2009 Edition

MINISTRY OF HEALTH
MANATŪ HAUORA

New Zealand Cardiovascular Guidelines Handbook

A summary resource for primary care practitioners

Cardiovascular risk assessment and diabetes screening

Cardiovascular risk factor management

Smoking cessation

Atrial fibrillation

Coronary heart disease

Stroke and transient ischaemic attack

Rheumatic fever

Prevention of infective endocarditis

Heart failure

Kidney Health NEW ZEALAND

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diabetes new zealand

Heart Foundation

New Zealand GUIDELINES GROUP
Te Rōpū Rarangi Tohutohu
Promoting Effective Health and Disability Services

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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.

Funding and development

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About the 2009 edition of the Handbook

The *New Zealand Cardiovascular Guidelines Handbook: A Summary Resource for Primary Care Practitioners* is an updated revision of the original Handbook published in 2005.

The original Handbook was a condensed version of advice derived from a number of separate full guidelines. All guidelines require reviewing and updating at intervals. In this Handbook, cardiovascular risk assessment and management and diabetes screening is updated pending the proposed revision of the source guidelines during the next two years. Smoking cessation advice is updated to reflect the revised 2007 guideline. Summarised advice on cardiac rehabilitation, stroke management and the management of atrial fibrillation and flutter is unchanged from the 2005 edition of the Handbook.

New information from the 2008 guideline on transient ischaemic attack is included (see section on Stroke and Transient Ischaemic Attack, page 53). Summary content from the rheumatic fever and infective endocarditis prevention guidelines is also included, as are management algorithms for heart failure. Guidelines on obesity were in development at the time of publication and summary content is therefore not available for this edition. Further information on the process for updating the Handbook is contained in Appendix F.

The Handbook provides summary guidance from the collection of guidelines 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 Assessment and Management of Cardiovascular Risk* (2003)*
- *Management of Type 2 Diabetes* (2003)*
- *Life after Stroke: New Zealand Guideline for Management of Stroke* (2003)*
- *Cardiac Rehabilitation* (2002)*
- *The Management of People with Atrial Fibrillation and Flutter* (2005)*
- *New Zealand Smoking Cessation Guidelines* (2007)†
- *New Zealand Guideline for Rheumatic Fever* (2007)‡
- *Prevention of Infective Endocarditis associated with Dental and other Medical Interventions* (2008)‡
- *A Guideline for the Management of Heart Failure* (2001 – in revision at time of publishing)‡
- *New Zealand Guideline for the Assessment and Management of People with recent Transient Ischaemic Attack (TIA)* (2008)§

* www.nzgg.org.nz

† www.moh.govt.nz

‡ www.nhf.org.nz

§ www.stroke.org.nz

Contents

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-modifiying and blood pressure lowering (BP lowering) medication, diabetes care, and medication after myocardial infarction (MI), stroke and other cardiovascular disease.

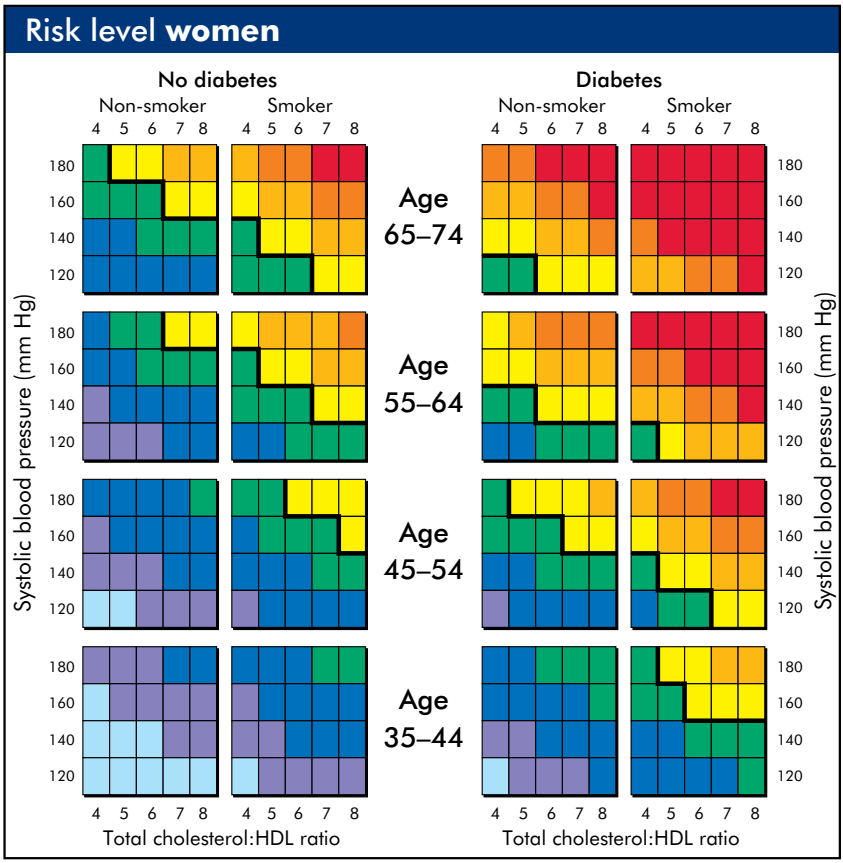
The overall goal is to reduce 5-year cardiovascular risk to **less than 15%**.

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

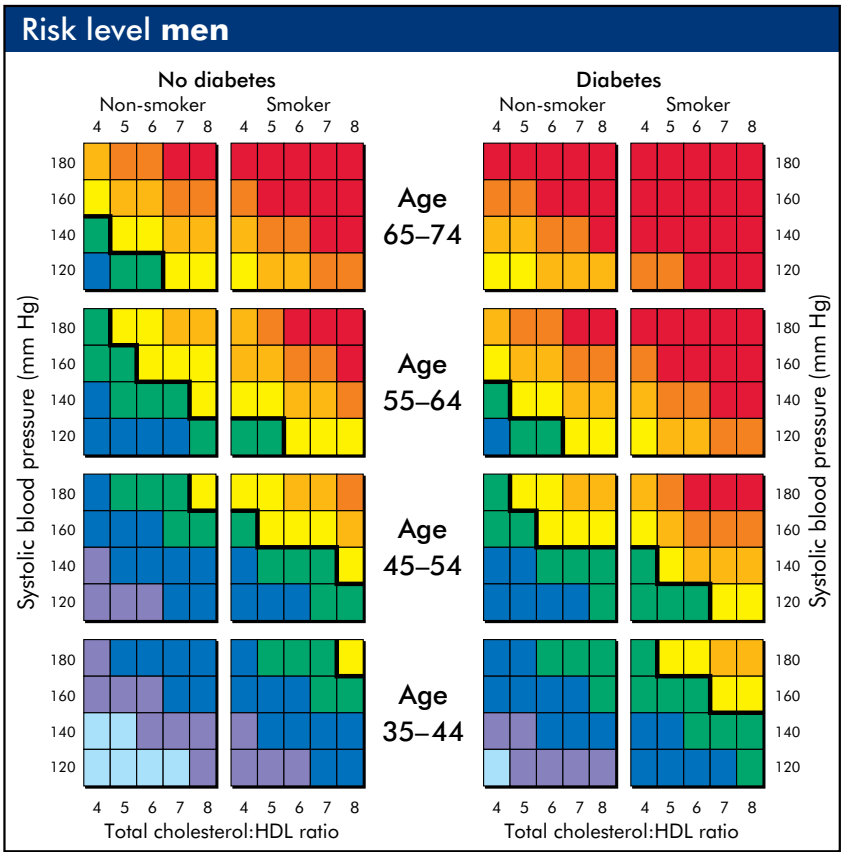


Risk level (for women and men)
5-year cardiovascular disease (CVD) risk (fatal and non-fatal)

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 and diabetes risk assessment

Group	Men	Women
Asymptomatic people without known risk factors	Age 45 years	Age 55 years
Māori, Pacific peoples or people from the Indian subcontinent*	Age 35 years	Age 45 years
People with other known cardiovascular risk factors or at high risk of developing diabetes **Family history risk factors** • Diabetes in first-degree relative (parent, brother or sister) • Premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother <55 years, mother or sister <65 years) **Personal history risk factors** • People who smoke (or who have quit only in the last 12 months) • Gestational diabetes, polycystic ovary syndrome • Prior blood pressure (BP) ≥160/95 mm Hg, prior TC:HDL ratio ≥7 • Known IGT (impaired glucose tolerance) or IFG (impaired fasting glucose) • BMI ≥30 or truncal obesity (waist circumference ≥100 cm in men or ≥90 cm in women) • eGFR† <60 ml/min/1.73 m^2	Age 35 years	Age 45 years
People with diabetes	Annually from the time of diagnosis	

* People from the Indian subcontinent = Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani, Tibetan.

† Estimated glomerular filtration rate (eGFR).

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%	**These people do not need their risk assessed using the New Zealand Cardiovascular Risk Charts:** • previous CVD event: angina, MI, percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), transient ischaemic attack (TIA), ischaemic stroke, peripheral vascular disease • some genetic lipid disorders: familial hypercholesterolaemia (FH), familial defective ApoB (FDB), familial combined dyslipidaemia (FCH) • diabetes with overt nephropathy (albumin:creatinine ratio ≥30 mg/mmol OR urinary albumin ≥200 mg/L) • diabetes with other renal disease causing renal impairment (eGFR ≤60 ml/min/1.73m^2)
Isolated elevated single risk factors: 5-year risk of >15%	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 ≥15% • TC ≥8 mmol/L • TC:HDL ratio ≥8 • BP consistently ≥170/100
People aged 35–74 years: calculate the 5-year CVD risk	Calculate 5-year risk using the New Zealand Cardiovascular Risk Charts or electronic decision-support tool based upon the Framingham risk equation (stand alone or incorporated into some practice software) **These groups should be moved up one risk category (5%):*** • family history of premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother <55 years, mother or sister <65 years) • Māori, Pacific peoples or people from the Indian subcontinent† • Diabetes with microalbuminuria OR for ≥10 years OR with HbA1c consistently ≥8%

continued over...

Table 2: continued...

Risk group	Estimating risk
People aged <35 years with known risk factors	**All calculations outside the age ranges of the Framingham equation are approximations, but can be useful** **Aged under 35 years:** 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 • Low HDL <0.7 mmol/L (because of the risk of a genetic lipid disorder – see Chapter 9 of the guideline: *The Assessment and Management of Cardiovascular Risk*) • Known familial dyslipidaemias or suspected genetic lipid disorders • Type 1 diabetes, type 2 diabetes with microalbuminuria or type 2 diabetes of long duration (≥10 years)
People aged ≥75 years	**Aged over 75 years:** calculate the risk as if they were 65–74 years 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 A clinical judgment should take into account: • likely benefits and risks of treatment • life expectancy and comorbidities • personal values
People with diabetes aged 20–34 years	The Framingham data is based on people ≥35 years. An alternative risk-calculation tool based on the UKPDS can be used for this group. See www.dtu.ox.ac.uk

* Make the 5% adjustment **once** only for people with >1 criterion.

† People from the Indian subcontinent = Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani, Tibetan.

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. A purpose-built electronic tool is being developed by the National Heart Foundation.

Table 3	What to measure and record for cardiovascular risk assessment and diabetes screening	
Everyone	History	• Age • Gender • Ethnicity • Smoking status (if stopped smoking for <12 months, assess as a smoker)
	Family history	• Premature coronary heart disease or ischaemic stroke in a first-degree relative (father or brother <55 years, mother or sister <65 years) • Type 2 diabetes • Genetic lipid disorder (see Appendix A)
	Past medical history	• Past history of CVD (MI, PCI, CABG, angina, ischaemic stroke, TIA, peripheral vascular disease [PVD]) • Genetic lipid disorder (FH, FDB, FCH: see Appendix A) • Renal impairment
	Measure	• Average of two sitting BP measurements • Pulse • BMI, waist circumference • Fasting lipid profile* • Fasting glucose*
Diabetes	History and examination	• Date of diagnosis • Type of diabetes (type 1, type 2, including type 2 on insulin, gestational diabetes) • HbA1c • Urine albumin: creatinine ratio (ACR) • eGFR† and history of renal disease
Atrial fibrillation (AF), confirmed on electro-cardiogram (ECG)	History and examination	• Echocardiogram (where possible) • Past history of stroke, TIA, heart failure, rheumatic or mitral valve disease (See section on AF [page 43] for calculating the risk of stroke in people with AF)

* When a fasting sample is not possible, measure non-fasting total cholesterol, HDL-cholesterol and HbA1c. A HbA1c ≥6% indicates need for measurement of a true fasting plasma glucose.

† Estimated glomerular filtration rate (eGFR).

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

Table 4 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 ≥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

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 ≥8 mmol/L or if there is a family history of premature coronary heart disease. See Appendix A 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 B for recommended method of measuring BP. See Table 5 for cuff size to use when taking blood pressure.

Table 5 Acceptable blood pressure cuff dimensions for arms of different sizes

Cuff	Arm circumference range at midpoint (cm)	Bladder width (cm)	Bladder length (cm)
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, phaeochromocytoma, Cushing's syndrome and Conn's syndrome.

Interpreting the fasting plasma glucose in people without diabetes

- Fasting plasma glucose is recommended for an initial risk assessment. The recommended action taken depends on the result and risk group (see Table 6). For people found to have elevated levels see Table 7 for the international agreed thresholds for diagnosing diabetes
- When a fasting sample is not possible, measure non-fasting HbA1c. A HbA1c ≥6% indicates need for measurement of a true fasting glucose

Table 6 What to do following the fasting venous plasma glucose result

Result	Action	Why
7.0 mmol/L or more	Repeat a fasting plasma glucose	Two results above this level, on separate occasions,* are diagnostic of diabetes and do not require an OGTT†
6.1–6.9 mmol/L	Request an OGTT†	This level is diagnostic of impaired fasting glucose. Diabetes or impaired glucose tolerance have not been excluded
5.5–6.0 mmol/L	Request an OGTT† in high-risk groups‡	The result may be normal, but some patients will show diabetes or impaired glucose tolerance in an OGTT†
5.4 mmol/L or less	Retest in 5 years or earlier if risk factors for diabetes present	This result is normal

* The diagnosis of diabetes should always 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.

Table 7 Values of venous plasma glucose for diagnosis of diabetes mellitus and other categories of hyperglycaemia

Category	Blood test	Venous plasma glucose (mmol/L)
Diabetes mellitus	Fasting	≥7
	or 2-h post glucose load	≥11.1
	or both	
Impaired glucose tolerance (IGT)	Fasting (if measured)	<7.0
	and 2-h post glucose load	≥7.8 and <11.1
Impaired fasting glycaemia (IFG)	Fasting	≥6.1 and <7.0
	and (if measured) 2-h post glucose load	<7.8

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 (kg/m^2) using Table 8
- A BMI <25 kg/m^2 is desirable
- For people with a BMI ≥35, an initial goal of 10% weight loss may be a realistic target
- For people of Asian descent, a lower BMI may be desirable

Table 8 Classification of weight in adults

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

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.

Cardiovascular risk factor management

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 9)
- 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 9 Goals for people without known cardiovascular disease

CVD risk ≥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.

Goals can be more easily achieved by the simultaneous reduction in several risk factors (see Table 13, page 18).

Risk factors can be used as targets for people at high risk (see Table 10)

Table 10 Optimal levels (targets) for people with known cardiovascular disease, or cardiovascular risk >15% or diabetes

	Known cardiovascular disease or cardiovascular risk >15%	Diabetes	Diabetes and overt nephropathy, microalbuminuria or other renal disease
Lipids			
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		
Blood pressure			
BP	<130/80 mm Hg	<130/80 mm Hg	<125/75 mm Hg
Glycaemic control in people with diabetes			
HbA1c	n/a	HbA1c as close to physiological levels as possible (aim for <7%)	HbA1c as close to physiological levels as possible (aim for <7%)
Smoking cessation			
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			

Graded lifestyle advice is appropriate for everyone (see Table 11)

Table 11 Recommended lifestyle interventions (diet, physical activity, weight management) based on cardiovascular risk assessment

5-year CVD risk	Intervention
• Calculated >20% • Cardiovascular disease • Genetic lipid disorders • Diabetes	**Intensive lifestyle interventions** (see page 23)
• Calculated 10–20%	**Specific lifestyle interventions** (see page 20)
• Calculated <10%	**General lifestyle advice** (see page 19)

Drug therapy is indicated for people with CVD risk ≥15% (see Table 12)

Table 12 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 28, 33 and 34)
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 ≥8 mmol/L or TC:HDL ratio ≥8 or BP ≥170/100)
All levels of CVD risk	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

The higher an individual's absolute risk of a cardiovascular event the more aggressive the management should be

Table 13 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)

* 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.

† Smoking cessation treatment should combine pharmacotherapy and behavioural support.

‡ People with isolated high risk-factor levels, either total cholesterol ≥8 mmol/L or TC:HDL ratio ≥8 or blood pressure ≥170/100 mm Hg, should have these risk factors treated and their risk recalculated.

General lifestyle interventions

Offer everyone advice promoting 'healthy heart' foods and a smoke-free, active lifestyle (see Table 14)

Table 14 General lifestyle advice for people at 5-year cardiovascular risk <10%

The Heart Foundation's 9 steps to eating for a healthy heart	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 2. Choose fruits and/or vegetables at every meal and most snacks 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 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 5. Choose low fat milk, low fat milk products, or replace with soy products 6. Use small amounts of oil, margarine, nuts or seeds 7. Drink plenty of fluids each day, particularly water, and limit sugar-sweetened drinks and alcohol 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 9. Mostly avoid or rarely include butter, deep-fried and fatty foods, and only occasionally choose sweet bakery products
Physical activity	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 Push Play – http://pushplay.sparc.org.nz
Healthy weight	• BMI <25 • Waist circumference <100 cm in men or <90 cm in women
Quit smoking	Quitting smoking has major and immediate health benefits for smokers of all ages and their families 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 19–23 (pages 24–30)

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
- 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 15) 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 16 and 17)

Table 15 Assessment of physical activity

	Level	Description
Inactive	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
Active	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

Table 16 Specific lifestyle and behavioural risk factor management for people at 5-year cardiovascular risk of 10–20%

Risk factor	Assessment and advice
Nutrition	• Assess general dietary habits against the National Heart Foundation's 9 steps to eating for a healthy heart (Table 14) • Complete a lifestyle assessment diary • Quantify intake and offer advice on the cardioprotective dietary pattern table (Appendix C)
Physical activity	• Assess the current level of physical activity: duration and frequency (Table 15), intensity and type (Appendix D). Sports and leisure activities with energy expenditure of 3–6 METs meet the definition of 'moderate physical activity' • Complete a lifestyle assessment diary • 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 • People who are already active at level 3 should be encouraged to do physical activity of higher intensity or for longer (aim for ≥6 METS or level 4) • Consider issuing a green prescription/referring to a local sports trust
Weight	• Assess/monitor waist circumference and BMI. Commence lifestyle change if BMI ≥25 (especially if ≥30) • Ask about previous weight loss attempts and programmes • Complete a lifestyle assessment diary • Set achievable goals, prevent weight gain, achieve and sustain moderate weight loss (5–10%) where appropriate and increase physical fitness • 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 • Reduce foods rich in fats and oils, particularly saturated fat-rich foods and deep-fried products • Reduce white flour products and partially replace with whole grain products • Reduce foods and drinks rich in added sugars (bakery and confectionery items) • Ensure nutritional adequacy and cardiovascular protection • Consider the metabolic profile and other goals (including glycaemic, LDL-C, HDL-C, triglyceride levels and BP)

continued over...

Table 16: continued...

Risk factor	Assessment and advice
Smoking	• Ask about and document smoking status prominently in medical record • 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) • Offer cessation treatment to all smokers and provide treatment to those who want to stop (details of smoking cessation treatments are given in Tables 19–23, pages 24–30)

Table 17 Specific lifestyle changes to modify biomedical risk factors

Risk factor	Assessment and advice
Lipid modification	• Adopt a cardioprotective dietary pattern (Appendix C) • Consider adding plant sterol or stanol-fortified spreads • Eat oily fish regularly • Choose foods which are low in saturated fatty acids, transunsaturated fat and dietary cholesterol
BP lowering	• Adopt a cardioprotective dietary pattern (Appendix C) • Reduce excessive alcohol intake (no more than 3 standard drinks/day for men or 2 standard drinks/day for women) • Reduce sodium intake to no more than 2 g/day (6 g sodium chloride)
Diabetes IGT IFG	• Intensive lifestyle advice for people with disorders of carbohydrate metabolism should be given in individual/group sessions with a dietitian. See *Management of Type 2 Diabetes* guideline (Chapter 2 and Appendix B) for details • The specific interventions that are known to reduce behavioural, lipid and BP risk factors in people without diabetes are also recommended for people with diabetes • A cardioprotective diet in people with type 2 diabetes who are overweight or obese should be tailored to promote weight loss • Reduce foods rich in saturated fat, added sugars and white flour bakery products • To control post-prandial hyperglycaemia, include high-fibre foods with a low to moderate glycaemic index at each meal, distribute carbohydrate foods evenly through the day and avoid a large volume of carbohydrate-rich foods at any one meal • Refer to a dietitian and diabetes nurse specialist

Intensive lifestyle interventions

- Intensive lifestyle advice is recommended for people with 5-year CVD risk >20% and some other high risk groups (see Table 18)
- 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 dietician

Table 18 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 dietician 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 19–23, pages 24–30) • Refer to smoking cessation treatment provider (eg, Quitline, Aukati Kai Paipa, local provider www.smokefreecontacts.org.nz)

Smoking cessation interventions

- Smoking cessation has major and immediate health benefits for all smokers
- Smoking cessation treatment should **follow the ABC approach** (see Table 19)
- The provision of pharmacotherapy to smokers with CVD risk is highly indicated (see Tables 20–22, pages 26–29)
- The ABC approach should be repeated at follow-up visits to provide further assistance and to ensure that repeated quit attempts are made

Table 19 The ABC of smoking cessation

The New Zealand Smoking Cessation Guidelines 2007* recommend the use of ABC as a memory aid for smoking cessation interventions:

A is for asking all people if they smoke

B is for giving brief advice to stop smoking

C is for cessation support, which should be offered to all smokers who have an interest in stopping

A	**Ask about and document smoking status** • All patients should have their smoking status documented in their clinical record as a vital sign • Smoking status should be updated regularly
B	**Brief advice to quit** • One of the most important interventions a health professional can deliver • Brief advice to quit roughly doubles the chances of long-term quitting • It can be delivered in under a minute • Brief advice should: – contain a clear message to stop smoking completely (do not advise to 'just cut down') – be linked to a current illness if appropriate (eg, 'stopping smoking will reduce your risk of having a heart attack') – be given to all smokers regardless of whether they want to quit or not (assessment of the stage of behavioural change is not necessary) • 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')

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
  - **Aukati Kai Paipa**: a smoking cessation service provided by Māori organisations for Māori who smoke. Tel: (09) 638 5800 Website: 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
- provision of a **smoking cessation medicine** (see Tables 20–22, pages 26–29). These work by alleviating symptoms of nicotine withdrawal (eg, cravings, irritability, poor concentration)

* The full guideline is available at http://www.moh.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 20–22).

Table 20 Nicotine replacement therapy

Use of NRT

- Provides some of the nicotine a smoker would have otherwise got from cigarettes
- Roughly doubles the chances of quitting long-term compared to placebo
- Nicotine **patches**, **gum**, and **lozenges** are subsidised and available via the QuitCard Scheme
- Provide or refer for behavioural support and follow-up to increase likelihood of success
- The choice of NRT product can be guided by individual preference
- 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
- Combining two NRT products (eg, patch and gum) increases abstinence rates and is safe

Patches 21 mg/24 hr	• Also available as a 16-hour patch (15 mg/16 hr). There is no difference in efficacy between 16- and 24-hour patches • 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) • The advantages of patches are that they are very simple to use and people generally use them reliably as instructed • Patches are applied to a clean, dry, hairless area of skin and removed at the end of the day (16 hours) or the next day (24 hours) • Skin irritation is the most common side effect
Gum 2 mg and 4 mg	• People who are highly dependent should use 4 mg gum • 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 • 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) • 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) • 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

Lozenge 1 mg and 2 mg	• People who are highly dependent should use the higher dose lozenge • Use one lozenge per hour
Sublingual tablet 2 mg	• These are placed under the tongue where they are left to dissolve • They should be used on an hourly basis
Inhaler	• The inhaler is a small plastic tube containing a replaceable nicotine cartridge • The user should puff on the inhaler for 20 minutes each hour. After four 20-minute puffing sessions, the cartridge should be changed
Exchange card programme (QuitCards)	• Gives a 4-weeks supply of nicotine patches (24 hr only), gum and lozenges to any smoker • Cost: $5 per item on each card • Available via all prescribers, QuitCard providers, Quitline (0800 778778), Quit Group website (www.quit.org.nz)

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

Table 21	Non-nicotine pharmacotherapies for smoking cessation
Varenicline (Champix)	• This medicine was designed specifically for smoking cessation. It acts on nicotinic acetylcholine receptors to reduce the severity of nicotine withdrawal symptoms • It approximately triples the chances of quitting long-term compared to placebo • It is not recommended for use in children under the age of 18 years and women who are pregnant or breastfeeding • There are no clinically-significant drug interactions to consider • 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 • The most common adverse effect is nausea • People should be warned that they may experience a change in their mood and to report anything concerning to their doctor
Bupropion (Zyban)	• Bupropion is an atypical antidepressant that also increases the chances of stopping smoking long-term (approximately doubles the chances compared to placebo) • It is a prescription-only medicine and is not subsidised • There are a number of contraindications and cautions (see NZ Smoking Cessation Guidelines 2007, 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 • People need to start this medicine a week before their quit day. The dosage is a 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 • Adverse effects include dry mouth, headache, and there is a small seizure risk
Nortriptyline	• This tricyclic antidepressant has also been found to approximately double the chances of long-term abstinence compared to placebo • 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 • 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 • Adverse effects, such as dry mouth and sedation, are common

Current evidence shows NRT to be safe in people with cardiovascular disease (see Table 22)

Table 22 Cardiovascular disease and smoking cessation therapies

NRT	• NRT can be provided to people with cardiovascular disease; dosage adjustment is required • 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
Varenicline	• Suitable treatment, if appropriate • There are no data regarding use of varenicline in people with acute CVD
Bupropion	• Suitable treatment, if appropriate
Nortriptyline	• Contraindicated in acute recovery phase after MI

Quitting at any point in pregnancy can be beneficial for the foetus and mother (see Table 23)

Table 23 Smoking cessation in pregnancy and breastfeeding

NRT in pregnancy	• Manufacturers do not recommend NRT; however, NRT is safer than smoking • 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 • NRT may be used in women who are breastfeeding. More detailed information can be found in the *New Zealand Smoking Cessation Guidelines*, 2007

Minor weight gain is common when people stop smoking (see Table 24)

Table 24 Smoking cessation and weight gain

Weight gain	• On average, people can expect to gain 4–5 kg in the first year of abstinence • Although this is a significant gain, the benefits of stopping smoking outweigh the health risks of the additional weight gain • '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 • For smokers concerned with weight gain, consider bupropion or NRT, in particular gum, which has been shown to delay weight gain after quitting

Therapy

Complementary and alternative therapies

Clinicians should enquire about the use of alternative and complementary medicines when assessing cardiovascular risk or prescribing medication (see Table 25)

Table 25 Adverse effects of some complementary or alternative medicines

Complementary or alternative medicine	Effect
Feverfew, garlic, *Ginkgo biloba*, 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

Note: There is **insufficient evidence** to recommend the following complementary and alternative therapies for the treatment or prevention of CVD: herbal medicines/botanicals (garlic, *Ginkgo biloba*, rosemary, horse-chestnut seeds, xin bao), acupuncture, chelation therapy, traditional Chinese medicine, aromatherapy, homeopathy, hypnosis, meditation, yoga, tai chi, intercessionary prayer, Strauss heart drops.

* Randomised controlled trials

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 26). There is no normal or ideal lipid level. Risk factors can be viewed as treatment targets for people at high risk (see Table 27)

Table 26 Clinical scenario and intervention recommendations

TC 4–8 mmol/L	• All decisions to treat should be based on the individual's cardiovascular risk
Isolated risk factors: TC ≥8 mmol/L or TC:HDL ratio ≥8	• Assume 5-year CVD risk to be at least 15% • Calculate risk using the charts as CVD risk may be higher than this • Commence specific individualised lifestyle advice, smoking cessation treatment, aspirin, lipid-modifying therapy, +/- BP lowering therapy
CVD risk >20% clinically*	• Commence statin (unless contraindicated) simultaneously with intensive lifestyle advice, smoking cessation treatment, aspirin, and other appropriate medication
Calculated CVD risk >20%	• Aim is to reduce 5-year CVD risk to <15%, which can be more easily achieved by reduction of all modifiable risk factors • Commence intensive lifestyle advice, smoking cessation treatment, simultaneously with aspirin and drug treatment of all modifiable risk factors
Calculated CVD risk 15–20%	• Aim is to reduce 5-year CVD risk to <15%, which can be more easily achieved by simultaneous reduction of all modifiable risk factors • Smoking cessation treatment • Commence specific individualised lifestyle advice for 3–6 months before considering aspirin, lipid-modifying or BP lowering therapy
Calculated CVD risk 10–15%	• Smoking cessation treatment • Specific individualised lifestyle advice on a cardioprotective dietary pattern and physical activity
Calculated CVD risk <10%	• Smoking cessation treatment • General lifestyle advice, including dietary advice on a cardioprotective diet, physical activity

* 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.

Table 27 Optimal lipid levels (targets) for people with known cardiovascular disease, or cardiovascular risk >15% or diabetes

	Known cardiovascular disease or cardiovascular risk >15%	Diabetes	Diabetes and overt nephropathy, microalbuminuria or other renal disease
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:
  - For people with known CVD or CVD risk >20%, simvastatin 40 mg
  - For people with 5-year CVD risk 15–20% if initiating drug therapy, simvastatin 20 mg and titrate if needed
- 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 of 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 ≥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
- <125/75 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 C) 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

Acute blood pressure lowering therapy in ischaemic stroke

- Continue existing antihypertensive drugs unless the person has symptomatic postural hypotension
- Do not treat raised BP in the acute phase unless systolic BP is ≥220 mm Hg or diastolic BP ≥120 mm Hg. Avoid sublingual nifedipine. If BP lowering is required, use short-acting agents that have minimal effects on cerebral vessels, such as labetalol

Secondary prevention post-acute ischaemic stroke or transient ischaemic attack

- Start or increase BP lowering medication irrespective of the BP level (unless the person has symptomatic hypotension). Benefits are seen when systolic BP lowering in the order of 12 mm Hg or greater is achieved compared with pre-treatment levels. Two drugs are often required
- 7 to 14 days delay is usual before starting BP lowering medication

- Treatment should start concurrently with intensive lifestyle advice
- The combination of an ACE inhibitor and thiazide diuretic is proven to reduce recurrent stroke and other major vascular events. There is insufficient evidence to determine if other BP lowering medications/ combinations are equally effective
- BP lowering therapy should be given in addition to other appropriate medication such as aspirin, a statin or warfarin (if indicated)
- All people who smoke should be strongly advised to stop and offered smoking cessation treatment
- Individualising treatment targets for people after a stroke should take into account the number and dose of medications prescribed, as well as comorbidities

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
- Aggressive BP control (<125/75 mm Hg) is indicated in people with diabetes and overt nephropathy, or diabetes and microalbuminuria, or diabetes and other renal disease
- ACE inhibitors are preferred therapy in diabetes, but the addition of low dose thiazide diuretics, calcium channel blockers or beta-blockers may be helpful in lowering BP and reducing the risk of CVD. A2 receptor blockers should be considered where ACE inhibitors are not tolerated
- Commence an ACE inhibitor or A2 receptor blocker (if there are no contraindications) **irrespective** of BP levels in diabetes and overt nephropathy or diabetes and confirmed microalbuminuria, because of the additional renal protection benefits that are obtained. While the addition of an ACE inhibitor or an A2 receptor blocker has been shown to reduce albuminuria, an improvement in cardiovascular and long-term renal outcomes has not been confirmed and side effects (eg, hypotension) are more frequent
- All people who smoke should be strongly advised to stop and offered smoking cessation treatment

Chronic kidney disease

- Aggressive management of blood pressure (target BP levels <125/75 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 28)

Table 28 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 Low dose aspirin is as effective as higher daily doses and may be associated with less bleeding
Risk assumed to be >15%: isolated high-risk factors • TC ≥8 mmol/L • TC:HDL ratio ≥8 • BP ≥170/100 mm Hg	
No clinical CVD and calculated 5-year CVD risk <15%	The risk of a significant bleed or major haemorrhage **outweighs** 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.

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 29)
- All people who smoke should be strongly and repeatedly advised to stop and offered smoking cessation treatment

Table 29 Recommended medications after myocardial infarction or angina

Drug	Recommendation
Aspirin	Aspirin 75–150 mg should be given routinely and continued for life. These doses are at least as effective as higher doses
Clopidogrel	Clopidogrel (75 mg/day) is an effective alternative to aspirin for people with contraindications to aspirin or those who are intolerant of aspirin
Warfarin	Warfarin should be prescribed for high-risk MI survivors including those with: • atrial fibrillation or paroxysmal atrial fibrillation • a large left ventricular aneurysm • thrombus demonstrated in the left ventricle at the infarction site by echocardiography • systemic embolism Consider warfarin in people who cannot be given antiplatelet agents after MI The target INR should be 2.5 (range 2.0–3.0)

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Table 29: continued...

Beta-blockers	Beta-blockers (eg, metoprolol, timolol, propranolol) should be considered for everyone following MI unless contraindicated Beta-blockers are also recommended in those with left ventricular dysfunction and heart failure • The initial dose of beta-blockers may be low and the dose may then be slowly titrated • Beta-blockers given at night may reduce the risks of postural hypotension and alleviate symptoms of tiredness and lethargy • Before discontinuing beta-blockers because of side effects, a lower dose or alternative beta-blocker should be tried • 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
ACE inhibitors	An ACE inhibitor should be considered for everyone after MI Treatment should be started early and continued, especially in those with anterior infarction, LV dysfunction or heart failure
Statins	A statin equivalent to simvastatin 20–40 mg daily should be started after MI
Calcium channel blockers	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
Nitrates	Nitrates can be used after MI for controlling symptoms of angina, but are not indicated for reducing the risk of further events
Smoking cessation treatments	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, page 26)

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.

Atrial fibrillation and atrial flutter: assessment and therapy

Assessment of a first episode

A new diagnosis of atrial fibrillation (AF) will be suspected after detecting an irregular pulse or irregular heart rhythm and an electrocardiogram (ECG) should be performed to confirm AF.

All people presenting with AF or atrial flutter (AFL) for the first time should have the following investigations:

- history and clinical examination
- ECG
- transthoracic echocardiogram (TTE)
- blood tests – thyroid function, renal function (creatinine), INR (pre-warfarin).

Stroke risk assessment in people with atrial fibrillation

The risk of ischaemic stroke and MI should be assessed using the New Zealand Cardiovascular Risk Charts (see Figure 2, page 44) and a decision made on the appropriateness of lipid-modification and BP lowering medication.

The thromboembolic stroke risk should be assessed (see Figure 2) and a decision made on the appropriateness of warfarin or aspirin therapy.

The risk of bleeding (see Table 30) and contraindications to warfarin (see Table 31) should be considered and discussed with the person.

People with previous AF or paroxysmal AF who are in sinus rhythm remain at increased thromboembolic risk and should have their risk of stroke calculated to determine appropriate therapy – oral anticoagulation or aspirin (see Figure 2).

Figure 2 Baseline risk of stroke in people with new-onset atrial fibrillation (and without prior TIA or stroke) from Framingham Data (5-year stroke risk in %)

People with atrial fibrillation (AF) and either significant valvular disease, prior stroke or TIA are at **VERY HIGH** risk of stroke and do not need risk stratification. They should receive long-term warfarin, unless contraindicated

People with AF and either left ventricular dysfunction (LVEF ≤40%) or a past episode of decompensated heart failure are at **HIGH** risk and should receive long-term warfarin, unless contraindicated

Men				Women			
Systolic blood pressure (mm Hg)	No Diabetes	Diabetes		Systolic blood pressure (mm Hg)	No Diabetes	Diabetes	
180	13	22	Age ≥75	180	23	37	Age ≥75
160	11	19		160	20	34	
140	10	17		140	18	31	
120	9	15		120	16	28	
180	10	17	Age 65–74	180	18	29	Age 65–74
160	9	15		160	16	27	
140	8	13		140	14	24	
120	7	12		120	13	21	
180	7	13	Age <65	180	13	22	Age <65
160	6	11		160	12	20	
140	6	10		140	11	17	
120	5	9		120	10	16	

Key

Risk of stroke over 5 years		Treatment
Very high	≥ 20%	Long-term anticoagulant treatment with adjusted dose warfarin (after discussion) aiming for an INR 2.5 (range 2.0 to 3.0) unless there are clear contraindications
or High	15–19%	
Intermediate	10–14%	Discuss the individual's potential benefits, risks and preferences for or against anticoagulant or aspirin treatment
Low	<10%	Commence aspirin (75 to 300 mg) after discussion

Note: In people with a contraindication to warfarin, consider using aspirin (75 to 300 mg) after discussion.

How to use the charts

- Identify the chart relating to the person's age, sex and diabetic status
- Within the chart choose the cell nearest to the person's usual systolic BP. For example, the lower left cell contains all men without diabetes who are less than 65 years and have a usual systolic BP less than 130 mm Hg
- People who fall exactly on a threshold between cells are placed in the cell indicating higher risk

Note: Stroke risk may be greater for people with a history of treated hypertension than for those without such a history, for a given level of BP.

Source: Wang TJ, Massaro JM, Levy D et al. A Risk Score for Predicting Stroke or Death for Individuals with New-Onset Atrial Fibrillation in the Community: The Framingham Heart Study. JAMA 2003;290:(8)1049–56.

Table 30 Benefits and harms of treatment with warfarin compared to aspirin

Benefit of warfarin*			Benefit of aspirin†		Bleeding with warfarin‡		Intracranial haemorrhage with aspirin
5 year stroke risk %	Strokes prevented per 100 people treated for 5 years	NNT for 5 years to prevent one stroke	Strokes prevented per 100 people treated for 5 years	NNT for 5 years to prevent one stroke	Major bleeding with warfarin per 100 people treated for 5 years	Intracranial haemorrhage per 100 people treated with warfarin for 5 years	Intracranial haemorrhage per 100 people treated with aspirin for 5 years
30	20	5	6	17	10	2.5	1.5
20	13	8	4	25	10	2.5	1.5
15	10	10	3	33	10	2.5	1.5
10	7	15	2	50	10	2.5	1.5
5	3	30	1	100	10	2.5	1.5

Note: Major bleeding is that which requires hospital admission, transfusion or was fatal (the definition includes intracranial, respiratory or abdominal bleeds). The risk of intracranial haemorrhage is 0.5 per 100 patients per year on warfarin and 0.3 per 100 patients per year on aspirin.

NNT = Number needed to treat

Estimates:

* based on the estimate that warfarin reduces strokes in people with AF by 66%.

† based on the estimate that aspirin reduces strokes in people with AF by 20%.

‡ based on the estimate that the incidence of major bleeding with warfarin is 2% per year.

Source: meta-analysis data from van Walraven, C. Hart, R. Singer, D. et al. JAMA 2002;288(19):2441–2448.

Table 31 Contraindications to treatment with warfarin

Absolute contraindications	Relative contraindications	NOT contraindications to receiving warfarin
• Bleeding diathesis • Thrombocytopenia • Poorly controlled hypertension (BP consistently ≥160/100 mm Hg) • Non-compliance with medication or INR monitoring • Previous intracranial bleed or retinal haemorrhage • Recent gastrointestinal/ genitourinary bleeding • First trimester and last month of pregnancy	• Significant alcohol use (≥60 ml/day or ≥5 standard drinks/day) or liver disease • Conventional NSAID use (without cytoprotection) • Participation in activities predisposing to trauma • Unexplained anaemia • Dementia • Multiple comorbidity • Unexplained recurrent syncope	• Predisposition to falling – clinical judgment required • Advanced age alone – clinical judgment required • NSAID use with misoprostol or a proton pump inhibitor • COX2-inhibitor use • Recent resolved peptic ulcer disease with successful treatment of *Helicobacter pylori* • Previous ischaemic stroke

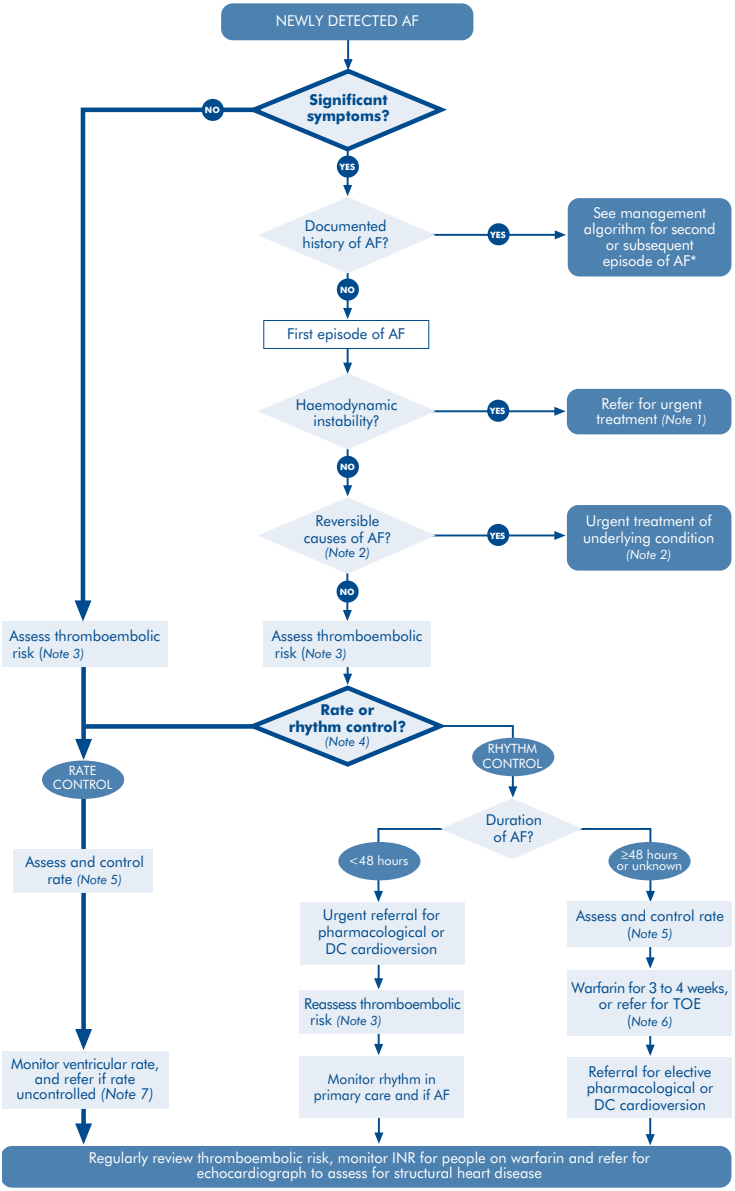
Therapy for atrial fibrillation or flutter

- Antithrombotic treatment (oral anticoagulation or aspirin) should be administered to all people with AF/AFL, except those with lone AF (AF in people <60 years with no hypertension or heart disease)
- People with previous AF or paroxysmal AF who have converted to sinus rhythm remain at increased thromboembolic risk. They should be assessed for thromboembolic risk (see Figure 2, page 44) and treated with warfarin or aspirin
- Rate control together with anticoagulant therapy, rather than rhythm control, is recommended for the majority of people with asymptomatic AF/AFL
- If a rhythm control strategy is chosen for people who are not anticoagulated they should be cardioverted within 48 hours of onset. If they cannot be cardioverted within 48 hours of onset then they should **either** have a therapeutic INR (2.0–3.0) for at least 3 weeks **or** a transoesophageal echocardiogram to exclude atrial thrombi before cardioversion

- The efficacy and safety of antiarrhythmic drugs vary depending on the indication and individual clinical factors. For example, sotalol should NOT be used solely for rate control. It appears to be ineffective for pharmacological cardioversion, but is effective for maintenance of sinus rhythm
- People on antiarrhythmic therapy require regular monitoring. The main risk of antiarrhythmic therapy is ventricular proarrhythmia
- The principles of rate control and thromboembolic prophylaxis apply equally to people with AF and AFL

For more details on how to initiate warfarin therapy see the guideline, *The Management of People with Atrial Fibrillation and Flutter,* 2005, or the guideline summary available at www.nzgg.org.nz

Figure 3 First episode of atrial fibrillation



* See the guideline *The Management of People with Atrial Fibrillation and Flutter*, 2005, available at www.nzgg.org.nz

Notes

Note 1 Haemodynamic instability

People who are critically ill with the following conditions as a result of their rapid AF/AFL should be considered for **immediate** direct current (DC) cardioversion and other emergency procedures as appropriate

- Shock or impending shock
- Rate-related angina
- Rate-related myocardial ischaemia (on ECG)
- Acute pulmonary oedema
- Prompt DC cardioversion is indicated for people with pre-excited AF (WPW syndrome)

Note 2 Reversible causes of atrial fibrillation

People with the following should be considered for urgent management of the underlying condition (spontaneous AF reversion rate is high and recurrence low). Consider rate control and thromboembolic risk as usual

- MI
- Pulmonary embolus
- Pneumonia
- Cardiac or other surgery
- Thyrotoxicosis

Note 3 Thromboembolic risk and warfarin

(Also see Figure 2, page 44)

All people with AF/AFL (whether paroxysmal, persistent or permanent) should have their thromboembolic risk assessed. In the acute setting, IV or subcutaneous heparin should be started pending a decision on possible cardioversion or warfarin therapy, unless contraindicated

continued over...

Figure 3 continued...

Note 4	Rhythm control; conversion of AF to sinus rhythm

(See Chapter 5 of the AF guideline)

Rhythm control is the preferred treatment for people with:

- unacceptable symptoms from AF/AFL
- pre-excited AF/WPW syndrome
- haemodynamic compromise due to AF/AFL
- younger age, paroxysmal AF and little or no heart disease

(See section 5.2 of the AF guideline)

Electrical cardioversion: appropriate synchronised shock energy levels are:

- monophasic waveform – initially 200 J, then 300 to 360 J
- biphasic waveform – initially 100 or 120 J, then 150 to 200 J

Pharmacological cardioversion: amiodarone, flecainide or propafenone are effective

Note 5	Rate control

(See Chapter 7 of the AF guideline)

Rate control is the recommended choice for most people with asymptomatic AF/AFL (see Table 32)

Management of all people with AF/AFL (including those in whom a rhythm control strategy is chosen) should include assessment and control of ventricular rate

The aim is to achieve a resting ECG/apical rate of <80 bpm AND a moderate walk rate (eg, after 6 minutes walking) of <115 bpm. Heart-rate control can be further assessed by 24-hour Holter monitoring or exercise testing (either formal treadmill or corridor walk to the point of breathlessness). Occasionally, AV node ablation and permanent pacemaker implantation are required if heart rate control is suboptimal

continued over...

Note 5 continued...

Selection of a rate control agent for people with AF

Comorbidity	First-line	Second-line	Less effective or desirable
No heart disease	Beta-blockers* OR calcium channel blockers†		Digoxin‡
Hypertension	Beta-blockers* OR calcium channel blockers†		Digoxin‡
Ischaemic heart disease	Beta-blockers*	Calcium channel blockers† OR digoxin‡	Ablation + pacing
Congestive heart failure	Digoxin in overt heart failure Carvedilol or metoprolol in stable heart failure	Beta-blockers* OR diltiazem	Amiodarone Ablation + pacing should be considered
Chronic obstructive pulmonary disease	Calcium channel blockers†	Beta-blockers* (if no significant reversible bronchospasm)	Digoxin‡

* beta-blockers – atenolol, carvedilol, metoprolol, nadolol, propranolol (NOT sotalol).

† diltiazem or verapamil.

‡ digoxin is not as good at controlling the rate with exercise, but can be added to the above therapeutic groups or used as first-line in people unlikely to be active.

Sotalol should NOT be used for the purpose of rate control because of its higher incidence of life-threatening ventricular arrhythmias (particularly torsade de pointes)

A combination of rate control agents is sometimes required to achieve adequate rate control. The combination of a beta-blocker with verapamil should be used with considerable caution

continued over...

Figure 3 continued...

Note 6	Transoesophageal echocardiography (TOE) guided DC cardioversion

(See section 8.1.3, Electrical cardioversion in the AF guideline)

No visible left atrial thrombus on TOE allows safe DC cardioversion even >48 hours after AF onset. Anticoagulation for 4 weeks after cardioversion is still required. If LA thrombus is detected, DC cardioversion should be delayed for 3 to 6 weeks and the thrombus should be reassessed by TOE prior to proceeding with DC cardioversion

Note 7	Nonpharmacological therapy

(See sections 7.3 and 8.2.3 of the AF guideline)

Carefully selected people may be considered for nonpharmacological therapy, such as:

- AV node ablation and permanent pacemaker implantation for rate control
- atrial pacemaker implantation for rhythm control
- atrial defibrillator implantation for rhythm control
- catheter ablation for rhythm control
- surgical ablation (eg, MAZE procedure) for rhythm control

Stroke and transient ischaemic attack

Transient ischaemic attack

Transient ischaemic attack (TIA) is a medical emergency – people with TIA are at high risk of early stroke.

- The risk of stroke 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

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

- Aspirin should be started immediately if fully recovered and no contraindications (see Table 32, page 56)
- All people with suspected TIA should be assessed at initial point of health care contact for their risk of stroke, including their ABCD2 score (see Figure 4, page 54)
- The ABCD2 tool (see Figure 4) 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

People at high risk (include those with ABCD2 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 ABCD2 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 4 ABCD2 tool: assessment of stroke risk

Prediction of stroke risk after transient ischaemic attack

ABCD2 items (score: 0–7)		Points
A	**Age:** ≥60 years	1
B	**Blood pressure:** ≥140/90 mm Hg	1
C	**Clinical features:**	
	unilateral weakness or	2
	speech impairment without weakness	1
D	**Duration of symptoms:**	
	≥60 minutes or	2
	10–59 minutes	1
D	**Diabetes: (on medication/insulin)**	1

Risk of stroke according to ABCD2 scores

ABCD2 score	0–3	4–5	6–7
Proportion of all TIAs	34%	45%	21%
Stroke risk (%) at:			
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(9558):283–92 cited in the *New Zealand Guideline for the Assessment and Management of People with recent Transient Ischaemic Attack (TIA)*. www.stroke.org.nz

Secondary prevention after transient ischaemic attack

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 (see Table 32, page 56) including:

- anti-platelet agent(s) – aspirin, aspirin plus dipyridamole or clopidogrel
- BP lowering therapy

- statin
- warfarin – if atrial fibrillation or other cardiac source of emboli
- nicotine replacement therapy or other smoking cessation aid.

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

Stroke

All people with a definitive/presumptive diagnosis of stroke should be admitted unless:

- symptoms have fully resolved or are rapidly recovering so that there is no significant disability affecting functioning **and**
- urgent outpatient assessment by a specialist stroke service is available **or** the person is already in appropriate institutional care **or** the person/family prefer home care despite explanation of the benefits of hospital care.

If not admitted, the treating doctor must consider diagnosis, secondary prevention, home support and rehabilitation needs.

A CT scan should be obtained within 48 hours of onset of symptoms. Ischaemic and haemorrhagic stroke cannot be reliably distinguished on clinical grounds.

- Aspirin 150 to 300 mg should be given as soon as possible after the onset of a stroke in most patients if intracerebral haemorrhage has been excluded with brain imaging. If brain imaging will be delayed, then treatment may be initiated safely prior to imaging and discontinued if intracerebral haemorrhage detected subsequently.

	All people after an ischaemic stroke or TIA should be on aspirin and a statin unless contraindicated (see Table 34). Start or increase BP lowering therapy, irrespective of BP level
	All people who smoke should be strongly advised to stop and offered smoking cessation treatment

Table 32	Recommended medication after ischaemic stroke or transient ischaemic attack
Drug	**Recommendation**
Aspirin	Acute aspirin therapy in ischaemic stroke or TIA • Aspirin 150–300 mg/day should be given as soon as possible after the onset of a stroke in most patients if intracerebral haemorrhage has been excluded with brain imaging. If symptoms/signs have completely resolved (TIA) or brain imaging will be delayed, then treatment may be initiated safely prior to imaging and discontinued if intracerebral haemorrhage detected subsequently Long-term aspirin therapy in ischaemic stroke or TIA • Aspirin 75–150 mg should be given routinely, long-term after ischaemic stroke or TIA, unless there is an indication for anticoagulation with warfarin. These doses are at least as effective as higher doses
Clopidogrel	Clopidogrel (75 mg/day) can be used as a safe and effective alternative to aspirin after stroke
Dipyridamole	Insufficient evidence to recommend dipyridamole as a first-line treatment for the secondary prevention of vascular events, either as monotherapy or in combination with aspirin 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

Warfarin	Warfarin should not be prescribed for people with TIA or minor strokes unless cardiac embolism is suspected Warfarin should be considered for people after ischaemic stroke associated with: • AF unless contraindicated • mitral valve disease • prosthetic heart valves • MI in the previous 3 months. Warfarin should ideally be started in hospital. • For minor stroke, it can be started after the first 48 hours if haemorrhage has been excluded by brain imaging • For major stroke a delay for 7–14 days may be preferable The target INR should be 2.5 (range 2.0–3.0)
BP lowering medication	Acute BP lowering therapy in ischaemic stroke • Continue existing antihypertensive drugs unless the person has symptomatic postural hypotension • Do **not** treat raised BP unless systolic BP is ≥220 mm Hg or diastolic BP ≥120 mm Hg. Avoid sublingual nifedipine. If BP lowering is required, use short-acting agents that have minimal effects on cerebral vessels, such as labetalol Long-term BP lowering in ischaemic stroke • BP lowering therapy is recommended for all people after stroke or TIA, irrespective of baseline BP (unless they have symptomatic hypotension). Two drugs are often required • It is usually advisable to wait 7–14 days after an acute stroke before starting BP lowering medication • BP targets after a stroke should take into account the number and dose of medications prescribed as well as comorbidities • The combination of an ACE inhibitor and thiazide diuretic is proven to reduce recurrent stroke and other major vascular events. There is insufficient evidence to determine if other BP lowering medications/combinations are equally effective • Periodically monitor electrolytes and renal function

continued over...

Table 32: continued...

Lipid modification	A statin is recommended for most people following ischaemic stroke or TIA. Statin therapy should preferably be started in hospital
Smoking cessation treatment	Nicotine replacement therapy can be used after ischaemic stroke or TIA. See Tables 19–23 (pages 24–30) for further details on smoking cessation treatment.

Rheumatic fever

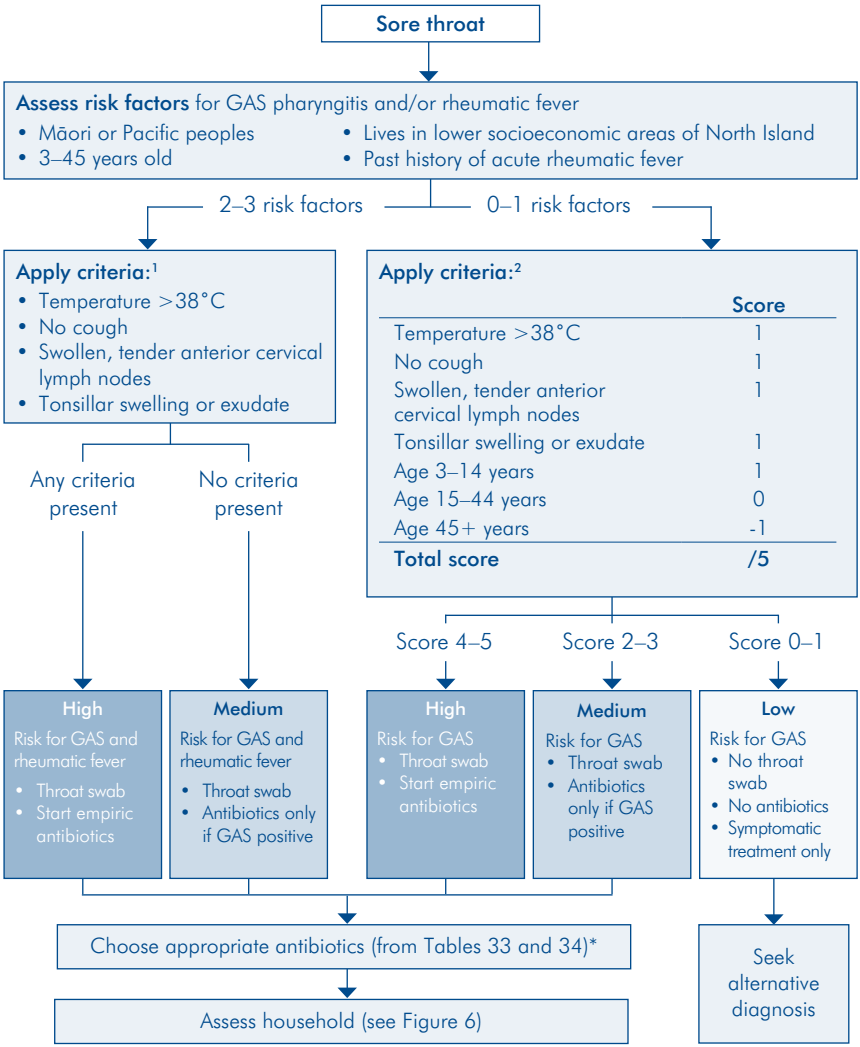
A guideline for the management of rheumatic fever was produced by the Heart Foundation in 2006. The following content is taken from this guideline. A full copy of the guideline is available from www.nhf.org.nz

	Treatment of Group A streptococcus pharyngitis with appropriate antibiotics reduces the occurrence of acute rheumatic fever (ARF)
	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
	Jones' (1992) diagnostic criteria (modified for the New Zealand guidelines) should be used to determine definite, probable and possible ARF (see Table 35). The criteria should not be rigidly adhered to when ARF is the most likely diagnosis
	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

Sore throat management

- GAS sore throats are considered to be the only clinically significant bacterial throat infection in the New Zealand population
- Children with GAS pharyngitis should be kept home from school or day-care for 24 hours until treatment is established
- Treatment of GAS pharyngitis can be delayed until culture results are available for up to nine days, as rheumatic fever is unlikely to occur in this time
- 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 33 and 34)

Figure 5 Guide for sore throat management



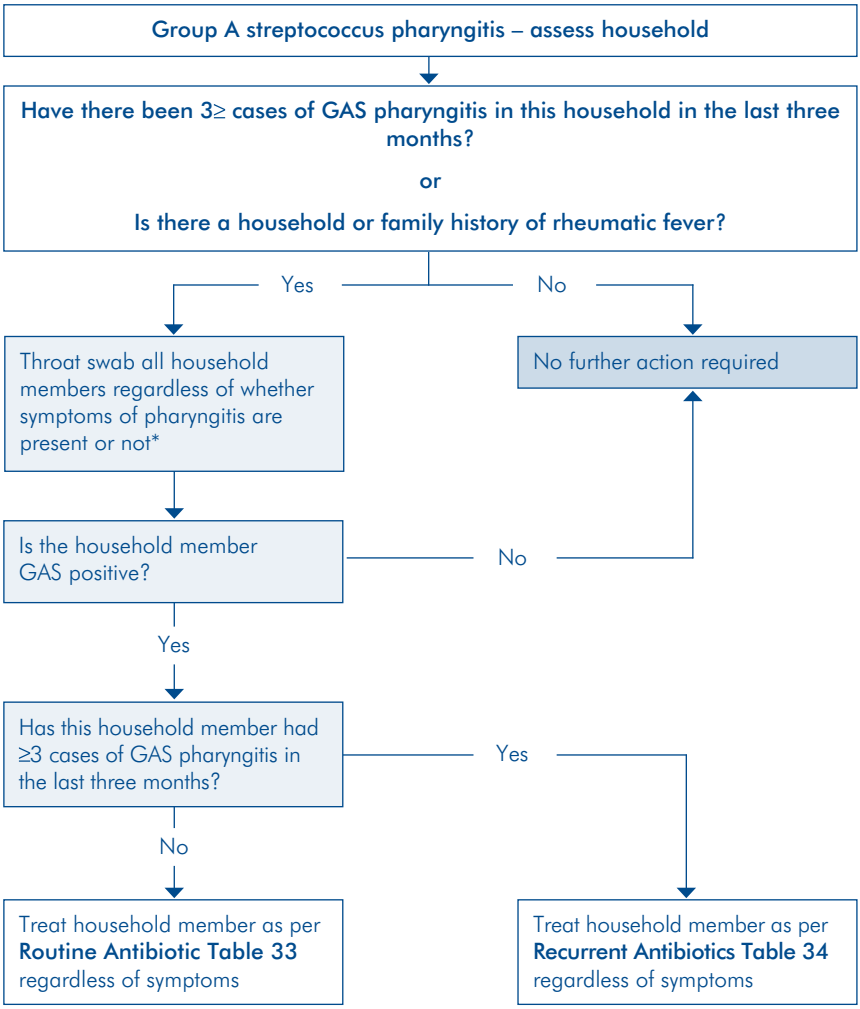
* **If patient is on benzathine penicillin IM prophylaxis for ARF, and is GAS positive on throat swab, treat in the following way:**

- if GAS positive in the first two weeks after IM penicillin injection, treat with a 10-day course of erythromycin (see Table 33)
- if GAS positive in the 3rd and 4th weeks after IM penicillin injection, treat with a 10-day course of oral penicillin (see Table 33).

Sources:

1. Centor RM, et al. Med Decis Making. 1981;1:239–246.
2. McIsaac WJ, et al. JAMA. 2004;291(13):1587–1595.

Figure 6 Guide for household sore throat management



* If impractical to swab, consider empiric antibiotic treatment

Source: based on the evidence-based best practice *New Zealand Guideline for Sore Throat Management* (Algorithm 4) (2006), produced by The National Heart Foundation of New Zealand and The Cardiac Society of Australia and New Zealand. www.nhf.org.nz

Table 33 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
Penicillin V Give as first choice 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
Erythromycin ethyl succinate (EES) Give if allergy to penicillin is reliably documented	PO	Children: 40 mg/kg/day in 2–4 divided doses Maximum 1 g/day Adults: 400 mg twice daily	10 days
Benzathine penicillin G (BPG) Give 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
Amoxycillin Useful alternative as can be given with food, may improve compliance	PO	Weight <30 kg: 750 mg once daily Weight >30 kg: 1500 mg once daily	10 days

PO = Orally
IM = Intramuscular

Table 34 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
Oral		
Clindamycin	Children: 20–30 mg/kg/day in 3 divided doses	10 days
	Adults: 600 mg/day in 2–4 divided doses	10 days
Amoxycillin; clavulanic acid	Children: 40 mg/kg/day in 3 divided doses*	10 days
	Adults: 500 mg twice daily	10 days
Parenteral with or without oral		
Benzathine penicillin G	For IM dosages, see Table 34†	1 dose
Benzathine penicillin G with rifampicin	For IM dosages, see Table 34†	4 days
	Rifampicin: 20 mg/kg/day orally in 2 divided doses	

* Maximum dose, 750mg of amoxycillin per day.

† 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.

Source: Bisno A, et al. Clin Infect Dis. 2002; 35:113–125. Adapted with permission.

Diagnosis of acute rheumatic fever

Diagnose ARF using diagnostic criteria (see Table 35 and Figure 5). 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.nhf.org.nz – Algorithm 2: Guide for the use of echocardiography in acute rheumatic fever).

Table 35 New Zealand guidelines for the diagnosis of acute rheumatic fever

	Diagnostic requirements	Category
Initial episode of ARF	2 major **or** 1 major and 2 minor criteria **plus** 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)[1]	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 **or** 1 major and 2 minor **or** several minor **plus** evidence of a preceding GAS infection (Jones, 1992)[2]	
Major criteria modified from Jones, 1992. (See guideline for further information on major criteria)	Carditis (including evidence of subclinical rheumatic valve disease on echo)* Polyarthritis† (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 Polyarthralgia† 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 polyarthritis 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.nhf.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 35), 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.nhf.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.

Prevention of infective endocarditis

A guideline for the *Prevention of Infective Endocarditis associated with Dental and Other Medical Interventions* was developed by the Heart Foundation in 2008. The following content forms part of this guideline. A full copy of the guideline will be available from www.nhf.org.nz. It should be noted that the new guidelines identify fewer procedures requiring prophylaxis and that there are changes to recommended prophylactic antibiotic regimens.

Cardiac conditions for prophylaxis

Table 36 Cardiac conditions for which endocarditis prophylaxis is recommended

- Prosthetic heart valves (bio- or mechanical)
- Rheumatic valvular heart disease
- Previous endocarditis
- Unrepaired cyanotic congenital heart disease (includes palliative shunts and conduits)
- Surgical or catheter repair of congenital heart disease within 6 months of repair procedure

Dental procedures for prophylaxis

Table 37 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 38), unless the procedure is at a site of established infection.

Table 38 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 39 Antibiotic regimen for dental procedures (plus tonsillectomy/adenoidectomy)

Amoxycillin 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 40 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 – amoxycillin (clindamycin or clarithromycin if penicillin allergy)
- gastrointestinal, hepatobiliary, genitourinary or obstetric/gynaecological infections – amoxycillin (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)

Heart failure

The guideline for the management of heart failure produced by the Heart Foundation in 2001 is being updated and will be finalised in 2009. The following algorithm is part of this guideline. A full copy of the guideline will be available from www.nhf.org.nz

Figure 7 Heart failure diagnostic algorithm

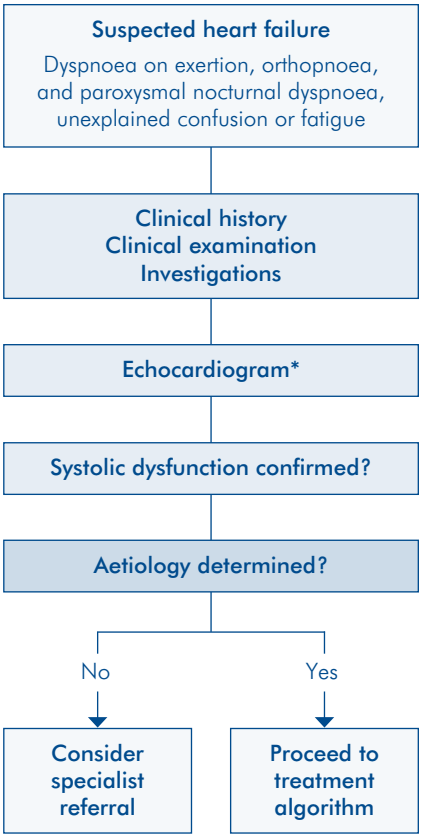


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Figure 7 continued...

Notes

Clinical history

- Age
- Onset of symptoms
- Previous heart disease
  - Myocardial infarction
  - Angina
  - Hypertension
  - Valvular disease, rheumatic fever
  - Palpitations
- Alcohol/tobacco use
- Medications
- Diabetes

Clinical examination

- Elevated JVP
- Third heat sound
- Pulse rate and rhythm
- Displaced apex beat
- Pulmonary rales
- Peripheral oedema
- Pulsus alternans
- Baseline weight

Aetiology

- Coronary artery disease
- Hypertension
- Valvular heart disease
- Endocrine disorders (eg, thyrotoxicosis)
- Myocarditis
- Idiopathic dilated cardiomyopathies
- Chronic arrhythmias (eg, rapid AF, complete heart block)
- Congenital heart disease
- Secondary to medications

Investigation	Suspected diagnosis
Full blood count	
• Anaemia	– CHF due to decreased oxygen carrying capacity
Serum creatinine	– Renal failure
Serum albumin	– Oedema due to hypoalbuminaemia
Plasma BNP	– Can rule out CHF
Thyroid function tests (indicated only with AF, age >65 years, evidence of thyroid disease)	
• Abnormal T4 or TSH	– Hypo/hyperthyroidism
Urinalysis	
• Proteinuria	– Nephrotic syndrome
• Red cells, casts	– Glomerulonephritis
Electrocardiogram	
• Acute ST/T wave changes	– Myocardial ischaemia
• Q waves	– Previous MI
• AF, other tachyarrhythmia	– Thyrotoxicosis, CHF due to rapid heart rate
• Bradyarrhythmias	– Hypothyroid CHF due to slow heart rate
• Left ventricular hypertrophy	– Diastolic dysfunction
Chest x-ray	
• Pulmonary congestion	– Heart failure
• Pulmonary disease	– CORD etc
Echocardiogram*	– Confirm systolic disfunction

Abbreviations:

AF = atrial fibrillation; MI = myocardial infarction; TSH = thyroid stimulating hormone

* Assessment of left ventricular function is an important part of the investigation. However, if this is delayed due to local resource constraints, then treatment should continue on an empirical basis.

Figure 8 Heart failure treatment algorithm

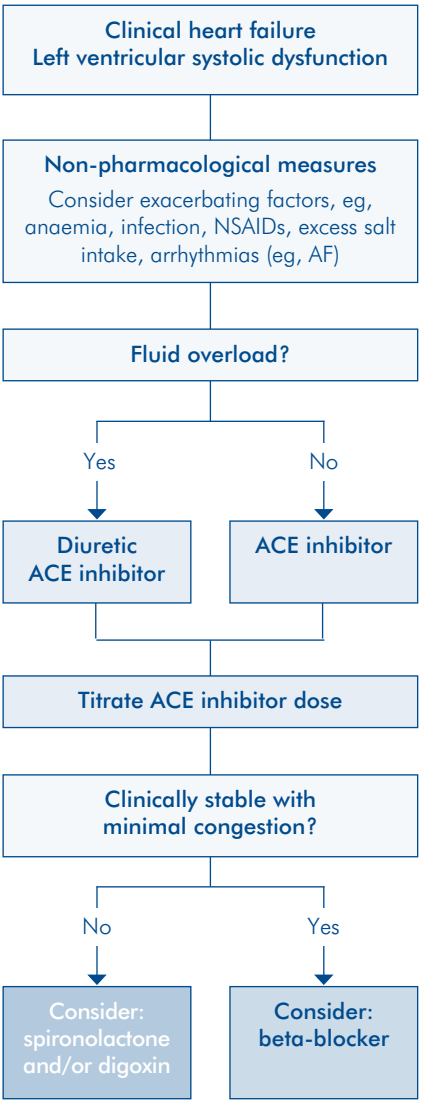

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Figure 8 continued...

Notes

Non-pharmacological management

- General counselling (including compliance, prognosis)
- Record weight daily (for diuretic titration)
- Avoid smoking
- Regular exercise
- Low-salt diet
- Limited alcohol

Diuretics

- Titrate according to symptoms and dry weight
- Mild CHF – thiazide alone may suffice (eg, bendrofluazide 2.5–5 mg daily)
- Moderate-severe CHF – loop diuretic (eg, initially frusemide 40 mg daily)
- Monitor K^+/creatinine weekly during titration, then 3 monthly
- K^+ supplementation usually not required with concomitant ACE inhibitor
- Serious hyperkalaemia can arise with combination of high-dose K^+-sparing diuretic and ACE inhibitors (see also spironolactone)
- In cases of resistant oedema, double the daily dose of diuretic, rather than give the same dose twice daily

ACE inhibitors

- Start at low dose (eg, captopril 6.25 mg tds, enalapril 2.5 mg daily)
- Titrate to target dose over 2–3 weeks (eg, captopril 25–50 mg tds, cilazapril 5 mg daily, enalapril 10 mg bd, quinapril 10–20 mg bid)
- Risk of first dose hypotension if SBP <90 mm Hg. Over-diuresis
- Consider lower doses if elderly or renal impairment
- Monitor K^+creatinine/BP weekly while titrating
- Contraindications: K^+ >5.5 mmol/L, creatinine >0.25 mmol/L, symptomatic hypotension or SBP <80 mm Hg, angioedema

Beta-blockers

- Consider for patients with chronic stable CHF and:
  - mild-moderate symptoms
  - minimal signs of congestion
  - stable for one month on adequate doses of ACE inhibitors and diuretics
- Contraindications: asthma 2nd/3rd degree heart block, symptomatic hypotension, SBP <80 mm Hg, HR <50 bpm
- Initiation and titration may require referral (see NHF CHF Doctors Guide)

Spironolactone

- Consider for patients with New York Heart Association class III/IV (moderate–severe) CHF symptoms
- Recommended dose =25 mg daily
- Hyperkalaemia/renal failure may arise if higher doses are used with ACE inhibitor
- Contraindications K^+>5 mmol/l, creatinine >0.25 mmol/l
- Monitor K^+/creatinine 3–4 days after starting
- 10% of males may suffer breast pain or gynaecomastia

Digoxin

- Consider for patients in AF or in sinus rhythm if CHF is severe and not controlled with ACE inhibitor and diuretic
- If normal renal function – start with 0.25 mg daily and check levels in 1 week
- If elderly or renal impairment – start at 0.125 or 0.0625 mg daily, check levels in 2–3 weeks
- Toxicity: confusion, anorexia, nausea, visual disturbance, arrhythmias
- Drugs which increase levels: antibiotics, amiodarone, diltiazem, verapamil, quinidine

Appendices

A: Genetic lipid abnormalities

B: Recommended method of blood pressure measurement

C: The New Zealand cardioprotective dietary pattern

D: Metabolic equivalents (METs) for selected activities

E: Land Transport NZ requirements

F: Process for updating the Handbook

Appendix A

Genetic lipid abnormalities

Genetic lipid disorders potentially putting people at a 5-year CVD risk >20%. Assume high risk clinically in this group	
Familial hypercholesterolaemia (FH)	**People presenting with cholesterol levels ≥8 mmol/L plus a family history of premature coronary heart disease, or tendon xanthelasma should be referred and offered family tracing** 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 Family tracing of the siblings and children of people with FH is recommended 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
Familial defective ApoB (FDB)	These people should be managed and referred as for people with FH
Familial combined dyslipidaemia (FCH)	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
Genetic lipid disorders potentially putting people at a 5-year CVD risk <20%. Calculate CVD risk in this group	
Low HDL-C syndromes	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 <0.7 mmol/L
High LP(a)	The genetic cause of high LP(a) is unknown. High values are refractory to most drug interventions
Isolated high triglycerides (≥8 mmol/L)	The management of people with isolated high triglycerides should be discussed with the appropriate specialist
Broad beta disease	If the TC:triglyceride ratio approaches one, with both lipid fractions elevated, then further investigation is needed

Appendix B

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 C

The New Zealand cardioprotective dietary pattern

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

Food	Healthy servings (per day)	Serving size examples	Notes
Low-fat or fat-free milk products	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
Fish, seafood	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
Peas, beans, soy products (legumes)	4–5 servings weekly	1 cup cooked dried beans, chickpeas, lentils, dahl ½ cup tofu or tempeh 1 glass fortified soy milk (250 ml)	
Skinned chicken or very lean meats	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
Eggs	3 eggs weekly	1 egg	

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The New Zealand cardioprotective dietary pattern continued...

Food	Healthy servings (per day)	Serving size examples	Notes
Liquid oils, unsaturated margarines and spreads or avocado	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.
Nuts, seeds	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 1 serving of nuts replaces other oils and spreads
Confectionery and added sugar	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
Minimise added salt	Limit high salt seasonings to 1/day	1 tsp seasoning paste ⅙ stock cube or ⅛ tsp stock powder ⅓ tsp gravy mix or 1 tbsp liquid seasoning	Use minimal salt in cooking Do not add salt to meals
Limit high salt foods	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 ½ 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
Alcoholic drinks	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	
Non-alcoholic drinks	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

* 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.

Source: *The Assessment and Management of Cardiovascular Risk,* 2003 available at www.nzgg.org.nz

Appendix D

Metabolic equivalents (METs) for selected activities*

Activity		METs (min)	METs (max)
METs for leisure activities			
Aerobics		6	9
Cycling	8 km per hour 16 km per hour 21 km per hour	2 5 8	3 6 9
Music	Playing an instrument	2.5	4
Dancing	Ballroom	4	5
Gardening	Mowing lawn (pushing) Weeding/cultivating	3 4	6 5
Running	General light jogging Training 10 km per hour	6 9	8 11
Skipping	<80/min	8	10
Swimming	Breast stroke Freestyle	8 9	9 10
Tennis		4	9
Walking	1–3 km per hour 3–6 km per hour	1 3	3 6
METs for activities of daily living			
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.

Source: *The Assessment and Management of Cardiovascular Risk*, 2003. www.nzgg.org.nz. Adapted from: Ainsworth BE. *The Compendium of Physical Activities Tracking Guide*. University of South Carolina; 2002.

Appendix E

Land Transport NZ requirements

The publication *Medical Aspects of Fitness to Drive: A Guide for Medical Practitioners* is available online at www.landtransport.govt.nz, search by publication title.

This guide assists medical practitioners in assessing the fitness to drive of any individual, including people with heart disease, diabetes, heart failure, severe hypertension, those on anticoagulation therapy and those with other cardiovascular conditions.

Appendix F

Process for updating the Handbook

The foremost recommendation from the Expert Advisory Group for the *Diabetes and Cardiovascular Disease Quality Improvement Plan* (Ministry of Health 2007) was to revise and update relevant NZGG guidelines on cardiovascular risk and type 2 diabetes together with the Cardiovascular Guidelines Handbook.

In the first instance, it was considered appropriate and expeditious to revise the content in the Handbook, which provides a convenient and useful summary of the guidelines and has been in particularly high demand in primary care. It was also well-recognised that guideline revision is an ongoing process and that there is a need to transition future guidelines to an electronic format to allow regular and efficient revision and dissemination in the light of new evidence.

For the 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 reference guidelines should follow.

The New Zealand Guidelines Group convened a Guideline Revision Team with wide stakeholder representation (see page 86 for a list of the Team members).

Content update

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 this edition of the handbook.

For smoking cessation, a summary of the recently revised *New Zealand Smoking Cessation Guidelines* (Ministry of Health 2007) replaced former content. New information from the 2008 guideline on TIA was also included (see the section on Stroke and Transient Ischaemic Attack).

Summaries of recommendations and algorithms related to rheumatic fever prevention, diagnosis and management, infective endocarditis prevention and heart failure diagnosis and management were included to provide a complete collection of cardiovascular guidelines for reference in primary care.

Summarised advice on cardiac rehabilitation, stroke management and the management of atrial fibrillation and flutter remains unchanged from the 2005 edition of the Handbook.

A list of source guidelines is provided at the front of the Handbook in the section 'About the 2009 Edition of the Handbook' (see page iii).

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List of tables and figures

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Cardiovascular risk factor management

Angina and myocardial infarction: long-term therapy

Atrial fibrillation and atrial flutter: assessment and therapy

Stroke and transient ischaemic attack

Rheumatic fever

Prevention of infective endocarditis

Abbreviations and acronyms

A2	Angiotensin II
ACE	Angiotensin converting enzyme
ACR	Albumin:creatinine ratio
AF	Atrial fibrillation
AFL	Atrial flutter
ApoB	Apolipoprotein B
BMI	Body mass index
BP	Blood pressure
bpm	Beats per minute
CABG	Coronary artery bypass graft
CHF	Chronic heart failure
CK	Creatine kinase
cm	Centimetres
COX2	Cyclooxygenase-2 inhibitor
CT	Computed tomography
CVD	Cardiovascular disease
CYP3A4	Cytochrome P4503A4
DBP	Diastolic blood pressure
DC	Direct current
DHA	Docosahexaenoic acid
dL	Decilitre
dsp	Dessert spoon
ECG	Electrocardiogram
ED	Emergency department
EPA	Eicosapentaenoic acid
FCH	Familial combined dyslipidaemia
FDB	Familial defective ApoB
FH	Familial hypercholesterolaemia
g	Gram
GCW	Gross combined weight
GFR	Glomerular filtration rate
GI	Glycaemic index
GLW	Gross laden weight
h	Hour
HbA1c	Haemoglobin type A1c
HDL	High density lipoprotein
HDL-C	High density lipoprotein cholesterol
HRT	Hormone replacement therapy
ICH	Intracranial haemorrhage
IFG	Impaired fasting glycaemia
IGT	Impaired glucose tolerance
INR	International normalised ratio
IV	Intravenous
J	Joules
kg	Kilogram
LDL	Low density lipoprotein

LDL-C	Low density lipoprotein cholesterol
LP(a)	Lipoprotein (a)
LV	Left ventricular ejection fraction
METs	Metabolic equivalents
mg	Milligram
MI	Myocardial infarction
ml	Millilitre
mm Hg	Millimetres of mercury
mmol/L	Millimole per litre
NNT	Number needed to treat
NRT	Nicotine replacement therapy
NSAID	Non-steroidal anti-inflammatory agents
OGTT	Oral glucose tolerance test
PCI	Percutaneous coronary intervention
PVD	Peripheral vascular disease
SBP	Systolic blood pressure

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