>)

## Modification to diet

See Appendix E for details of the New Zealand Cardioprotective Dietary Pattern. This contains information on appropriate serving sizes, including servings of salt.

- Restricting dietary salt intake is important in the management of hypertension
- Reducing daily salt intake by 5 g/day (a teaspoon) on average reduces blood pressure by 5/3 mm Hg
- A suggested dietary target for daily sodium intake is 1600 mg (4 g of salt)

## Figure 4 Management of raised blood pressure and microalbuminuria in type 2 diabetes

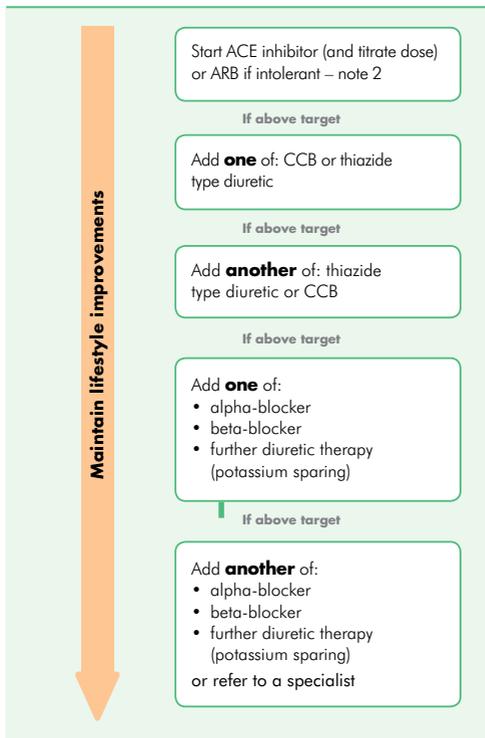
**Target BP is <130/80 mm Hg – note 1**

Hypertension should be treated aggressively with lifestyle modification including dietary salt restriction and drug therapy.

Evidence suggests a blood pressure target <120 mm Hg may be harmful. Care should be taken to estimate likely treatment response for patients when BP approaches the target of <130 mm Hg.

### Start drug therapy if:

BP >130/80 mm Hg consistently for 3 months despite attempts at lifestyle modification



**Note 1.** Consider patient age. In younger people tighter control should be considered given their higher lifetime risk of diabetes-related complications.

**Note 2.** ACE inhibitor or ARB medication are contraindicated in pregnancy.

\* Consensus of NZGG Diabetes Advisory Group

### Approach to management

If hypertensive, intensive monthly follow-up and stepwise protocol adjustments to medication are advised until consistently below target.

BP should be reviewed at least 6 monthly once at target.

Refer to Appendix C for the recommended method of BP measurement.

### Renal disease

Microalbuminuria is confirmed if, in the absence of infection or overt proteinuria, two out of three specimens have an elevated ACR.

People with confirmed microalbuminuria should be treated with an ACE inhibitor or an ARB whether or not hypertension is present.

Māori, Pacific Island and South Asian peoples are at a higher risk of renal complications. More frequent monitoring of renal status is indicated.

Any evidence of renal disease based on decreasing eGFR should be treated with urgency.

Loop diuretics may be used instead of or in combination with thiazide diuretics in patients with significant renal impairment (eGFR <45 ml/min/1.73m<sup>2</sup>)\*.

**ACE Inhibitor:** angiotensin converting enzyme inhibitor

**ARB:** angiotensin 2 receptor blocker

**CCB:** calcium channel blocker

**Source:** National Institute of Clinical Excellence (2008). Adapted with permission by the New Zealand Guidelines Group Diabetes Advisory Group from CG 66 Type 2 diabetes: National clinical guideline for management in primary and secondary care (update). London: NICE. Content consistent with SIGN Guideline 116, 2010.

## Improved glycaemic control

Good glycaemic control has a clear benefit on microvascular outcomes and if started early enough, on long-term macrovascular outcomes.

Treatment targets should be set for an individual in order to balance benefits with harms, in particular hypoglycaemia and weight gain.

- A target of HbA1c 50–55 mmol/mol is recommended *or as individually agreed*
- It is important to consider patient age. In younger people, tighter control should be considered given their higher lifetime risk of diabetes-related complications
- Any reduction in HbA1c is beneficial

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**Note:** Since October 2011 New Zealand laboratories report HbA1c values only in IFCC-aligned format (molar units measured in mmol/mol), not in DCCT-aligned format (measured in percentage). Appendix G provides a conversion table for HbA1c formats.

### Setting a target HbA1c for a patient

Take into account for that individual:

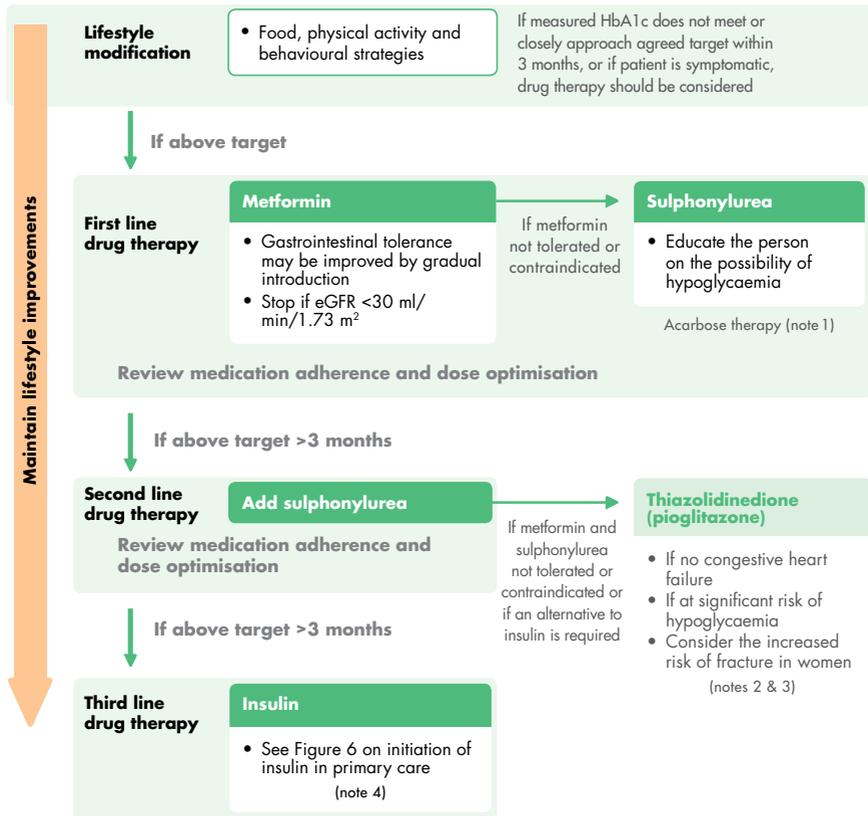
- risk of microvascular and macrovascular complications (see section ‘Early identification of patients at high risk of diabetes-related complications’ in this chapter)
- risk and consequences of hypoglycaemia, and weight gain
- personal preferences of the individual with respect to managing diabetes and preventing complications.

See Figure 5 for a summary algorithm outlining appropriate management of glycaemic control for people with type 2 diabetes. The value of using proven agents, such as metformin, sulphonylureas and insulin is emphasised.

More intensive treatment may be required as the condition progresses to achieve the target HbA1c. It is important to make the individual aware that insulin is likely to be required as future treatment and to prepare them for this eventuality well in advance.

For guidance on specific lifestyle modification strategies including diet and physical activity see Chapter 5 *Weight management*.

**Figure 5** Management of glycaemic control

**Target HbA1c 50–55 mmol/mol or as individually agreed**


**Note 1.** Acarbose can also be used as a first line drug therapy, if tolerated.

**Note 2.** Medsafe is currently monitoring the safety of pioglitazone following reports of increased adverse effects. See [www.medsafe.govt.nz](http://www.medsafe.govt.nz) for latest updates. Special authority for pioglitazone may be sought if: i) patient has not achieved glycaemic control on maximum dose of metformin or sulphonylurea or where either or both are contraindicated or not tolerated; or ii) patient is on insulin.

**Note 3.** DPP-IV inhibitor may be an alternative agent if patient is at significant risk of hypoglycaemia or weight gain is a concern. At time of publication (2011), DPP-IV inhibitors are not subsidised.

**Note 4.** DPP-IV inhibitor and GLP-1 agonist are possible alternatives. GLP-1 agonists may be used if BMI >30 kg/m<sup>2</sup> or there is a desire to lose weight. At time of publication (2011), neither DPP-IV inhibitors nor GLP-1 agonists are subsidised.

**Source:** Scottish Intercollegiate Guidelines Network (2010). Adapted with permission by the New Zealand Guidelines Group Diabetes Advisory Group from SIGN 116: *Management of diabetes: a national clinical guideline*. Edinburgh: SIGN.

## Self-monitoring blood glucose

Benefits of self-monitoring blood glucose (SMBG) by people with type 2 include:

- assisting patients and health practitioners in adjustment of insulin or other medication
- encouraging self-empowerment
- promoting better self-management behaviours.

However, self-monitoring may fail to improve diabetes control and negative psychological outcomes have been reported in some studies.

See Table 30 for guidance on when SMBG is recommended.

<b>Table 30 Recommended use of self-monitoring blood glucose</b>	
<b>Medication used as treatment</b>	<b>Is SMBG recommended?</b>
Insulin	Yes
Sulphonylureas	Yes. If the patient is motivated they may benefit from routine SMBG to reduce risk of hypoglycaemia
Metformin and other oral hypoglycaemic agents	In general SMBG is NOT recommended, but there are specific occasions when SMBG may be considered for those: <ul style="list-style-type: none"> <li>• at increased risk of hypoglycaemia</li> <li>• experiencing acute illness</li> <li>• undergoing significant changes in pharmacotherapy or fasting eg, during Ramadan</li> <li>• with unstable or poor glycaemic control (HbA1c &gt;64 mmol/mol)</li> <li>• who are pregnant or planning pregnancy</li> </ul>
<b>Source:</b> SIGN guideline 116 <i>Management of Diabetes</i> (2010)	

SMBG may be of value to individuals with newly-diagnosed type 2 diabetes who as part of self-management wish to determine the effect of changes to food or exercise on their blood glucose levels. Selected individuals may benefit from continuing SMBG where this is having a positive impact on their management.

For information on SMBG in relation to insulin therapy see section 'Insulin initiation' in this chapter.

## Insulin initiation

This content is a guide. Seek specialist advice to support patient management as needed.

Assist the individual to understand their insulin regimen and encourage them to take an active role in management during the initiation of insulin.

### When to consider insulin

Consider insulin therapy if the individual with type 2 diabetes has unsatisfactory glycaemic control (measured HbA1c does not meet or closely approach agreed target\*) or there are signs and symptoms of hyperglycaemia despite:

- management including appropriate food/diet, physical exercise and behavioural strategies (see Chapter 5 *Weight management*)
- review of medication adherence and dose optimisation of oral hypoglycaemic agents (see Figure 5).

People who have an HbA1c above 65 mmol/mol should be seriously considered for insulin therapy.

\* Target HbA1c is 50–55 mmol/mol or as individually agreed.

Assess the individual's readiness for commencing insulin therapy and address any patient concerns (see Appendix H). See Appendix I for a patient education checklist for practitioners relating to initiation of insulin therapy.

## Assess blood glucose profile

### Prior to initiating insulin therapy

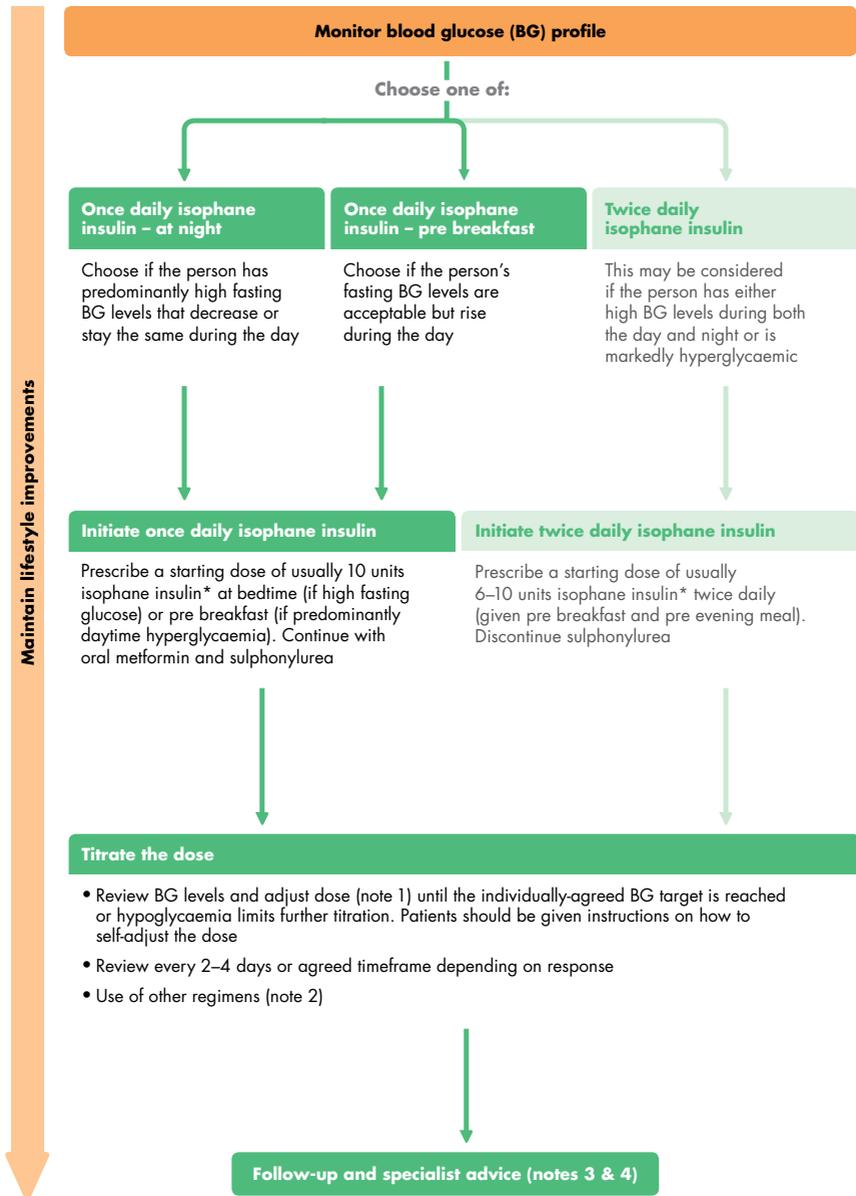
- It is essential that the patient regularly self-monitors blood glucose levels to assist decision-making about an appropriate insulin regimen

#### Assessing blood glucose profile: practice points

- Educate the patient on how to measure blood glucose levels using a meter and how to record results using a log book (see Appendix J for an example) to determine their current blood glucose profile
- Review recorded blood glucose results with the patient to identify their current blood glucose profile and 'problem' times of the day
- Use their blood glucose profile to help you and the patient decide on an appropriate insulin regimen (see Appendix J, which includes a logbook interpretation as an example)

## Insulin therapy

See Figure 6 for a summary algorithm outlining the recommended approach to initiating insulin for a patient with type 2 diabetes in the primary care setting.

**Figure 6** Initiation of insulin in primary care


\* Currently funded isophane insulin is Protaphane or Humulin NPH

continued over...

Table 1. Once daily isophane insulin – at night

Pre breakfast (fasting) BG	Insulin dose increase
Usually >8 mmol/L and never less than 4 mmol/L	Increase dose by 4–6 units
Usually 6–8 mmol/L and never less than 4 mmol/L	Increase dose by 2–4 units
<b>Once receiving &gt;20 units daily</b> 3 consecutive pre breakfast (fasting) BG results higher than agreed BG target AND BG never less than 4 mmol/L	Insulin dose can be increased by 10–20% of total daily dose

Table 2. Once daily isophane insulin – pre breakfast

Pre evening meal BG	Insulin dose increase
Usually >8 mmol/L and never less than 4 mmol/L	Increase dose by 4–6 units
Usually 7–8 mmol/L and never less than 4 mmol/L	Increase dose by 2–4 units
<b>Once receiving &gt;20 units daily</b> 3 consecutive pre evening meal BG results higher than agreed BG target AND BG never less than 4 mmol/L	Insulin dose can be increased by 10–20% of total daily dose

Table 3. Twice daily isophane insulin

Pre breakfast (fasting) BG	Insulin dose increase
Usually >8 mmol/L and never less than 4 mmol/L	Increase night-time insulin dose by 4–5 units
Usually 6–8 mmol/L and never less than 4 mmol/L	Increase night-time insulin dose by 2–4 units
Pre evening meal BG	Insulin dose increase
Usually >8 mmol/L and never less than 4 mmol/L	Increase pre breakfast insulin dose by 4–5 units
Usually 7–8 mmol/L and never less than 4 mmol/L	Increase pre breakfast insulin dose by 2–4 units
<b>Once receiving &gt;20 units daily</b> 3 consecutive BG results (either pre breakfast or pre evening meal) higher than agreed BG target AND BG never less than 4 mmol/L	Appropriate insulin dose can be increased by 10–20% of total daily dose

<b>Note 2</b>	<b>Other regimens</b>
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- Basal insulin analogues should be considered if there are concerns regarding hypoglycaemia
- Premixed insulin can be considered if post prandial levels are elevated and HbA1c target has not been met
  - Consider seeking specialist advice if instigating a premixed insulin regimen
- The option of adding short-acting insulin relates to the intensification of insulin therapy and is not covered in this guidance

<b>Note 3</b>	<b>Follow-up</b>
---------------	------------------

- Review BG levels every 2–4 days, depending on the individual and response
- Once BG levels are stable, re-evaluate BG profile regularly (3–6 monthly) and change regimen if required
- Check for risk of hypoglycaemia
- Measure HbA1c 3–6 monthly, according to individual need
- Monitor weight (if gaining weight, review lifestyle advice)

<b>Note 4</b>	<b>Specialist advice</b>
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Seek specialist advice when:

- patient is very lean or has experienced rapid weight loss
- HbA1c persistently above individual target despite initiation of insulin, titration, and review of lifestyle modification
- patient has recurrent hypoglycaemia
- patient is an adolescent or child with type 2 diabetes
- patient is a vocational driver.

## When initiating insulin therapy for a given patient

- Ensure that the patient understands that the initial insulin dose is a starting point for dose titration
- Discuss and agree on the frequency of follow-up

## Isophane insulin

- Once daily isophane insulin\* at night (or pre breakfast if the patient has daytime hyperglycaemia) should be used when adding insulin to metformin and/or sulphonylurea therapy
- Twice daily isophane insulin\* may be considered if the person has high blood glucose levels during both the day and night or is markedly hyperglycaemic. When prescribing twice daily insulin therapy sulphonylurea therapy should be stopped

\* Currently funded isophane insulin is Protaphane or Humulin NPH.

## Other regimens

- Basal insulin analogues should be considered if there are concerns regarding hypoglycaemia
- Premixed insulin can be considered if post prandial levels are elevated and the HbA1c target has not been met
  - Consider seeking specialist advice if instigating a premixed insulin regimen
- The option of adding short-acting insulin relates to the intensification of insulin therapy and is not included in this guidance

## Maintenance self-monitoring blood glucose

Frequency of blood glucose testing can be reduced once the patient is established on insulin and blood glucose levels are stable, but should still be such as to show the blood glucose profile over the course of the day (see Appendix J).

- If the patient chooses to test less frequently, ask them to vary testing across different times of the day
- Patients may choose to test in other patterns, eg, 4 times a day on one or two days of the week

Maintenance SMBG can be combined with checking HbA1c levels (3–6 monthly) to assess glycaemic control and the need for medication changes.

# 5 Weight management

The following content on weight management is taken from the summary key messages and algorithms section of *Clinical Guidelines for Weight Management in New Zealand Adults (2009)*. The full guideline is available on the Ministry of Health website [www.health.govt.nz](http://www.health.govt.nz) – search on title.

**Note:** Guidance on the use of pharmacotherapy for weight management has been updated (see Jull A, Lawes CMM Eyles H, et al, *Journal of Primary Health Care* 2011;3:66-71) since publication of the guideline to reflect the product withdrawal of sibutramine from the market. Content in this Handbook reflects this later guidance on pharmacotherapy.

For specific guidance on weight management in children and young people see *Clinical Guidelines for Weight Management in New Zealand Children and Young People* at [www.health.govt.nz](http://www.health.govt.nz) – search on title.



Overweight and obesity increase the risk of mortality and morbidity, particularly from cardiovascular disease, some cancers, type 2 diabetes, as well as other comorbidities



Reducing the risks of excess weight is largely about changing lifestyle and behaviour

## Step 1: Engage with the person and raise awareness

- Measure body mass index (BMI) as part of routine practice for estimate of risk (use Table 31)

Classification	Body mass index kg/m <sup>2</sup>	Disease risk	
		Waist ♂ 94–102 cm ♀ 80–88 cm	Waist ♂ > 102 cm ♀ > 88 cm
Normal	18.5–24.9	–	–
Overweight	25.0–29.9	+	++
Obese I	30.0–34.9	++	+++
Obese II	35.0–39.9	+++	+++
Obese III	40.0+	++++	++++

+ Increased risk; ++ High risk; +++ Very high risk; ++++ Extremely high risk.

**Note:** BMI may not be as accurate in highly muscular people or in ethnic groups with smaller body stature. (Therefore, in South Asians, for example, consider lowering the treatment threshold in the presence of central fatness or additional risk factors.)

## Step 2: Identify need and context for action

If the person is in a high-risk category, assess the person's lived realities and clinical need.

- Consider the person's:
  - family/whānau, culture, work, and community, beliefs and values
  - weight-related concerns and previous experiences with weight loss
  - nutrition and activities of choice
  - age, sex, and ethnicity (Māori, Pacific and South Asian population groups)
  - family history of cardiovascular disease
  - smoking status
  - blood pressure and lipid profile
  - common comorbidities (eg, diabetes)
  - psychiatric history and use of anti-psychotics or mood stabilisers.
- Discuss risks and motivations for action and use other guidelines as required

## Step 3: Determine options for action

- The most effective approach to weight loss uses three key interventions in combination, called 'the FAB approach':
  - changes to **food**/diet
  - increased **physical activity**
  - **behavioural strategies**.
- The only effective approach to weight management is a permanent change to how people live their lives
- A realistic target for weight loss varies by individual. Benefits start to accrue when 5–10% of initial body weight is lost. Aim for a modest weekly weight loss
- Consider referral to professional and community providers

### Diet

- Low energy, low glycaemic index/load, and modified macronutrient approaches (ie, low carbohydrate, low fat, high protein or high carbohydrate diets) are all similarly effective for weight loss providing the diet results in some energy restriction
- Consider sustainability and the individual's preference for diet and that of their family/whānau. Do not use fad diets
- Very low energy diets require close supervision

### Physical activity

- For weight loss, aim to increase periods of physical activity to at least 60 minutes every day. Start with small achievable goals (eg, 5–10 minutes per day) and build up to target
- Reduce screen time (eg, watching television, videos and DVDs, and using the computer)

### Behavioural strategies

- Include the person's partner and family/whānau in the person's weight management plan
- Identify the changes the person/whānau wishes to work on first. Use problem-solving and goal-setting strategies to achieve changes

### Pharmaceuticals

- Consider anti-obesity drugs when BMI  $\geq 30$  kg/m<sup>2</sup>
- Note that anti-obesity drugs, such as orlistat, must be used in conjunction with lifestyle changes. Counsel a low-fat diet when considering orlistat

### Surgery

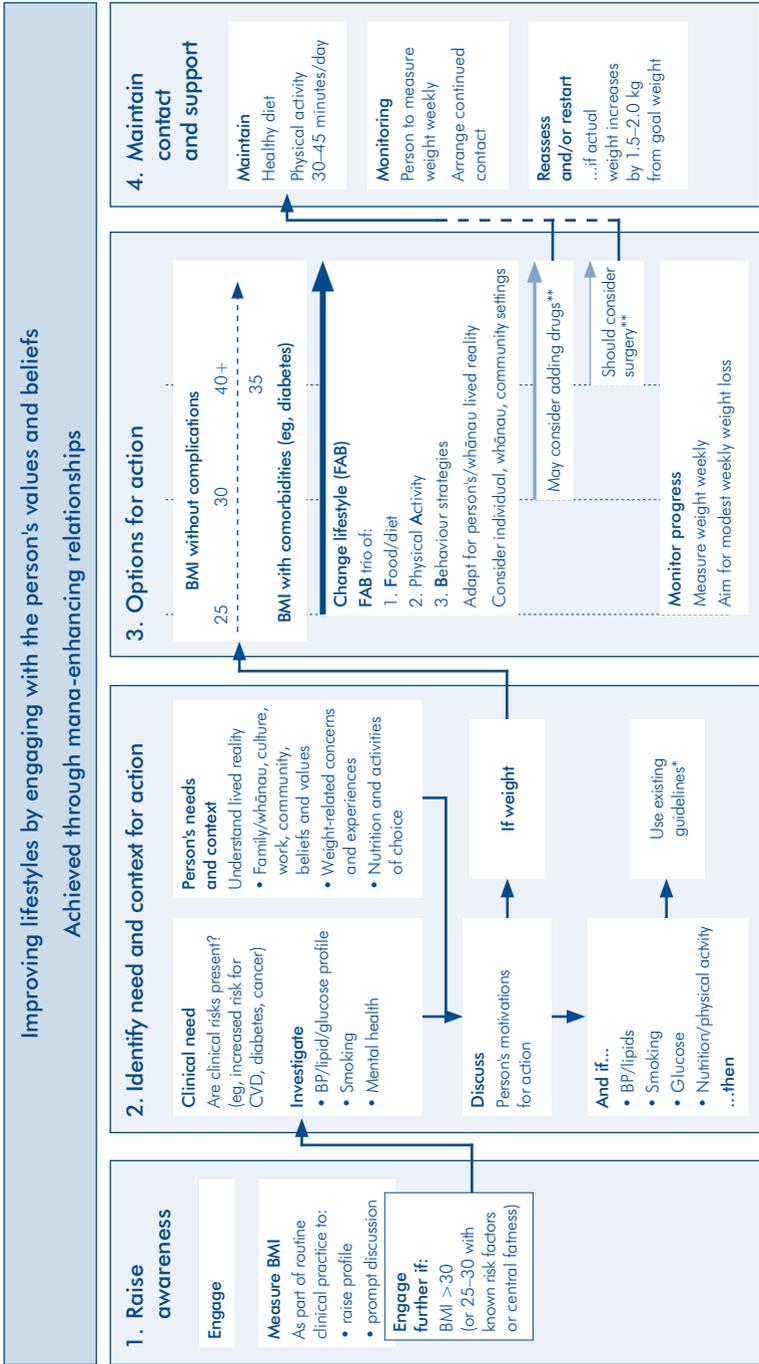
- Consider referral for bariatric surgery when BMI  $\geq 40$  kg/m<sup>2</sup> or  $\geq 35$  kg/m<sup>2</sup> with significant comorbidities

## Step 4: Arrange ongoing contact and support (once reach goal weight)

- Make arrangements to reinforce lifestyle change through regular brief contact (eg, ongoing clinical, family/whānau or community contact)
- Encourage the person to weigh themselves regularly (eg, weekly) and have strategies to manage weight regain
- Encourage the person to maintain a healthy diet and to do at least 30–45 minutes physical activity every day
- Restart weight management programme immediately, if person's weight regain increases 1.5–2.0 kg over goal weight
- Consider anti-obesity drugs for weight-loss maintenance

Figure 7 provides a summary algorithm for weight management in adults.

**Figure 7** Algorithm for weight management in adults



\* New Zealand Primary Care Handbook 2012 (NZCG, 2012); New Zealand Physical Activity Guidelines (Sport and Recreation New Zealand, 2005); Food and Nutrition Guidelines for Healthy Adults (Ministry of Health, 2003).

\*\* Drugs and surgery only used in addition to lifestyle changes when other attempts have failed. They are not suitable for lifestyle change.

# 6 Stroke and transient ischaemic attack

This updated content on stroke and transient ischaemic attack (TIA) is a summary derived by the Stroke Foundation of New Zealand from *New Zealand Clinical Guidelines for Stroke Management* (2010) and *New Zealand Guideline for the Assessment and Management of Transient Ischaemic Attack* (2008). The full guidelines are available on the Stroke Foundation website [www.stroke.org.nz](http://www.stroke.org.nz) – search on title.

## Transient ischaemic attack

**Transient Ischaemic Attack is a medical emergency – people with TIA are at high risk of early stroke.**

- This risk can be as high as 12% at 7 days and 20% at 90 days
- About half of these strokes will occur within the first 48 hours after TIA
- Up to 85% of strokes that follow TIA will be fatal or disabling

This risk is higher than that for chest pain. TIA warrants urgent attention.

The ABCD<sup>2</sup> tool (see Figure 8) can identify people with TIA most at risk; usually those with unilateral weakness and/or speech disturbance, especially if symptoms last more than 60 minutes.

**A diagnosis of TIA is more likely to be correct if the history confirms:**

- sudden onset of symptoms, with maximal neurological deficit at onset
- symptoms typical of focal loss of brain function, such as unilateral weakness or speech disturbance
- rapid recovery of symptoms, usually within 30–60 minutes; if the patient still has **any** residual symptoms or signs at the time of assessment, they should be managed for **stroke** (see section ‘Acute Stroke’ in this chapter), not TIA.

## Immediate assessment and intervention

Urgent assessment and intervention reduces the risk of stroke after TIA.

### Aspirin

Aspirin should be started immediately if fully recovered and no contraindications; 300 mg stat if aspirin naïve and 75–150 mg daily.

### Risk assessment

All people with suspected TIA should be assessed at initial point of health care contact for their risk of stroke, including their ABCD<sup>2</sup> score (see Figure 8).

#### People at high risk

- Include those with ABCD<sup>2</sup> scores of 4 or more, crescendo TIAs, atrial fibrillation or who are taking anticoagulants
- Require urgent specialist assessment as soon as possible but definitely within 24 hours

#### People at low risk

- Include those with ABCD<sup>2</sup> scores of less than 4 or those who present more than one week after TIA symptoms
- Require specialist assessment and investigations within 7 days

If the treating doctor is confident of the diagnosis of TIA, has ready access to brain and carotid imaging, and can initiate treatment, then specialist review may not be required.

**Figure 8** ABCD<sup>2</sup> tool: assessment of stroke risk**ABCD<sup>2</sup> score – prediction of stroke risk after transient ischaemic attack**

ABCD <sup>2</sup> items (score: 0–7)		Points
<b>A</b>	<b>Age:</b> ≥60 years	1
<b>B</b>	<b>Blood pressure:</b> ≥140/90 mm Hg	1
<b>C</b>	<b>Clinical features:</b>	
	unilateral weakness; or speech impairment without weakness	2 1
<b>D</b>	<b>Duration of symptoms:</b>	
	≥60 minutes; or 10–59 minutes	2 1
<b>D</b>	<b>Diabetes: (on medication/insulin)</b>	1

**Risk of stroke according to ABCD<sup>2</sup> scores**

ABCD <sup>2</sup> score	0–3	4–5	6–7
<b>Proportion of all TIAs</b>	34%	45%	21%
<b>Stroke risk (%) at:</b>			
2 days	1.0	4.1	8.1
7 days	1.2	5.9	11.7
90 days	3.1	9.8	17.8

**Source:** Johnston SC, et al. Lancet 2007; 369:283–92. Reproduced from *New Zealand Guideline for the Assessment and Management of Transient Ischaemic Attack* (2008).

## Immediate secondary prevention measures

As soon as the diagnosis is confirmed all people with TIA should have their risk factors addressed and be established on an appropriate individual combination of secondary prevention measures including:

- antiplatelet agent(s) – aspirin, aspirin plus dipyridamole or clopidogrel
- blood pressure lowering therapy
- statin
- anticoagulation therapy – if atrial fibrillation or other cardiac source of emboli and no contraindication. For further details see section ‘Long-term secondary prevention for stroke and TIA’ in this chapter. Note that brain imaging is required prior to commencement of anticoagulation therapy
- nicotine replacement therapy or other smoking cessation aid.

**These treatments should be initiated at the first point of health care contact to prevent early risk of stroke.** Stroke secondary prevention medications should be commenced **immediately** and apart from anticoagulation therapy, do not need to wait for brain imaging to be performed. This applies only to patients who have **fully recovered** rapidly, for whom the risk of intracerebral haemorrhage (ICH) is very low. Patients who have any residual signs or symptoms at the time of assessment should be managed as for **stroke** (see section ‘Acute stroke’ in this chapter).

## Follow-up

Follow-up, either in primary or secondary care, should occur within one month so that medication and other risk factor modification can be reassessed.

## Early assessment and diagnosis

All patients with suspected TIA should have a full assessment that includes thorough history and clinical, prognostic (eg, ABCD<sup>2</sup> score) and investigative tests (eg, blood tests, brain and carotid imaging and ECG) at the initial point of health care contact whether first seen in primary or secondary care.

The following investigations should be undertaken routinely for all patients with suspected TIA: full blood count, electrolytes, erythrocyte sedimentation rate (ESR), renal function tests, lipid profile, glucose level, and ECG.

Patients classified as high risk (ABCD<sup>2</sup> 4–7 or those with any one of the following: AF, tight carotid stenosis, or crescendo TIA) should have urgent brain imaging (preferably MRI); ('urgent' is immediately where available, but within 24 hours). Carotid imaging should also be undertaken urgently (within 24 hours) in patients with carotid territory symptoms who would potentially be candidates for carotid revascularisation. In settings with limited access to these investigations, referral within 24 hours should be made to the nearest centre where such tests can be quickly conducted.

Patients classified as low risk (ABCD<sup>2</sup> 0–3 or late presentations, ie, after a week) should have brain and carotid imaging (where indicated) within 7 days.

## Acute stroke

All patients with suspected stroke should be referred urgently for specialist care. Same-day admission to hospital is recommended for all patients, with the possible exception of some for whom a palliative approach is deemed appropriate.

## Stroke thrombolysis

Patients who present to primary care within 4.5 hours of symptom onset may be candidates for stroke thrombolysis. Immediate transfer to hospital is required.

## Brain imaging

All patients with stroke should have an urgent brain CT or MRI ('urgent' is immediately where available, but within 24 hours). Patients who are candidates for thrombolysis should undergo brain imaging immediately. Urgent brain imaging is required, even for patients with relatively mild stroke, to exclude intracerebral haemorrhage (ICH) and to allow appropriate secondary prevention treatments to be initiated rapidly. Patients with TIA (who by definition have completely recovered rapidly) have a very low risk of ICH and should have secondary prevention treatments initiated immediately, and with the exception of anticoagulation therapy, without waiting for brain imaging.

## Other acute stroke management

Other aspects of acute stroke management are generally performed in a hospital setting and are beyond the scope of this Handbook. Details can be found in the full guideline: *New Zealand Clinical Guidelines for Stroke Management* (2010) available at [www.stroke.org.nz](http://www.stroke.org.nz) – search on title.

# Long-term secondary prevention for stroke and TIA

## Lifestyle modifications

Every person with stroke or TIA should be assessed and informed of their risk factors for a further stroke and possible strategies to modify identified risk factors. Interventions for risk factors include:

- smoking cessation
- improving diet
- increasing regular exercise.

## Blood pressure lowering recommendations

All patients after stroke or TIA, **whether normotensive or hypertensive**, and whether already taking antihypertensive medications or treatment-naïve should receive new blood pressure lowering therapy for secondary prevention, unless contraindicated by symptomatic hypotension. The most direct evidence of benefit is for the use of an ACE inhibitor (alone or in combination with a diuretic); however, different agents have generally been found to be effective in stroke prevention, with the exception of beta-blockers (Lakhan & Sapko 2009; Rashid, et al 2003; see full guideline for details of references).

New blood pressure lowering therapy should commence prior to discharge for those with stroke or TIA or soon after TIA if the patient is not admitted.

Cautious introduction of BP lowering medication may be required in older people with frailty due to risk of complications such as symptomatic hypotension.

## Antiplatelet therapy

Long-term antiplatelet therapy should be prescribed to all people with ischaemic stroke or TIA who are not prescribed anticoagulation therapy.

Low dose aspirin and modified release dipyridamole or clopidogrel alone should be prescribed to all people with ischaemic stroke or TIA taking into consideration patient comorbidities.

Aspirin alone can also be used, particularly in patients who do not tolerate aspirin plus dipyridamole or clopidogrel.

The combination of aspirin plus clopidogrel is **not** recommended for the long-term secondary prevention of cerebrovascular disease in patients who do not have acute coronary disease or recent coronary stent.

## Anticoagulation therapy

### Anticoagulation therapy after ischaemic stroke

- Anticoagulation therapy for secondary prevention for those people with ischaemic stroke or TIA from presumed arterial origin should **not** be routinely used as there is no evidence of additional benefits over antiplatelet therapy
- Anticoagulation therapy for long-term secondary prevention should be used in all people with ischaemic stroke or TIA who have atrial fibrillation or cardioembolic stroke and no contraindication
- In patients with TIA, commencement of anticoagulation therapy should occur once CT or MRI has excluded intracranial haemorrhage as the cause of the current event

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**Note:** Dabigatran is available in New Zealand as an alternative to warfarin for prevention of stroke in people with non-valvular atrial fibrillation. Dabigatran is contraindicated in severe renal impairment and should be used with caution in people over 80 years. At the time of publication, clinical experience with dabigatran is limited and data on longer-term safety is lacking.

### Anticoagulation after intracerebral haemorrhage

- All patients with ICH should have their individual risk of future thromboembolic events and their risk of recurrent ICH assessed, taking into account patient specific factors
- The risk of recurrent ICH is thought to be greatest in those with lobar ICH and less with deep 'hypertensive ICH' when blood-pressure control can be optimised. In general, thromboembolism risk is highest in patients with mechanical heart valves (particularly mitral valves), and is high in those with atrial fibrillation and patients with previous ischaemic events
- Expert advice should be sought and the potential benefits and risks of anticoagulant and antiplatelet therapy after ICH discussed with patients and their families, and documented

## Cholesterol lowering

Therapy with a statin should be considered for all patients with ischaemic stroke or TIA.

Statins should **not** be used routinely for patients with intracerebral haemorrhage.

## Diabetes management

Patients with glucose intolerance or diabetes should be managed in line with appropriate guidelines for diabetes.

## Carotid surgery

Carotid endarterectomy should be undertaken in patients with non-disabling carotid artery territory ischaemic stroke or TIA with ipsilateral carotid stenosis measured at 70–99% (NASCET criteria) if it can be performed by a specialist surgeon with low rates (<6%) of peri-operative mortality/morbidity. Carotid endarterectomy can be undertaken in highly selected ischaemic stroke or TIA patients (considering age, gender and comorbidities) with symptomatic carotid stenosis of 50–69% (NASCET criteria) or asymptomatic carotid stenosis >60% (NASCET criteria) only if it can be performed by a specialist surgeon with very low rates (<3%) of peri-operative mortality/morbidity.

Eligible stable patients should undergo carotid endarterectomy as soon as possible after stroke event (ideally within two weeks).

## Stroke rehabilitation, recovery and community participation

Detailed content on these topics is included in the full guideline *New Zealand Clinical Guidelines for Stroke Management (2010)* available at [www.stroke.org.nz](http://www.stroke.org.nz) – search on title.

# 7 Heart failure

The following summary content on clinical evaluation, brain natriuretic peptide and the management of heart failure with left ventricular systolic dysfunction was derived by the Heart Foundation from the updated *New Zealand Guideline for Management of Chronic Heart Failure (2009)*. The full guideline is available on the Heart Foundation website [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz) – search on title.

## Clinical evaluation

Table 32	Clinical evaluation
<b>Recommendation</b>	
<p>Evaluation for heart failure should be undertaken in all patients who complain of new-onset shortness of breath on exertion, orthopnoea or paroxysmal nocturnal shortness of breath unless history and physical examination clearly indicate a non-cardiac cause for their symptoms</p> <p>The most specific signs of heart failure are elevated jugular venous pressure, a third heart sound and a laterally displaced apical impulse, and are virtually diagnostic in a patient with compatible symptoms</p>	
<b>Clinical Practice Points</b>	
<ul style="list-style-type: none"> <li>• Diagnosis of the clinical syndrome of heart failure can be difficult, especially in patients who are elderly, obese or with comorbidities, and when a patient presents with milder symptoms in the community</li> <li>• Careful attention should be given to obtaining history of causative factors for heart failure, including a history of any of the following:             <ul style="list-style-type: none"> <li>– hypertension</li> <li>– myocardial infarction</li> <li>– valvular heart disease</li> <li>– atrial fibrillation</li> </ul> </li> <li>• Exertional shortness of breath and ankle swelling are common symptoms which can be due to a variety of conditions and, alone, have low specificity for heart failure</li> <li>• Orthopnoea and paroxysmal nocturnal shortness of breath are features of more marked decompensation and are more specific for heart failure</li> <li>• The presence of more than one physical sign, such as an elevated jugular venous pressure, third heart sound and pulmonary crepitations, increases the likelihood of heart failure</li> </ul>	

# Brain natriuretic peptide

**Table 33** Brain natriuretic peptide

## Recommendation

Brain natriuretic peptide (BNP) assists in the diagnosis of patients presenting with symptoms of suspected heart failure

## Clinical Practice Points

- BNP-32 and NT-proBNP are both useful tests to aid clinical decision-making in patients presenting with symptoms of suspected heart failure. Suggested values for BNP are as follows:

	<b>Heart failure unlikely (Rule out test)</b>	<b>Heart failure likely (Rule in or confirm test)</b>
<b>BNP-32</b>	<100 pg/mL (approx 30 pmol/L)	>500 pg/mL (approx 145 pmol/L)
<b>NT-proBNP</b>	<300 pg/mL (approx 35 pmol/L)	>10,000 pg/mL (approx 1180 pmol/L) Recommended age-adjusted optimal cut points: Age <50 yrs: 450 pg/mL (~50 pmol/L) Age 50–75 yrs: 900 pg/mL (~100 pmol/L) Age >75 yrs: 800 pg/mL (~210 pmol/L)

- Intermediate or 'grey zone' values can be considered as those that fall above the cut points for ruling out heart failure but below those cut points for confirming heart failure (see above table)
- Age stratification for NT-proBNP reduces the likelihood of a grey zone value
- BNP levels may be elevated in the absence of heart failure due to atrial fibrillation, chronic obstructive pulmonary disease, acute coronary syndromes, pulmonary embolism, pulmonary hypertension or renal impairment
- BNP levels may be normal or only marginally elevated even if heart failure is present in patients who are obese, or who have recently been commenced on diuretic therapy (it is recommended that the blood test for NT-proBNP is done prior to commencing diuretics in a patient presenting with new symptoms), or in those who have had very sudden onset of ('flash') pulmonary oedema

continued over...

**Table 33** continued...

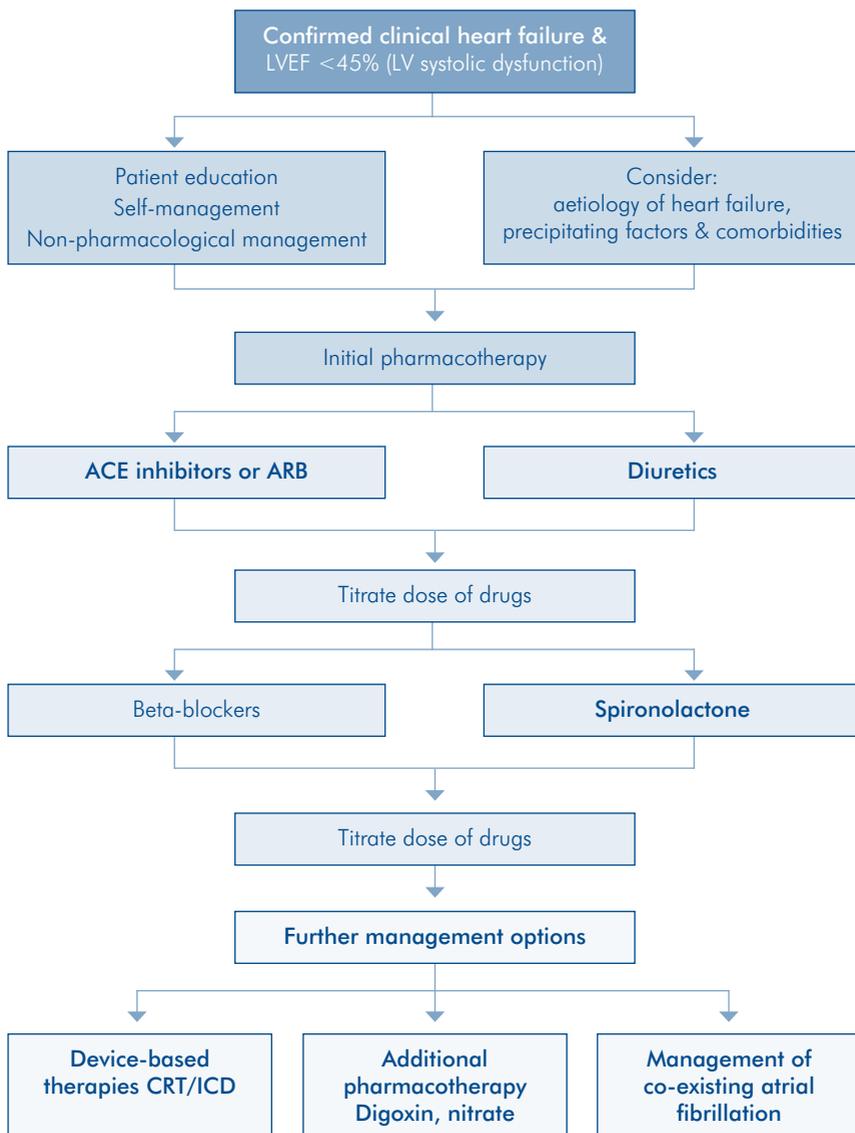
- Patients with grey zone NT-proBNP levels who present with symptoms and/or signs with good specificity for heart failure (such as paroxysmal nocturnal shortness of breath and/or an elevated jugular venous pressure) are more likely to have heart failure
- Patients in whom the diagnosis of heart failure is likely from clinical assessment and other tests, such as ECG or chest X-ray, do not require BNP testing for diagnosis
- While use of BNP can aid in the early assessment of patients with suspected heart failure, this biomarker does not replace the need for cardiac imaging in a patient with confirmed heart failure

## Management of heart failure with left ventricular systolic dysfunction

The algorithm (Figure 9) suggests a path for treatment of a patient diagnosed with heart failure and left ventricular systolic function (left ventricular ejection fraction <45%). It is recognised that individual drugs may be introduced at varying times depending on the individual patient. Titration of drug dosages to those utilised in the clinical trials should always be considered.

**Figure 9**

**Management of heart failure with left ventricular systolic dysfunction**



LVEF left ventricular ejection fraction

ACE Inhibitor angiotensin converting enzyme inhibitor

ARB angiotensin 2 receptor blocker

CRT cardiac resynchronization therapy

ICD implantable cardioverter defibrillator

# 8 Prevention of infective endocarditis

The guideline *Prevention of Infective Endocarditis associated with Dental and Other Medical Interventions* was prepared by the Heart Foundation in 2008. The following content forms part of this guideline. A full copy of the guideline is available from [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz).

## Cardiac conditions for prophylaxis

Table 34	Cardiac conditions for which endocarditis prophylaxis is recommended
<ul style="list-style-type: none"> <li>• Prosthetic heart valves (bio- or mechanical)</li> <li>• Rheumatic valvular heart disease</li> <li>• Previous endocarditis</li> <li>• Unrepaired cyanotic congenital heart disease (includes palliative shunts and conduits)</li> <li>• Surgical or catheter repair of congenital heart disease within 6 months of repair procedure</li> </ul>	

## Dental procedures for prophylaxis

**Table 35** Dental procedures (plus tonsillectomy/adenoidectomy) for which endocarditis prophylaxis is recommended

All dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa\*

- \* The following procedures and events do NOT need prophylaxis:
- routine anaesthetic injections through non-infected tissue
  - taking dental radiographs
  - placement of removable prosthodontic or orthodontic appliances
  - adjustment of orthodontic appliances
  - placement of orthodontic brackets
  - shedding of deciduous teeth
  - bleeding from trauma to the lips or oral mucosa.

## Non-dental procedures NOT requiring prophylaxis

Endocarditis prophylaxis is no longer recommended for non-dental procedures including respiratory, gastrointestinal and genitourinary procedures (see Table 36), unless the procedure is at a site of established infection.

**Table 36** Non-dental procedures for which endocarditis prophylaxis is NOT recommended\*†

The following procedures do NOT need endocarditis prophylaxis:

- surgery involving respiratory mucosa (other than tonsillectomy/adenoidectomy)
- bronchoscopy
- oesophageal, gastrointestinal or hepatobiliary procedures (including oesophageal stricture dilatation, ERCP)
- gastrointestinal endoscopy
- genitourinary or gynaecologic procedures (including TURP, cystoscopy, urethral dilatation, lithotripsy and hysterectomy)
- vaginal or caesarean delivery
- cardiac procedures (including percutaneous catheterisation)

\* Endocarditis prophylaxis may be recommended if the procedure is at a site of established infection.

† Antibiotic prophylaxis to prevent non-endocarditis infection after these procedures may be indicated.

## Antibiotic regimen for dental procedures

**Table 37** Antibiotic regimen for dental procedures  
(plus tonsillectomy/adenoidectomy)

Amoxicillin 2 g (child: 50 mg/kg up to 2 g), administered:

- orally, 1 hour before the procedure, or
- IV, just before the procedure, or
- IM, 30 minutes before the procedure

*Administer parenterally if unable to take medication orally; administer IV if IV access is readily available.*

For penicillin allergy or if a penicillin or cephalosporin-group antibiotic taken more than once in the previous month (including those on long-term penicillin prophylaxis for rheumatic fever):

Clindamycin 600 mg (child: 15 mg/kg up to 600 mg), administered:

- orally, 1 hour before the procedure, or
- IV, over at least 20 minutes, just before the procedure, or
- IM, 30 minutes before the procedure

or

Clarithromycin 500 mg (child: 15 mg/kg up to 500 mg) orally, 1 hour before the procedure

Clindamycin is not available in syrup form in New Zealand. Beware potential interactions between clarithromycin and other medications

If the antibiotic is inadvertently not administered before the procedure, it may be administered up to 2 hours after the procedure

## Antibiotic regimen for surgery with established infection

**Table 38**

### **Antibiotic regimen for surgery/procedures at sites of established infection**

Treat promptly with antibiotics expected to cover the majority of causative organisms. For the purposes of endocarditis prevention, this should include:

- dental or upper respiratory tract infections – amoxicillin (clindamycin or clarithromycin if penicillin allergy)
- gastrointestinal, hepatobiliary, genitourinary or obstetric/gynaecological infections – amoxicillin (vancomycin if penicillin allergy)
- skin, skin structure or musculoskeletal infections – flucloxacillin (a cephalosporin if mild penicillin allergy; clindamycin if severe penicillin allergy or suspect MRSA)

## 9 Rheumatic fever and sore throat management

This chapter on rheumatic fever includes guidance for primary care practitioners when a patient presents with a sore throat.

The guidance is drawn from *New Zealand Guidelines for Rheumatic Fever* produced by the Heart Foundation in 2006, available from [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz) and a systematic evidence review of key areas in the management of Group A streptococcus (GAS) throat infection for the prevention of acute rheumatic fever (ARF) undertaken by NZGG in 2011, available from [www.nzgg.org.nz](http://www.nzgg.org.nz).

### Key findings of the review

The NZGG review had two key findings:

- there is **no** evidence to support the current practice of delaying treatment by up to 9 days and there is no evidence to support any other recommendation about the timing of treatment. Therefore, antibiotics should be initiated as soon as possible
- there is reliable evidence about the efficacy of rapid antigen diagnostic tests, which give a result much faster than swabbing and testing.

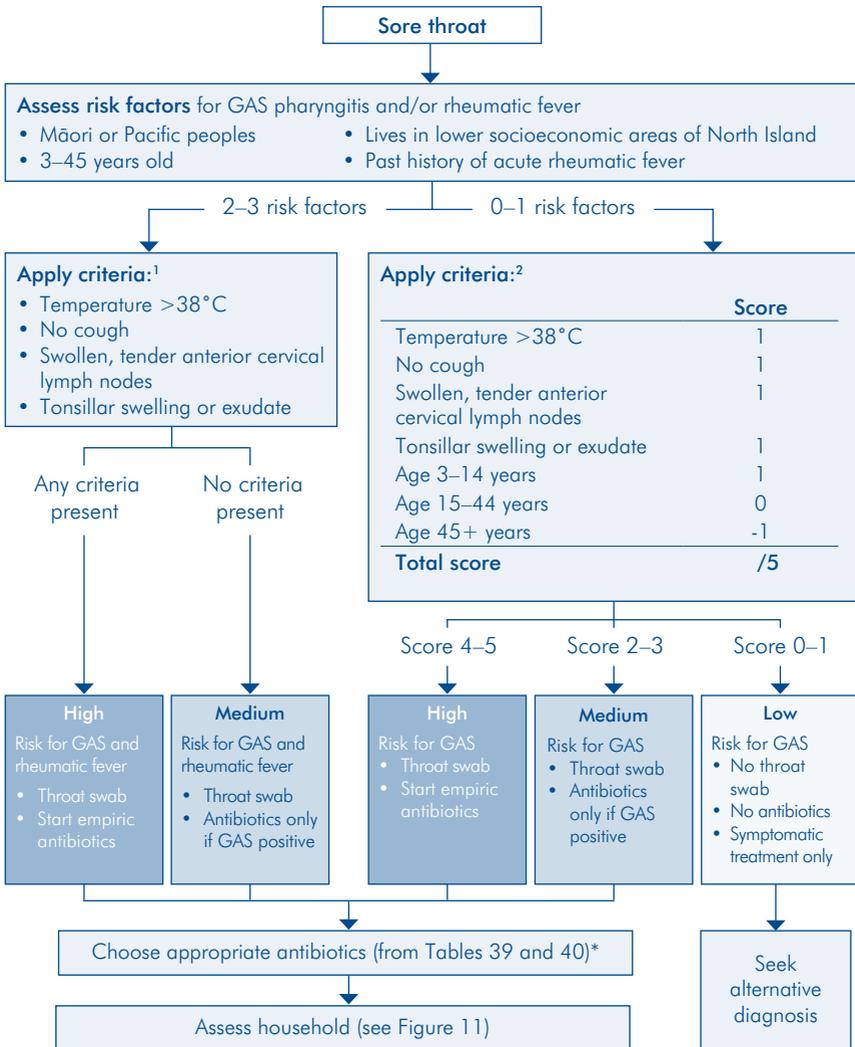
The Ministry of Health is considering the implications of the second finding and has decided that rapid antigen diagnostic tests must be first piloted in the settings where they are likely to be used (general practices in particular), and their cost implications analysed and addressed before the publication of a new algorithm (as developed by NZGG) for sore throat management in primary care that accounts for rapid antigen diagnostic tests. This could not be done in time for the publication of this 2012 edition of the Primary Care Handbook. Appropriate further guidance will therefore follow once these issues are addressed.

	Antibiotics should be initiated as soon as possible as there is no evidence to support current practice of delaying treatment by up to 9 days and there is no evidence to support any other recommendation about the timing of treatment	NZGG, 2011
	Children at <b>high</b> risk of developing rheumatic fever should continue to receive empiric (immediate) antibiotic treatment, and the presence of GAS should continue to be confirmed by laboratory culture	NZGG, 2011, NHF 2006
	Treatment of GAS pharyngitis with appropriate antibiotics reduces the occurrence of ARF	NHF 2006
	A diagnosis of ARF varies according to location and ethnicity, with high incidence rates in the northern half of the North Island and in Māori and Pacific peoples	NHF 2006
	Jones' (1992) diagnostic criteria (modified for the New Zealand guidelines) should be used to determine definite, probable and possible ARF (see Table 41). The criteria should not be rigidly adhered to when ARF is the most likely diagnosis	NHF 2006
	Priorities for managing ARF are: admission to hospital, confirmation of diagnosis, treatment (antibiotics and management of arthritis/arthritis, fever, carditis/heart failure and chorea), clinical follow-up and commencement of long-term preventive measures	NHF 2006

## Sore throat management

- Acute rheumatic fever is a sequela associated with a GAS infection (usually of the throat) (NZGG, 2011)
- The exact rate of GAS throat infection in the New Zealand population is unknown. Internationally, between 3% and 36% of sore throats are thought to be due to a GAS infection (NZGG, 2011)
- GAS sore throats are considered to be the only clinically significant bacterial throat infection in the New Zealand population (NHF, 2006)
- Children with GAS pharyngitis should be kept home from school or daycare for 24 hours until treatment is established (NHF, 2006)
- Antibiotic treatment is highly effective for GAS infection and significantly reduces the chance of ARF developing (NZGG, 2011)
- Antibiotics should be initiated as soon as possible (NZGG, 2011)
- Children at **high** risk of developing rheumatic fever should continue to receive empiric (immediate) antibiotic treatment, and the presence of GAS should continue to be confirmed by laboratory culture (NZGG, 2011, NHF, 2006)
- Antibiotic treatment varies according to whether it is the patient's first or third or more episode of GAS pharyngitis within a three-month period (see Tables 39 and 40) (NHF, 2006)
- Once daily amoxicillin is the first choice for antibiotic treatment for a GAS throat infection. Studies comparing amoxicillin with penicillin V report comparable outcomes. Amoxicillin is likely to achieve better compliance because of its daily dosing and ability to be taken with food compared to penicillin V's more frequent dosing and the requirement to take it on an empty stomach (NZGG, 2011). See Table 39 for details of dosage

**Figure 10 Sore throat management decision guide**



\* If patient is on benzathine penicillin IM prophylaxis for ARF, and is GAS positive on throat swab, treat in the following way:

- if GAS positive in the first two weeks after IM penicillin injection, treat with a 10-day course of erythromycin (see Table 39)
- if GAS positive in the 3rd and 4th weeks after IM penicillin injection, treat with a 10-day course of oral penicillin (see Table 39).

**Sources:**

1. Centor RM, et al. Med Decis Making. 1981;1:239–246.  
 2. McIsaac WJ, et al. JAMA. 2004;291(13):1587–1595. Adapted with permission. Copyright © 2004 American Medical Association. All rights reserved.



**Table 39** Routine antibiotics

Standard treatment of GAS positive pharyngitis for patient's first or second case of GAS pharyngitis in a three-month period

Antibiotic*	Route	Dose	Duration
<b>Amoxicillin</b> Give as first choice Can be given with food	PO	Weight <30 kg: 750 mg once daily Weight >30 kg: 1500 mg once daily	10 days
<b>Penicillin V</b> Give on empty stomach	PO	Children: 20 mg/kg/day in 2–3 divided doses Maximum 500 mg 3 times daily (250 mg 3 times daily for smaller children) Adults: 500 mg twice daily	10 days
<b>Benzathine penicillin G (BPG)</b> Can be given if compliance with 10 day regime likely to be a problem	IM	Children <20 kg: 600,000 U once only Adults and children >20 kg: 1,200,000 U once only	Single dose
<b>Erythromycin ethyl succinate (EES)</b>	PO	Children: 40 mg/kg/day in 2–4 divided doses Maximum 1 g/day Adults: 400 mg twice daily	10 days

PO Orally; IM Intramuscular

<sup>2012</sup> \* Order of antibiotics reflects new guidance that once daily amoxicillin is now the first choice for antibiotic treatment.

**Table 40** Recurrent antibiotics

Treatment of persons with multiple, recurrent, episodes of GAS pharyngitis proven by culture or rapid antigen testing. Use if this is the patient's third, or more, case of GAS pharyngitis in a three-month period

Antibiotic	Regimen	Duration
<b>Oral</b>		
Clindamycin	Children: 20–30 mg/kg/day in 3 divided doses	10 days
	Adults: 600 mg/day in 2–4 divided doses	10 days
Amoxicillin; clavulanic acid	Children: 40 mg/kg/day in 3 divided doses*	10 days
	Adults: 500 mg twice daily	10 days
<b>Parenteral with or without oral</b>		
Benzathine penicillin G	For IM dosages, see Table 39 <sup>†</sup>	1 dose
Benzathine penicillin G with rifampicin	For IM dosages, see Table 39 <sup>†</sup>	4 days
	Rifampicin: 20 mg/kg/day orally in 2 divided doses	
<p>* Maximum dose, 750mg of amoxicillin per day.</p> <p>† Addition of rifampicin to benzathine penicillin G may be beneficial for eradication of streptococci from the pharynx. The addition of rifampicin (20 mg/kg/day, once daily) during the final four days of a ten day course of oral penicillin V may achieve high rates of eradication. The maximum daily dose of rifampicin is 600 mg; rifampicin is relatively contraindicated for pregnant women.</p>		
<p><b>Source:</b> Bisno A, et al. Clin Infect Dis. 2002; 35:113–125. Adapted with permission. © 2002 by the Infectious Disease Society of America. All rights reserved.</p>		

## Diagnosis of acute rheumatic fever

Diagnose ARF using diagnostic criteria (see Table 41 and Figure 10). Hospital referral where expertise is available for accurate diagnosis particularly echocardiography, is usual.

All patients with suspected or definite ARF should undergo echocardiography to identify evidence of carditis (see [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz) – *Algorithm 2: Guide for the use of echocardiography in acute rheumatic fever*).

**Table 41** New Zealand guidelines for the diagnosis of acute rheumatic fever

	Diagnostic requirements	Category
Initial episode of ARF	2 major <b>or</b> 1 major and 2 minor criteria <b>plus</b> evidence of a preceding GAS infection	Definite ARF
Initial episode of ARF	1 major and 2 minor with the inclusion of evidence of a preceding GAS infection as a minor criteria (Jones, 1956) <sup>1</sup>	Probable ARF
Initial episode of ARF	Strong clinical suspicion of ARF, but insufficient signs and symptoms to fulfil diagnosis of definite or probable ARF	Possible ARF
Recurrent attack of ARF in a patient with known past ARF or established RHD	2 major <b>or</b> 1 major and 2 minor <b>or</b> several minor <b>plus</b> evidence of a preceding GAS infection (Jones, 1992) <sup>2</sup>	
Major criteria modified from Jones, 1992. (See guideline for further information on major criteria)	Carditis (including evidence of subclinical rheumatic valve disease on echocardiogram)* Polyarthritist (or aseptic monoarthritis with history of NSAID use) Chorea (can be stand-alone for ARF diagnosis) Erythema marginatum Subcutaneous nodules	
Minor criteria (See guideline for further information on minor criteria)	Fever Raised ESR or CRP Polyarthralgiat Prolonged P–R interval on ECG*	

**CRP** C-reactive protein; **ECG** electrocardiogram; **ESR** erythrocyte sedimentation rate

\* When carditis is present as a major manifestation (clinical and/or echocardiographic), prolonged P–R interval cannot be considered an additional minor manifestation.

† If polyarthritist is present as a major manifestation, polyarthralgia cannot be considered an additional minor manifestation.

**References:**

1. Circulation. 1956;13:617–620.
2. JAMA. 1992;268:2069–2073.

## Investigations in suspected ARF

### Recommended for all cases

- White blood cell count
- ESR – erythrocyte sedimentation rate (repeat weekly once diagnosis confirmed)
- C-reactive protein
- Blood cultures if febrile
- ECG (repeat as necessary if conduction abnormality more than first degree)
- Chest x-ray if clinical or echocardiographic evidence of carditis
- Echocardiogram (repeat as necessary in 2–4 weeks if equivocal, or if serious carditis) (see [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz) – *Algorithm 2: Guide for the use of Echocardiography in Acute Rheumatic Fever*)
- Throat swab (preferably before giving antibiotics) – culture for GAS
- Anti-streptococcal serology: both ASO and anti-DNase B titres, if available (repeat 10–14 days later if first test not confirmatory)

### Tests for alternative diagnoses, depending on clinical features

- Repeated blood cultures if possible endocarditis or septic arthritis
- Joint aspirate (microscopy and culture) for possible septic arthritis
- Joint x-ray
- Copper, caeruloplasmin, anti-nuclear antibody, drug screen, and consider CT/MRI head for choreiform movements
- Serology and auto-immune markers for auto-immune or reactive arthritis (including ANA – anti nuclear antibody)

## Management: patients not fulfilling diagnostic criteria for acute rheumatic fever

Patients who do not fulfil the diagnostic criteria (see Table 41), but in whom the clinician still suspects ARF, should be maintained on oral penicillin and reviewed in two to four weeks with a repeat echocardiogram to detect any new lesions. If there is evidence of rheumatic valve disease clinically or on echocardiogram, the diagnosis is confirmed, and long-term secondary prophylaxis can be commenced. If there is no evidence of carditis and no alternative diagnosis has been found then ARF is possible. Those with epidemiological risk factors (Māori, Pacific, low socioeconomic status) should be commenced on secondary prophylaxis with due consideration of an alternative diagnosis (such as rheumatological), and the need for ongoing review.

## Management of acute rheumatic fever

Priorities for managing ARF are: admission to hospital, confirmation of diagnosis, treatment (antibiotics and management of arthritis/arthralgia, fever, carditis/heart failure and chorea), clinical follow-up and commencement of long-term preventive measures.

## Secondary prevention

For guidance on the appropriate duration of secondary prophylaxis in ARF and appropriate antibiotic regimens see [www.heartfoundation.org.nz](http://www.heartfoundation.org.nz) – *Algorithm 3: Guide for the Duration of Secondary Prophylaxis in Acute Rheumatic Fever*. It is important that antibiotic prescribing is of appropriate length to prevent recurrence.

It is recommended that cases with established valvular disease have regular dental care and follow the guidelines for endocarditis prophylaxis.

# Appendices

- A Background: process for development of 2009 edition
- B Genetic lipid abnormalities
- C Recommended method of blood pressure measurement
- D Metabolic equivalents (METs) for selected activities
- E The New Zealand cardioprotective dietary pattern
- F Interpreting fasting plasma glucose results
- G Conversion table for HbA1c formats
- H Addressing patient concerns about insulin therapy
- I Patient education checklist: initiation of insulin therapy
- J Monitoring blood glucose profile

## Appendix A

### Background: process for development of 2009 edition

**2012** For information about the 2012 update of the Handbook see section 'About the 2012 edition of the Handbook' – front of Handbook.

The following content details aspects of the development of the 2009 edition for sections unchanged in this 2012 edition of the Handbook.

#### 2009 content update

For the 2009 Handbook revision, a selective and focused approach was taken to meet the immediate needs of the sector with the understanding that comprehensive revision of the full NZGG reference guidelines on cardiovascular risk and type 2 diabetes was needed and that this should follow.

The NZGG convened a Guideline Revision Team (GRT) with wide stakeholder representation (see next page for a list of the Team members). The most important topics requiring revision, particularly in relation to cardiovascular risk assessment and management and diabetes screening were identified. These topics were allocated to GRT members for literature review and presentation to the GRT for discussion and agreement on changes to be made for the 2009 edition.

For smoking cessation, a summary of the recently revised *Smoking Cessation Guidelines* (Ministry of Health 2007) replaced former content. Summaries of recommendations and algorithms related to rheumatic fever prevention, diagnosis and management, and infective endocarditis prevention were added. Summarised advice on cardiac rehabilitation remained unchanged from the 2005 edition of the Handbook.

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## Appendix B

### Genetic lipid abnormalities

<p><b>Genetic lipid disorders potentially putting people at a 5-year CVD risk &gt;20%. Assume high risk clinically in this group</b></p>	
<p><b>Familial hypercholesterolaemia (FH)</b></p>	<p><b>People presenting with cholesterol levels <math>\geq 8</math> mmol/L plus a family history of premature coronary heart disease, or tendon xanthelasma should be referred and offered family tracing</b></p> <p>People with FH usually have a family history of premature coronary heart disease compatible with autosomal dominant inheritance. Heterozygous FH has a prevalence in the general population of at least 1 in 500</p> <p>Family tracing of the siblings and children of people with FH is recommended</p> <p>Refer to a centre with expertise in management of lipid problems as mutation analysis allows more precise family tracing and screening. If referral is not possible these people should be discussed with an appropriate specialist</p>
<p><b>Familial defective ApoB (FDB)</b></p>	<p>These people should be managed and referred as for people with FH</p>
<p><b>Familial combined dyslipidaemia (FCH)</b></p>	<p>This is characterised by a strong family history of cardiovascular disease and a combined dyslipidaemia: high LDL-C, high triglycerides and usually a low HDL-C with small dense LDL-C particles</p>
<p><b>Genetic lipid disorders potentially putting people at a 5-year CVD risk &lt;20%. Calculate CVD risk in this group</b></p>	
<p><b>Low HDL-C syndromes</b></p>	<p>Low HDL-C confers a high risk for cardiovascular events. The causes of low HDL-C are multiple and these subjects are refractory to most drug interventions. Consider specialist review if HDL-C is <math>&lt; 0.7</math> mmol/L</p>
<p><b>High LP(a)</b></p>	<p>The genetic cause of high LP(a) is unknown. High values are refractory to most drug interventions</p>
<p><b>Isolated high triglycerides (<math>\geq 8</math> mmol/L)</b></p>	<p>The management of people with isolated high triglycerides should be discussed with the appropriate specialist</p>
<p><b>Broad beta disease</b></p>	<p>If the TC:triglyceride ratio approaches one, with both lipid fractions elevated, then further investigation is needed</p>

## Appendix C

### Recommended method of blood pressure measurement

1	Use a device with validated accuracy that is properly maintained and calibrated
2	Measure sitting blood pressure (BP) routinely. Measure sitting and standing blood pressure in the elderly or people with diabetes
3	Remove tight clothing, support arm with BP cuff at heart level, and ensure the hand is relaxed
4	Use cuff of appropriate size for arm circumference
5	Inflate the cuff until the radial pulse is no longer palpable
6	Lower mercury slowly, by not greater than 2 mm Hg per second
7	Read BP to the nearest 2 mm Hg
8	Measure diastolic BP as disappearance of sounds (phase 5)
9	Two measurements at a single visit are sufficient for calculating cardiovascular risk
10	At least two measurements should be made at each of three visits to determine BP thresholds if considering treatment – some of these can be recorded at nurse consultations using this measurement technique
11	Possible indications for 'home' or ambulatory BP monitoring include the diagnosis of 'white coat hypertension', suspected hypotension, excessive BP variability and resistance to drug therapy
12	Home-based measurement may be lower than office measurement and therefore treatment decisions should be based predominantly on office measurement

## Appendix D

### Metabolic equivalents (METs) for selected activities\*

Activity		METs (min)	METs (max)
<b>METs for leisure activities</b>			
Aerobics		6	9
Cycling	8 km per hour	2	3
	16 km per hour	5	6
	21 km per hour	8	9
Music	Playing an instrument	2.5	4
Dancing	Ballroom	4	5
Gardening	Mowing lawn (pushing)	3	6
	Weeding/cultivating	4	5
Running	General light jogging	6	8
	Training 10 km per hour	9	11
Skipping	<80/min	8	10
Swimming	Breast stroke	8	9
	Freestyle	9	10
Tennis		4	9
Walking	1–3 km per hour	1	3
	3–6 km per hour	3	6
<b>METs for activities of daily living</b>			
Carrying heavy groceries		5	7
Cleaning windows		3	4
Cooking		2	3
General housework		3	4
Grocery shopping		2	4
Loading/unloading washing machine		4	5
Mowing by hand		5	7
Painting/decorating		4	5
Sexual intercourse		3	5
Showering		3	4
Vacuuming		3	3.5
Walking up stairs		4	7
Washing a car		6	7
Washing dishes		2	3
* 1 MET equals oxygen consumption at rest which is about 3.5 ml/kg of body weight per minute. An individual exercising at 2 METs is consuming oxygen at twice the resting rate.			

## Appendix E

### The New Zealand cardioprotective dietary pattern

Food	Healthy servings (per day)	Serving size examples	Notes
<b>Vegetables</b>	At least 3–4 servings. Include at every meal	<p>½ cup cooked vegetables</p> <p>1 cup raw green vegetable or salad</p> <p>1 tomato or carrot</p>	Choose coloured varieties daily, especially the green, orange and red vegetables. Also includes cauliflower, onions, mushrooms, turnips
<b>Fruit</b>	At least 3–4 servings	<p>1 medium apple, pear, orange, small banana</p> <p>½ cup stewed, frozen, canned fruit (natural or 'lite')</p> <p>2–3 small apricots or plums</p> <p>10–15 grapes, cherries, strawberries</p> <p>1 cup other berries</p> <p>3 prunes, dates, figs or 1 tbsp raisins, sultanas</p> <p>6–8 halves of dried apricots</p> <p>180 ml 100% fruit juice</p>	No more than one serving of fruit juice per day
<b>Breads, cereals, grains</b>	At least 6 servings	<p>1 medium slice of whole grain bread or ½ bread roll</p> <p>30 g of other breads such as pita, naan, corn tortilla, wraps</p> <p>½ cup bran cereal or ⅔ cup wheat cereal or ½ cup cooked porridge or ⅓ cup muesli or 3 crispbreads</p> <p>½ cup cooked pasta or ⅓ cup cooked rice</p>	<p>Choose more or less depending on body weight and level of physical activity Include at every meal</p> <p>Choose a variety of grain products with at least half as whole grain products</p>
<b>Starchy vegetables</b>		<p>1 small potato ½ kumara</p> <p>⅓ cup yams ½ cup corn</p> <p>½ parsnip 1 small round of taro</p>	These replace bread/ grain products. Limit for weight and diabetes control

Food	Healthy servings (per day)	Serving size examples	Notes
<b>Low-fat or fat-free milk products</b>	2–3 servings or replace with soy products	1 glass trim or low-fat milk (250 ml) 1 pottle low-fat yoghurt ⅓ cup cottage cheese ½ cup low-fat cottage cheese ¼ cup quark or ricotta 2 tbsp parmesan or 3 tbsp grated cheddar cheese 2 cm cube cheddar cheese 3 cm cube soft cheese	Use 0 to 0.5% fat milk and <1% fat yoghurt Hard cheese and semi-soft cheeses can be included up to 4 times weekly in very small amounts Camembert, brie, edam, feta, mozzarella
<b>Fish, seafood</b>	1–2 servings weekly	2 small, 1 large fillet of cooked fish ½ cup tuna or 1 cup mussels ⅓ cup salmon or ½ can sardines	If eating fish, choose some oily fish: tuna, kahawai, trevally, kingfish, warehou, dory, salmon, sardines, eel, squid, mussels or oysters
<b>Peas, beans, soy products (legumes)</b>	4–5 servings weekly	1 cup cooked dried beans, chickpeas, lentils, dahl ½ cup tofu or tempeh 1 glass fortified soy milk (250 ml)	
<b>Skinned chicken or very lean meats</b>	Limit to 1–1½ servings	2 slices trimmed meat/chicken (100–120 g) ½ cup lean mince or casserole (125 g) 1 small lean steak (100 g) 1 small chicken breast (120 g) 2 small drumsticks or 1 leg, skinned	Use alternatives to meat several times a week
<b>Eggs</b>	3 eggs weekly	1 egg	

continued over...

Food	Healthy servings (per day)	Serving size examples	Notes
<b>Liquid oils, unsaturated margarines and spreads or avocado</b>	3 or more servings	1 tsp soft table margarine or oil 2 tsp light margarine (50–60% fat) 2 tsp mayonnaise or vinaigrette (50–60% fat) 3 tbsp reduced-fat mayonnaise or dressing (10% fat or less) 1 tbsp avocado	Choose more or less depending on body weight and level of physical activity. Choose products made from sunflower, soya bean, olive, canola, linseed, safflower or nuts and seeds, other than coconut.
<b>Nuts, seeds</b>	Eat regularly up to 30 g/day	1 dsp nuts or pumpkin seeds 1 dsp peanut butter 1 tbsp sunflower or sesame seeds	For weight control one serving of nuts replaces other oils and spreads
<b>Confectionery and added sugar</b>	Up to 1* servings or up to 3 servings	1 tbsp sugar, jam, syrup or honey 2 tbsp all-fruit jam spreads Small pottle reduced-fat ice-cream or frozen yoghurt 2 fruit slice biscuits	Best incorporated as part of the meal or snack only if diabetes is well controlled. Artificial sweeteners may be used for additional sweetness as a replacement for sugar
<b>Minimise added salt</b>	Limit high salt seasonings to 1/day	1 tsp seasoning paste 1/6 stock cube or 1/8 tsp stock powder 1/3 tsp gravy mix or 1 tbsp liquid seasoning	Use minimal salt in cooking Do not add salt to meals
<b>Limit high salt foods</b>	Limit these high salt foods to less than 4 servings/day	30 g lean ham/pastrami 1 tbsp pickles or 1 tsp marmite/vegemite 1 tsp soy sauce 20 to 30 g cheese 1/2 cup canned/packet soup 50 g canned or smoked salmon/tuna 30 g other smoked fish/sardines	Choose breads and cereals with less than 450 mg/100 g sodium and spreads with less than 400 mg/100 g sodium Choose low or reduced salt/sodium canned foods, soups, sauces seasonings, crispbreads, relishes and meals Check labels of cured, corned, pickled, smoked, marinated and canned foods

Food	Healthy servings (per day)	Serving size examples	Notes
<b>Alcoholic drinks</b>	Limit to <3 drinks for men and <2 for women	1 (300 ml) glass ordinary strength beer 1 (60 ml) glass fortified wine (sherry, port) 1 (30 ml) pub measure spirits (whisky, gin) 1 (100 ml) glass of table wine	
<b>Non-alcoholic drinks</b>	6–8 drinks/day	1 glass water (250 ml) 1 cup 'diet' soft drink (180 ml) 1 glass trim or low-fat milk (250 ml) 1 cup tea, coffee or cocoa 1 cup vegetable juices (180 ml)	Drink plenty of water every day Limit the consumption of fruit juice, cordial and fizzy drinks because of their high sugar content
<p>* Up to 1 serving per day for weight control or for people with high triglycerides or diabetes as part of a meal or snack. Up to 3 per day for people in the healthy weight range who are active with normal triglycerides and no diabetes.</p>			

## Appendix F

### Interpreting fasting plasma glucose results

The NZSSD position statement on the diagnosis of diabetes sets out that where glucose-based testing is used, the diagnostic criteria remain unchanged. This content on the diagnosis of diabetes or prediabetes using plasma glucose testing is adapted from the 2009 edition of the *New Zealand Cardiovascular Guidelines Handbook*.

Result	Action	Why
<b>7.0 mmol/L or more</b>	Repeat a fasting plasma glucose*	Two results above this level, on separate occasions,* are diagnostic of diabetes and <b>do not</b> require an OGTT†
<b>6.1–6.9 mmol/L</b> Request an OGTT†	Request an OGTT†	A 2-hr post glucose load of $\geq 11.1$ mmol/L is confirmation of diabetes A 2-hr post glucose load of $\geq 7.8$ and $< 11.1$ mmol/L is confirmation of prediabetes
<b>5.5–6.0 mmol/L</b>	Request an OGTT† in high-risk groups‡	The fasting plasma glucose result may be normal but some patients will show diabetes or prediabetes on the OGTT† A 2-hr post glucose load of $\geq 11.1$ mmol/L is confirmation of diabetes A 2-hr post glucose load of $\geq 7.8$ and $< 11.1$ mmol/L is confirmation of prediabetes
<b>5.4 mmol/L or less</b>	Retest at next cardiovascular risk assessment interval	This result is normal

\* The diagnosis of diabetes should be confirmed by repeating a fasting plasma glucose on another day unless there is unequivocal hyperglycaemia with acute metabolic decompensation or obvious symptoms of thirst or polyuria.

† **OGTT** Oral glucose tolerance test.

‡ Non-European ethnicity, first-degree relative with diabetes, past history of gestational diabetes.

## Appendix G

### Conversion table for HbA1c formats

Since October 2011 New Zealand laboratories report HbA1c values only in IFCC-aligned format (molar units measured in mmol/mol), not in DCCT-aligned format (measured in percentage).

The conversion formulae are:

IFCC-aligned HbA1c value = (10.93 x DCCT-aligned value) – 23.5 mmol/mol

DCCT-aligned HbA1c value = (0.0915 x IFCC-aligned value) + 2.15 %

Health practitioners are able to calculate HbA1c values at [www.diabetes.org.uk/hba1c](http://www.diabetes.org.uk/hba1c)

IFCC-aligned HbA1c (mmol/mol)	DCCT-aligned HbA1c (%)	IFCC-aligned HbA1c (mmol/mol)	DCCT-aligned HbA1c (%)
20	4.0	38	5.6
21	4.1	39	5.7
22	4.2	40	5.8
23	4.3	41	5.9
25	4.4	42	6.0
26	4.5	43	6.1
27	4.6	44	6.2
28	4.7	45	6.3
29	4.8	46	6.4
30	4.9	48	6.5
31	5.0	49	6.6
32	5.1	50	6.7
33	5.2	51	6.8
34	5.3	52	6.9
36	5.4	53	7.0
37	5.5	54	7.1

continued over...

IFCC-aligned HbA1c (mmol/mol)	DCCT-aligned HbA1c (%)
55	7.2
56	7.3
57	7.4
58	7.5
60	7.6
61	7.7
62	7.8
63	7.9
64	8.0
65	8.1
66	8.2
67	8.3
68	8.4
69	8.5
70	8.6
72	8.7
73	8.8
74	8.9
75	9.0
76	9.1
77	9.2
78	9.3
79	9.4
80	9.5
81	9.6
83	9.7
84	9.8
85	9.9
86	10.0
87	10.1
88	10.2

IFCC-aligned HbA1c (mmol/mol)	DCCT-aligned HbA1c (%)
89	10.3
90	10.4
91	10.5
92	10.6
93	10.7
95	10.8
96	10.9
97	11.0
98	11.1
99	11.2
100	11.3
101	11.4
102	11.5
103	11.6
104	11.7
105	11.8
107	11.9
108	12.0
109	12.1
110	12.2
111	12.3
112	12.4
113	12.5
114	12.6
115	12.7
116	12.8
117	12.9
119	13.0
120	13.1
121	13.2
122	13.3

IFCC-aligned HbA1c (mmol/mol)	DCCT-aligned HbA1c (%)
123	13.4
124	13.5
125	13.6
126	13.7
127	13.8
128	13.9
130	14.0
131	14.1
132	14.2
133	14.3
134	14.4
135	14.5
136	14.6
137	14.7
138	14.8
139	14.9
140	15.0
142	15.1
143	15.2

IFCC-aligned HbA1c (mmol/mol)	DCCT-aligned HbA1c (%)
144	15.3
145	15.4
146	15.5
147	15.6
148	15.7
149	15.8
150	15.9
151	16.0

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**Source:** Adapted from SIGN guideline 116 Management of Diabetes (2010)  
[www.sign.ac.uk/guidelines/fulltext/116/index.html](http://www.sign.ac.uk/guidelines/fulltext/116/index.html)

**IFCC** International Federation of Clinical Chemistry and Laboratory Medicine  
**DCCT** Diabetes Control and Complication Trial

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A patient info sheet can also be downloaded at  
[www.nzssd.org.nz/HbA1c/MoH%20Diabetes%20Flyer.pdf](http://www.nzssd.org.nz/HbA1c/MoH%20Diabetes%20Flyer.pdf)

## Appendix H

### Addressing patient concerns about insulin therapy

This content has been prepared by the NZGG Diabetes Advisory Group to assist primary care practitioners when discussing initiation of insulin therapy with patients. It draws on the experience of the Advisory Group.

#### Common misconceptions about insulin therapy and discussion points

It is important to enquire about and address an individual's concerns about insulin therapy.

##### Common misconceptions

- My diabetes has become worse, or is a more serious disease
- Insulin therapy is a sign of my personal failure to manage the condition
- Insulin therapy will adversely impact on my lifestyle and will be inconvenient, resulting in loss of my personal freedom and independence
- Insulin therapy leads to complications
- I will be treated differently by family and friends

For Māori and Pacific people with diabetes, particularly older people, a common misconception is that starting insulin therapy means that they will die soon.

##### Suggested discussion points

- Type 2 diabetes is progressive and medication needs change over time
- Lifestyle management efforts are of value and should be ongoing. (Acknowledge the individual's lifestyle management efforts.)
- Insulin therapy is an additional tool to use alongside lifestyle management efforts
- Present the benefits of insulin: 'can improve health and make them feel better'
- Insulin therapy is the next logical step in treatment if oral therapy is insufficient
- Insulin therapy does not cause diabetes complications (if needed, it reduces the risk)
- Initially, only once or twice daily insulin will be required
- Insulin types and delivery devices have changed and improved in recent years
- Insulin devices allow very discreet use. (Show an insulin pen as an example.)
- Self-monitoring of blood glucose means that insulin therapy is now safer and more easily managed than in the past

continued over...

## Addressing patient concerns about insulin therapy continued...

### Common misconceptions about insulin therapy and discussion points continued...

#### Other suggestions

- Include the patient's partner or family/whānau in discussion/education
- Provide information about local patient support groups
- Show the patient a 6 mml insulin needle and let them try it out
- Suggest a trial period of insulin therapy for eg, 8 weeks. 'Try it for 8 weeks and see how you feel about it.'

**Source:** Reproduced from *Guidance on the Management of Type 2 Diabetes* (2011).

## Appendix I

### Patient education checklist: initiation of insulin therapy

This content has been prepared by the NZGG Diabetes Advisory Group to assist primary care practitioners when initiating insulin therapy with a patient. It draws on the experience of the Advisory Group.

#### Education advice for your patients

- Self-monitoring of blood glucose
  - When to test, how to test, how to record in a log book style
  - Test if they have symptoms of hypoglycaemia
  - Increase frequency of testing if unwell
- Insulin regimen
  - Which insulin preparation
  - What the dose is, and when to administer it
  - How to use the insulin injection device
  - How to titrate the dose (if this is appropriate at this stage)
- How to administer insulin
- How to store the insulin and how to dispose of 'sharps'
- Dietary and lifestyle advice
  - Maintaining a healthy body weight by healthy eating and exercise
  - The risk of hypoglycaemia with excess alcohol consumption
- Managing hypoglycaemia
  - How to recognise the symptoms of hypoglycaemia
  - How to manage and prevent episodes of hypoglycaemia
- Driving: legal and practical issues
  - Ensure the patient understands their responsibility to maintain a reasonable level of glycaemic control while minimising their risk of hypoglycaemic episodes
  - If the patient is a vocational driver please refer for specialist advice
  - Refer to the NZ Transport Agency *Medical aspects of fitness to drive: A guide for medical practitioners July 2009*

continued over...

**Patient education checklist: initiation of insulin therapy** continued...**Education advice for your patients** continued...

- Provide Medic Alert bracelet information
- Provide contact and emergency telephone numbers
- Advise the patient where to get further self-help information (eg, Diabetes New Zealand website [www.diabetes.org.nz](http://www.diabetes.org.nz) or local diabetes societies)

**Provide your patient with appropriate written pamphlets**

Diabetes New Zealand has pamphlets on relevant topics eg, 'Diabetes and Insulin' and 'Diabetes and Healthy Food Choices'. These are available through Diabetes Supplies Ltd [www.diabetessupplies.co.nz](http://www.diabetessupplies.co.nz) or 0800 DIABETES

**Source:** Reproduced from *Guidance on the Management of Type 2 Diabetes* (2011).

## Appendix J

### Monitoring blood glucose profile

This content has been prepared by the NZGG Diabetes Advisory Group to assist primary care practitioners when initiating insulin therapy with a patient. It draws on the experience of the Advisory Group.

#### Monitoring blood glucose profile: use of a logbook

The use of a logbook to record the results of blood glucose testing assists initial and ongoing decision-making about insulin therapy and should be encouraged.

By varying the times of the day that the patient tests blood glucose, and recording these results in a logbook format, the patient's typical blood glucose profile across the course of a day will become apparent.

Patients can test more intensively when initiating insulin therapy and less intensively once insulin therapy is established.

In reviewing the logbook, focus on the trends on days that are representative of 'normal' for that person. Ignore outlier results or 'noise' (birthday parties, 'binges', 'not so good' days, sickness, excess alcohol).

**Table AJ.1** Example of a completed blood glucose profile logbook

	Before breakfast	After breakfast	Before lunch	After lunch	Before dinner	After dinner	Before bed
Mon	11.9		8.9				
Tues		10.8				7.6	
Wed	14.6				4.9		
Thur		11.9		6.3			
Fri	10.8		9.6		5.2		7.3
Sat							
Sun	13.6			7.2		7.5	

In this example, the person is only testing on average twice a day but is varying the times of the day and recording results in the correct columns. These results can thus be readily scanned to establish the person's usual daily pattern or profile.

The blood glucose levels shown in this example indicate the person would benefit from a once daily isophane insulin delivered in the evening to correct their morning high blood glucose level the next day.

**Source:** Reproduced from *Guidance on the Management of Type 2 Diabetes* (2011).

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# Abbreviations and acronyms

<b>A2</b>	Angiotensin II	<b>eGFR</b>	Estimated glomerular filtration rate
<b>ACE</b>	Angiotensin converting enzyme	<b>EPA</b>	Eicosapentaenoic acid
<b>ACR</b>	Albumin:creatinine ratio	<b>ERCP</b>	Endoscopic Retrograde holangiopancreatography
<b>AF</b>	Atrial fibrillation	<b>ESR</b>	Erythrocyte sedimentation rate
<b>AFL</b>	Atrial flutter	<b>FCH</b>	Familial combined dyslipidaemia
<b>ApoB</b>	Apolipoprotein B	<b>FDA</b>	Food and Drug Administration
<b>ARF</b>	Acute rheumatic fever	<b>FDB</b>	Familial defective ApoB
<b>BMI</b>	Body mass index	<b>FH</b>	Familial hypercholesterolaemia
<b>BNP</b>	Brain natriuretic peptide	<b>g</b>	Gram
<b>BP</b>	Blood pressure	<b>GAS</b>	Group A streptococcus
<b>bpm</b>	Beats per minute	<b>GCW</b>	Gross combined weight
<b>CABG</b>	Coronary artery bypass graft	<b>GFR</b>	Glomerular filtration rate
<b>CHF</b>	Chronic heart failure	<b>GI</b>	Glycaemic index
<b>CK</b>	Creatine kinase	<b>GLW</b>	Gross laden weight
<b>cm</b>	Centimetres	<b>h</b>	Hour
<b>COX2</b>	Cyclooxygenase-2 inhibitor	<b>HbA1c</b>	Haemoglobin type A1c
<b>CT</b>	Computed tomography	<b>HDL</b>	High density lipoprotein
<b>CVD</b>	Cardiovascular disease	<b>HDL-C</b>	High density lipoprotein cholesterol
<b>CYP3A4</b>	Cytochrome P450 3A4	<b>HRT</b>	Hormone replacement therapy
<b>DBP</b>	Diastolic blood pressure	<b>ICH</b>	Intracranial haemorrhage
<b>DC</b>	Direct current	<b>INR</b>	International normalised ratio
<b>DHA</b>	Docosahexaenoic acid		
<b>dL</b>	Decilitre		
<b>dsp</b>	Dessert spoon		
<b>ECG</b>	Electrocardiogram		
<b>ED</b>	Emergency department		

<b>IV</b>	Intravenous	<b>PVD</b>	Peripheral vascular disease
<b>J</b>	Joules	<b>SBP</b>	Systolic blood pressure
<b>kg</b>	Kilogram	<b>SMBG</b>	Self-monitoring blood glucose
<b>LDL</b>	Low density lipoprotein	<b>TC</b>	Total cholesterol
<b>LDL-C</b>	Low density lipoprotein cholesterol	<b>TIA</b>	Transient ischaemic attack
<b>LP(a)</b>	Lipoprotein (a)		
<b>LV</b>	Left ventricular		
<b>METs</b>	Metabolic equivalents		
<b>mg</b>	Milligram		
<b>MI</b>	Myocardial infarction		
<b>ml</b>	Millilitre		
<b>mm Hg</b>	Millimetres of mercury		
<b>mmol/L</b>	Millimole per litre		
<b>MRI</b>	Magnetic resonance imaging		
<b>MRSA</b>	Methicillin-Resistant Staphylococcus aureus		
<b>NASCET</b>	North American Symptomatic Carotid Endarterectomy Trial		
<b>NNT</b>	Number needed to treat		
<b>NRT</b>	Nicotine replacement therapy		
<b>NSAID</b>	Non-steroidal anti-inflammatory agents		
<b>NZSSD</b>	New Zealand Society for the Study of Diabetes		
<b>OGTT</b>	Oral glucose tolerance test		
<b>PCI</b>	Percutaneous coronary intervention		





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