

*Evidence-based
Best Practice
Guidelines*

JUNE 2005

New Zealand *Cardiovascular Guidelines Handbook*

Developed for Primary Care Practitioners

CD Rom included



Supported by Diabetes New Zealand

*Evidence-based
Best Practice
Guidelines*

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New Zealand *Cardiovascular Guidelines Handbook*

Developed for Primary Care Practitioners

Risk Assessment
Atrial Fibrillation
Heart Disease
Stroke
Diabetes
Smoking Cessation



www.nzgg.org.nz

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STATEMENT OF INTENT

Evidence-based best practice guidelines are produced to help health practitioners, patients and consumers make shared decisions about health care choices in specific clinical circumstances. If properly developed, communicated and implemented, guidelines can improve care. While they represent a statement of best practice based on the latest available evidence (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.

The clinical expertise (including skills and experience) of the practitioner is the key to integrating these elements to achieve the best possible outcome for an individual.

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Where guidelines are modified for local circumstances, significant departures from these national guidelines should be fully documented and the reasons for the differences explicitly detailed.

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USING THIS HANDBOOK

New Zealand Cardiovascular Guidelines Handbook: Developed for Primary Care Practitioners is a condensed version of the advice contained in six recent guidelines:

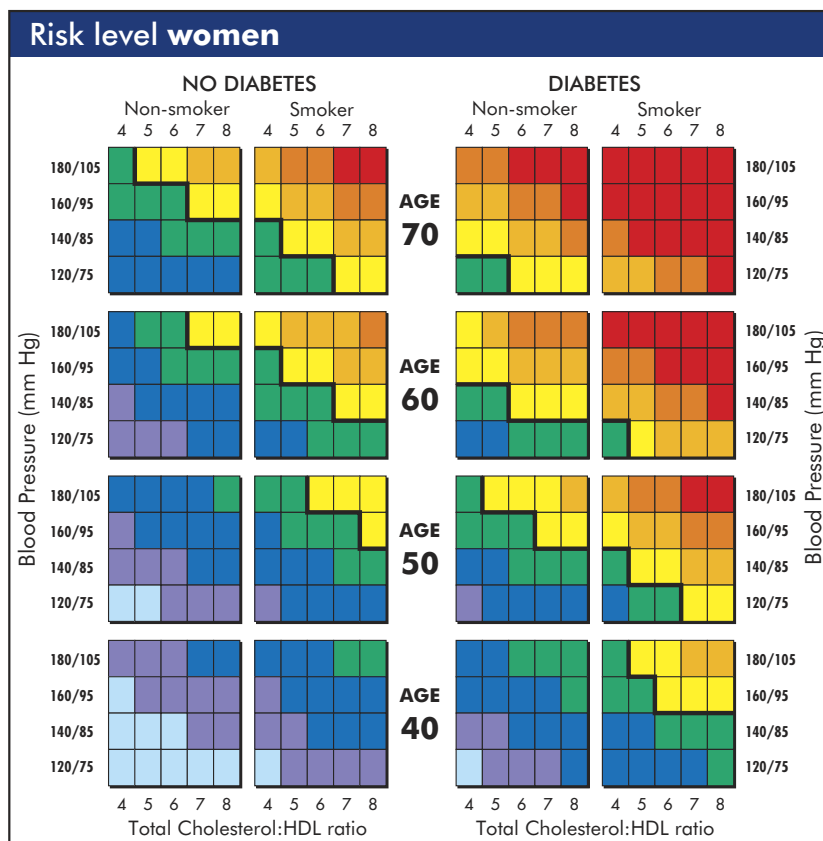
- *The Assessment and Management of Cardiovascular Risk*
- *Management of Type 2 Diabetes*
- *Life after Stroke: New Zealand Guideline for the Management of Stroke*
- *The Management of People with Atrial Fibrillation and Flutter*
- *Cardiac Rehabilitation*
- *Guidelines for Smoking Cessation.*

These guidelines were produced by more than 90 representatives from health professional, Māori, Pacific and consumer groups. A description of the guideline development processes and grading systems used by the guideline teams is contained in the full guidelines, which are available at www.nzgg.org.nz. This handbook is not intended to replace the health professional's judgment in each individual case. It offers a practical reference for primary care practitioners.

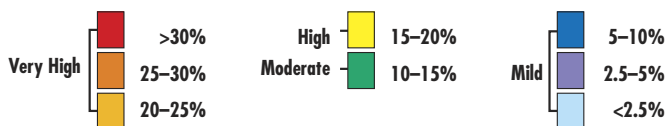
This is necessarily a brief synopsis of essential and hard-to-remember information. A complete description of management advice is contained in the full guidelines.

An electronic copy of the full guidelines this handbook is based on are available for download from www.nzgg.org.nz, or a printed copy is available from info@nzgg.org.nz, phone 64-4-471 4180 or write to PO Box 10 665, Wellington, New Zealand.

Figure 1: New Zealand cardiovascular risk charts



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

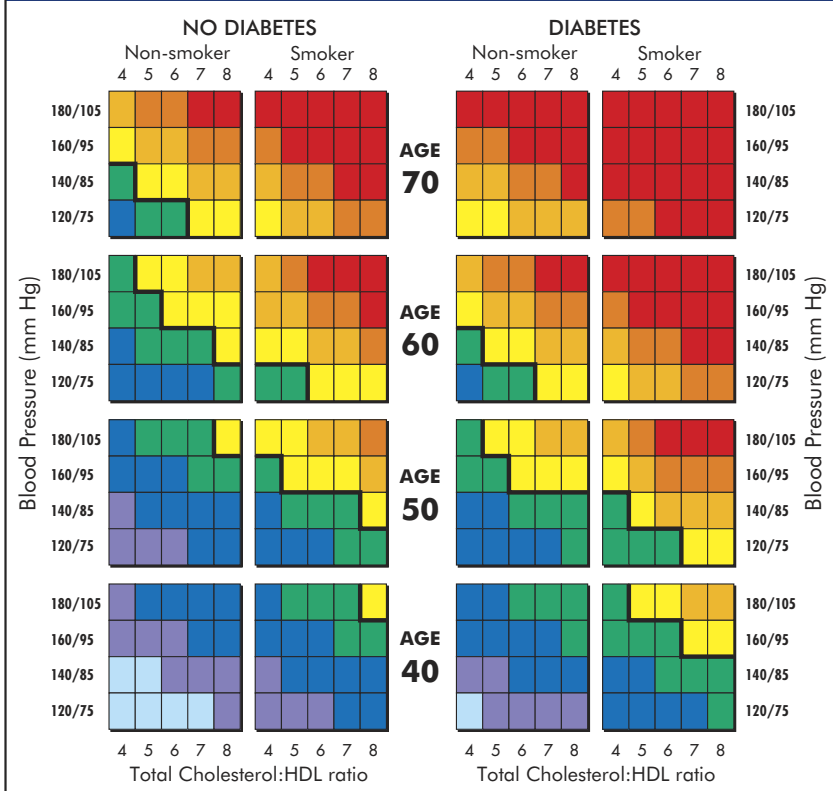


How to use the Charts

- 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, blood pressure (BP) and total cholesterol (TC) TC:HDL ratio. When the systolic and diastolic values fall in different risk levels, the higher category applies.
- For example, the lower left cell contains all non-smokers without diabetes who are less than 45 years and have a TC:HDL ratio of less than 4.5 and a BP of less than 130/80 mm Hg. People who fall exactly on a threshold between cells are placed in the cell indicating higher risk.

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

Risk level men



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)

Based on the conservative estimate that each intervention: aspirin, BP treatment (lowering systolic BP 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.

NNT = Number needed to treat

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			
	No Diabetes		Diabetes		No Diabetes		Diabetes	
Systolic Blood Pressure (mm Hg)	180	13	AGE ≥75	22	180	23	AGE ≥75	37
	160	11		19	160	20		34
	140	10		17	140	18		31
	120	9		15	120	16		28
Systolic Blood Pressure (mm Hg)	180	10	AGE 65–74	17	180	18	AGE 65–74	29
	160	9		15	160	16		27
	140	8		13	140	14		24
	120	7		12	120	13		21
Systolic Blood Pressure (mm Hg)	180	7	AGE <65	13	180	13	AGE <65	22
	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% or HIGH 15–19%	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
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 tables

- Identify the table relating to the person’s age, sex and diabetic status
- Within the table 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.

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CARDIOVASCULAR RISK ASSESSMENT

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

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

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

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

NEW ZEALAND CARDIOVASCULAR RISK CHARTS

See Figure 1 (pages iv–v) for New Zealand Cardiovascular Risk Charts.

- 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.

Table 1: The age to start cardiovascular risk assessment

Group	Men	Women
Asymptomatic people without known risk factors	Age 45 years	Age 55 years
Māori, Pacific and Indian subcontinent peoples*	Age 35 years	Age 45 years
People with other known cardiovascular risk factors or at high risk of developing diabetes Family history risk factors <ul style="list-style-type: none"> • 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 <ul style="list-style-type: none"> • Gestational diabetes, polycystic ovary syndrome, current or recent smoking • Prior blood pressure (BP) $\geq 160/95$ 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) 	Age 35 years	Age 45 years
People with diabetes	Annually from the time of diagnosis	

* Indian subcontinent peoples = Indian, including Fijian Indian, Sri Lankan, Afghani, Bangladeshi, Nepalese, Pakistani, Tibetan.

Table 2: Estimating 5-year cardiovascular risk: when to use the New Zealand cardiovascular risk charts

Risk Group	Estimating Risk
<p>Very high risk groups: 5-year risk assumed clinically >20%</p>	<p>These people do not need their risk assessed using the New Zealand cardiovascular risk charts.</p> <ul style="list-style-type: none"> • Previous CVD event: angina, MI, angioplasty, 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
<p>Isolated elevated single risk factors: 5-year risk of >15%</p>	<p>Calculate 5-year risk using the New Zealand cardiovascular risk charts. When all risk factors are taken into account, the risk may be even higher than the assumed 5-year CVD risk of >15%.</p> <ul style="list-style-type: none"> • TC ≥ 8 mmol/L • TC:HDL ratio ≥ 8 • BP consistently $\geq 170/100$
<p>People aged 35–74 years: calculate the 5-year CVD risk</p>	<p>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)</p> <p>These groups should be moved up one risk category (5%)*</p> <ul style="list-style-type: none"> • 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 peoples from the Indian subcontinent • Diabetes with microalbuminuria • Type 2 diabetes for ≥ 10 years • Type 2 diabetes with HbA1c consistently $\geq 8\%$ • The metabolic syndrome (Table 10) <p>* Make the 5% adjustment once only for people with >1 criterion</p>

Continued over...

Risk Group	Estimating Risk
<p>People aged <35 years</p>	<p>All calculations outside the age ranges of the Framingham equation are approximations, but can be useful.</p> <p>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</p> <ul style="list-style-type: none"> • Low HDL <0.7 mmol/L, or high HDL ≥2.0 mmol/L (because of the risk of a genetic lipid disorder – see Chapter 9 of the full guideline: <i>The Assessment and Management of Cardiovascular Risk</i>) • 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)
<p>People aged ≥75 years</p>	<p>Aged over 75 years: calculate the risk as if they were 70 years</p> <p>An assessment of the balance between the risks and benefits of treatment is more difficult in older people 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</p> <p>A clinical judgment should take into account:</p> <ul style="list-style-type: none"> • likely benefits and risks of treatment • life expectancy and comorbidities • personal values
<p>People with diabetes aged 20–34 years</p>	<p>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</p>

Table 3: What to measure and record for cardiovascular risk assessment

Everyone	History	Age Gender Ethnicity
	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 (Appendix A)
	Past medical history	Smoking history (if stopped smoking for <12 months, assess as a smoker) Past history of CVD (MI, angioplasty, percutaneous transluminal coronary angioplasty [PTCA], stent, CABG, angina, ischaemic stroke, TIA, peripheral vascular disease [PVD]) Genetic lipid disorder (FH, FDB, FCH: Appendix A)
	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) Serum creatinine and history of renal disease
Atrial fibrillation (AF), confirmed on electrocardiogram (ECG)	History and examination	Echocardiogram (where possible) Past history of stroke, TIA, heart failure, rheumatic or mitral valve disease <i>See AF section for calculating the risk of stroke in people with AF</i>



 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 5–15%	Further risk assessment in 5 years
5-year risk >15%, diabetes, or on lipid or BP-lowering medication	Annual risk assessment
People with diabetes, metabolic syndrome or receiving medication 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.

Two lipid measurements should be taken prior to initiating drug treatment or intensive lifestyle treatment. If the total cholesterol level varies 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 diabetes, obesity, insulin resistance, liver disorders, thyroid disorders, some haematological disorders and renal disease.

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. A rise in cholesterol is normal in pregnancy and a cholesterol level should not be measured at this time.

Genetic Lipid Disorders

Consider the possibility of a genetic lipid disorder if TC \geq 8 mmol/L or if there is a family history of premature coronary heart disease. See Appendix 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, liquorice intake and use of sympathomimetics.

Rarer causes that require further investigation 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.

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 oral glucose tolerance test (OGTT)
6.1 to 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 to 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	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.

[†] Non-European ethnicity, first-degree relative with diabetes, past history of gestational diabetes or the metabolic syndrome.

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

MEASURES OF WEIGHT AND TRUNCAL OBESITY

- Measure weight, height, waist circumference and calculate body mass index (BMI) in kg/m² using Tables 8 or 9.
- For people with a BMI ≥35, an initial goal of 10% weight loss may be more appropriate than a target in the healthy weight band.

Table 8: Classification of weight in non-Māori, non-Pacific adults

		BODY MASS INDEX																	
		20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37
		WEIGHT IN KILOGRAMS																	
HEIGHT IN METRES	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					

Table 9: Classification of weight in Māori and Pacific adults

		BODY MASS INDEX																	
		20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37
		WEIGHT IN KILOGRAMS																	
HEIGHT IN METRES	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

- 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.
- Record waist circumference midway between the lower rib margin and the iliac crest to the nearest centimetre.

THE METABOLIC SYNDROME (INSULIN RESISTANCE SYNDROME)



-  This cluster of clinical features identifies individuals at increased risk of many diseases despite only moderate elevations of individual risk factors (ischaemic heart disease, subfertility, diabetes, gout and heart failure).
-  Three or more of five risk factors are required for diagnosis (see Table 10).

Table 10: The recommended definition of the metabolic syndrome

Risk Factor	Sex	Defining level
1. Abdominal Obesity	Men	≥100 cm waist circumference
	Women	≥90 cm waist circumference
2. Fasting Triglycerides		≥1.7 mmol/L
3. HDL Cholesterol	Men	<1.0 mmol/L
	Women	<1.3 mmol/L
4. Blood Pressure		SBP ≥130 or DBP ≥85
5. Fasting Glucose		≥6.1 mmol/L

Adapted from the National Cholesterol Education Program ATP III 2001 definition (units rounded). Levels are an approximate guide only.

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.

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 11).
- 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 11: 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.

Risk goals can be more easily achieved by the simultaneous reduction in several risk factors (see Table 15).

🔑 Risk factors can be used as targets for people at high risk (see Table 12).

Table 12: Optimal levels (targets for people with known cardiovascular disease or diabetes)

	Known cardiovascular disease (stroke TIA, PVD, angina or after MI)	Diabetes	Diabetes and overt nephropathy, microalbuminuria or other renal disease
Lipids			
Total cholesterol	<4 mmol/L*		
LDL cholesterol	<2.5 mmol/L*		
HDL cholesterol	≥1 mmol/L		
TC/HDL ratio	<4.5		
Triglycerides	<1.7 mmol/L		
Blood pressure			
BP	<130/80 mm Hg	<130/80 mm Hg	Aggressive BP control is recommended
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%)

* Lower lipid targets are appropriate for people after CABG (TC <3.5 mmol/L or LDL-C <2.0 mmol/L).

🔑 Graded lifestyle advice is appropriate for everyone (see Table 13).

Table 13: Recommended lifestyle interventions (diet, physical activity, weight management and smoking cessation) based on cardiovascular risk assessment

Intervention	5-year CVD Risk
Intensive lifestyle interventions	<ul style="list-style-type: none">• Calculated >20%• Cardiovascular disease• Genetic lipid disorders• Diabetes• Metabolic syndrome
Specific lifestyle interventions	<ul style="list-style-type: none">• Calculated 10–20%
General lifestyle advice	<ul style="list-style-type: none">• Calculated <10%

🔑 Drug therapy is indicated for people with CVD risk >15% (see Table 14).

Table 14: Recommended drug interventions based on cardiovascular risk assessment

Intervention	5-year CVD Risk
Start low dose aspirin, unless contraindicated, and other drugs as appropriate to the condition (Tables 35, 39 or 40)	<ul style="list-style-type: none">• Clinically >20%
Start low dose aspirin, unless contraindicated, lipid modification and BP lowering simultaneously with intensive lifestyle advice	<ul style="list-style-type: none">• Calculated >20%
Start drug therapy after 3–6 months of lifestyle advice (if the calculated CVD risk is still >15%)	<ul style="list-style-type: none">• Calculated 15–20%
Start drug therapy for elevated isolated risk factors (TC \geq 8 mmol/L or TC:HDL ratio \geq 8 or BP \geq 170/100)	<ul style="list-style-type: none">• CVD risk >15%

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

Table 15: 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 more than 20%*	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, physical activity and smoking cessation interventions. 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)	Efforts should be made to reach optimal risk factor levels	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
CVD risk calculated more than 20%	Intensive lifestyle advice on a cardioprotective dietary pattern with a dietitian, physical activity and smoking cessation interventions. Lifestyle advice should be given simultaneously with drug treatment	Aspirin and drug treatment of all modifiable risk factors (BP lowering, lipid modification and glycaemic control)	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
15 to 20%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation. This lifestyle advice should be given by the primary health care team for 3 to 6 months prior to initiating drug treatment	Aspirin and drug treatment of all modifiable risk factors (BP lowering, lipid modification and glycaemic control). Drug therapy indicated for people with extreme risk factor levels [†]	Risk factors treated to a level that will lower 5-year cardiovascular risk to less than 15% (by recalculating risk)	Cardiovascular risk assessments at least annually, risk factor monitoring every 3 to 6 months
10 to 15%	Specific individualised lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation. This lifestyle advice should be given by the primary health care team	Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further cardiovascular risk assessment in 5 years
less than 10%	General lifestyle advice on a cardioprotective dietary pattern, physical activity and smoking cessation	Non-pharmacological approach to treating multiple risk factors	Lifestyle advice aimed at reducing cardiovascular risk	Further cardiovascular risk assessment in 5 to 10 years

* People who have had a previous cardiovascular event (angina, MI, angioplasty, coronary artery bypass grafts, 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.

[†] People with isolated high risk-factor levels either total cholesterol ≥ 8 mmol/L or TC:HDL ratio ≥ 8 or BP $\geq 170/100$ mm Hg should have these risk factors treated and their risk calculated.

GENERAL LIFESTYLE INTERVENTIONS


 Offer everyone advice promoting 'healthy heart' foods and a smoke-free, active lifestyle (see Table 16).

Table 16: General lifestyle advice for people at 5-year cardiovascular risk <10%

The National Heart Foundation of New Zealand Food-based Dietary Statements: a guide to healthy eating	
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
5	Choose low-fat milk and 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 – www.sparc.org.nz
Healthy weight	BMI <25 (<26 for Māori or Pacific peoples) Waist circumference <100 cm in men or <90 cm in women
Smoking cessation	Smoking cessation has major and immediate health benefits for smokers of all ages and their families QUITLINE – 0800 778 778, www.quit.org.nz

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.
- 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 17) at 3 to 6 METs intensity (see Appendix D) are meeting the minimum requirement for health. More intense activity for longer is preferred.
- 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, physical activity habits or to stop smoking. 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 18 and 19).

Table 17: 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 18: Specific lifestyle and behavioural risk-factor management for people at 5-year cardiovascular risk of 10 to 20%

Risk factor	Assessment and advice
Nutrition	<ul style="list-style-type: none"> Assess general dietary habits against the National Heart Foundation of New Zealand food-based dietary statements (Table 16) Complete a lifestyle assessment diary Quantify intake and offer advice on the cardioprotective dietary pattern table (Appendix C)
Physical activity	<ul style="list-style-type: none"> Assess the current level of physical activity; duration and frequency (Table 17), intensity and type (Appendix D). The 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	<ul style="list-style-type: none"> 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, insulin resistance and BP)

Continued...

Smoking	<ul style="list-style-type: none"> • Strongly encourage person and family to stop smoking • Document current smoking status/second-hand smoke exposure prominently in medical record • Determine if smokers are ready to quit (20–25% of smokers) • Brief, repetitive, consistent, positive advice to quit from multiple providers (or reinforcement of a recent quit attempt) doubles success rates • Offer self-help material • Help in setting a quit date (ideally within 2 weeks) and quit plan • Explore barriers to successful cessation • Refer to organised cessation support (eg, QUITLINE) • Encourage nicotine replacement therapy (NRT) as first-line pharmacotherapy • Arrange follow-up (in person or by phone) with smokers who are ready to quit: within the week, then again within the first month • Reinforce staying 'quit' during visits in first year post-cessation
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Table 19: Specific lifestyle changes to modify biomedical risk factors

Risk factor	Assessment and advice
Lipid modification	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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 Metabolic syndrome	<ul style="list-style-type: none"> • Intensive lifestyle advice for people with disorders of carbohydrate metabolism should be given in individual/group sessions with a dietitian. See <i>Management of Type 2 Diabetes</i> full 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 20).
- Intensive intervention usually requires referral; it assumes a quantitative assessment by a health professional specifically trained in the lifestyle area with arranged follow-up over a period of time. Intensive dietary advice should be given in individual or group sessions with a dietitian.

Table 20: Intensive lifestyle advice and referral guidelines for some high-risk groups

MI, angina, after CABG, angioplasty or coronary stent
<ul style="list-style-type: none"> • 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 cardiovascular disease 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
<ul style="list-style-type: none"> • Refer to organised stroke services
Diabetes
<ul style="list-style-type: none"> • Refer to a dietitian and diabetes nurse specialist
Genetic lipid disorders
<ul style="list-style-type: none"> • Refer to a specialist clinic for family tracing
Metabolic syndrome
<ul style="list-style-type: none"> • Refer to a dietitian

The same specific lifestyle advice (see Table 18) is delivered using advanced techniques in more frequent sessions of longer duration.

SMOKING CESSATION INTERVENTIONS

- Smoking cessation has major and immediate health benefits for all smokers. Approach smoking cessation advice using the 5 A's (see Table 21)
- Nicotine replacement therapy (NRT) is first-line pharmacotherapy (see Table 22).
- Bupropion or nortriptyline hydrochloride are second-line agents (see Table 23).

Table 21: The 5 A's of smoking cessation

Ask	<ul style="list-style-type: none"> • Document current smoking status and second-hand smoke exposure
Assess	<ul style="list-style-type: none"> • Determine if smokers are ready to quit (20–25% of smokers)
Advise	<ul style="list-style-type: none"> • Brief, repetitive, consistent, positive advice from multiple providers (or reinforcement of a recent quit attempt) doubles success rates • Sudden cessation versus gradual reduction has similar efficacy
Assist	<ul style="list-style-type: none"> • Offer self-help material • Help to set a quit date (ideally within 2 weeks) and quit plan • Provide practical counselling and support • Explore barriers to successful cessation • Offer referral to organised cessation support (eg, QUITLINE) • Encourage NRT as first-line pharmacotherapy
Arrange (Follow-up)	<p>Arrange follow-up (in person or by phone) with smokers who are ready to quit:</p> <ul style="list-style-type: none"> • first follow-up within the first week • second follow-up within the first month • reinforce staying 'quit' during visits in first year post-cessation

🔑 Consider NRT in smokers who smoke >10 cigarettes/day, whether they are motivated, understand the risks/benefits, and agree to 100% cessation, quit date and follow-up.

Table 22: Nicotine replacement therapy

Patches	<ul style="list-style-type: none"> • 16-hour patches are as effective as 24-hour patches • Doses >22 mg/24-hour are not more effective than lower doses • Treatment >8 weeks does not increase efficacy • Tapered (weaning) therapy is not better than abrupt withdrawal • Over-the-counter and subsidised (on presentation of a exchange card) • Consider addition of 'as required' gum/inhaler/nasal spray if refractory to monotherapy with NRT patch
Gum	<ul style="list-style-type: none"> • Over-the-counter and subsidised (on presentation of an exchange card)
Inhalers and nasal sprays	<ul style="list-style-type: none"> • Not subsidised
Exchange card programme	<ul style="list-style-type: none"> • Heavy smokers (≥10 cigarettes a day) are eligible for a subsidised 8-week course of NRT patches or gum • Available through QUITLINE (0800 778778) or eligible health providers registered to provide cards (contact quit@quit.org.nz) • Cost: \$5 – first 4 weeks, \$10 – second 4 weeks

Arrange follow up within 3 to 5 days, and then further follow-up to increase likelihood of success.

AVOID smoking while on NRT (avoids overdose symptoms, smoking while on NRT markedly decreases likelihood of quitting).

Table 23: Second-line smoking cessation therapies

Bupropion	<ul style="list-style-type: none"> • Quit rates at 1 year: increased ~1.3 to 4-fold compared with placebo (NNT = 6–10) • Insufficient evidence to recommend NRT/bupropion combination • Seizure risk as low as 1:5000 (if smokers with contraindications have been stringently excluded)
Nortriptyline	<ul style="list-style-type: none"> • Good evidence for effectiveness

🔑 Current evidence shows NRT to be safe in people with heart disease (see Table 24).

Table 24: Heart disease and smoking cessation therapies

NRT	<ul style="list-style-type: none"> • Start at a lower dose and increase dose if withdrawal symptoms occur • Use NRT cautiously (after discussion with a specialist) in the immediate post-MI period (4 weeks) and in those with serious arrhythmias, or severe or worsening angina • Follow-up patients closely
Bupropion	<ul style="list-style-type: none"> • Suitable treatment, if appropriate
Nortriptyline	<ul style="list-style-type: none"> • Contraindicated in acute recovery phase after MI

🔑 Quitting at any point in pregnancy can yield benefits for the foetus and mother (see Table 25).

Table 25: Smoking cessation in pregnancy and breast feeding

NRT in pregnancy	<ul style="list-style-type: none"> • Manufacturers do not recommend NRT; however, patches and gum are safer than smoking • Consider patches or gum if ≥ 15 cigarettes/day, motivation to quit, previous unsuccessful attempts to quit without NRT • Provide close follow-up and monitoring for symptoms of under or over-dosage, refer to QUITLINE
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Nicotine is also present in the breast milk of smokers.

• Minor weight gain is common when people stop smoking (see Table 26).

Table 26: Smoking cessation and weight gain

Weight gain	<ul style="list-style-type: none">• Weight gain is common, usually <4.5 kg, with up to 10% gaining as much as 13.5 kg• Tolerate moderate weight gain over the first 3 months and work on losing weight later on• For smokers concerned with weight gain, consider bupropion or NRT (in particular, gum, which has been shown to delay weight gain after quitting)
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TE HOTU MANAWA MĀORI – 09-638 5800, www.tehotumanawa.org.nz

Te Hotu Manawa Māori provide Aukati Kai Paipa training, which is by Māori, for Māori, one-on-one support to assist people to cease smoking.

QUITLINE – 0800 778 778, www.quit.org.nz

Free and confidential support, subsidised NRT.

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 27).

Table 27: 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 anti-oxidant 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 cardiovascular disease: herbal medicines, botanicals, garlic/ginkgo biloba/rosemary/horse-chestnut seeds/xin bao, acupuncture, chelation, oriental medicine, aromatherapy, homeopathy, hypnosis, meditation, yoga, tai chi, intercessory prayer, Strauss heart drops.

LIPID MODIFICATION

- 🔑 Lipid levels (TC from about 4 to 8 mmol/L) in people without CVD should be interpreted in the context of their cardiovascular risk (see Table 28). There is no normal or ideal lipid level. Risk factors can be viewed as targets for people at high risk (see Table 29).
- 🔑 People aged ≥ 75 years should be treated in the same way as younger people.

Table 28: Clinical scenario and intervention recommendations

TC 4–8 mmol/L	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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, aspirin, lipid-modifying therapy, +/- BP-lowering therapy
CVD risk $>20\%$ clinically*	<ul style="list-style-type: none"> • Commence statin (unless contraindicated) simultaneously with intensive lifestyle advice, aspirin and other appropriate medication
Calculated CVD risk $>20\%$	<ul style="list-style-type: none"> • Aim is to reduce 5-year CVD risk to $<15\%$. This can be more easily achieved by reduction of all modifiable risk factors • Commence intensive lifestyle advice, simultaneously with aspirin and drug treatment of all modifiable risk factors
Calculated CVD risk 15–20%	<ul style="list-style-type: none"> • Aim is to reduce 5-year CVD risk to $<15\%$. This can be more easily achieved by simultaneous reduction of all modifiable risk factors • Commence specific individualised lifestyle advice for 3–6 months before considering aspirin, lipid-modifying or BP-lowering therapy
Calculated CVD risk 10–15%	<ul style="list-style-type: none"> • Specific individualised lifestyle advice, including dietary advice on a cardioprotective diet, physical activity and smoking cessation advice
Calculated CVD risk $<10\%$	<ul style="list-style-type: none"> • General lifestyle advice, including dietary advice on a cardioprotective diet, physical activity and smoking cessation advice

* Previous CVD event (angina, MI, angioplasty, 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 29: Optimal lipid levels (targets) for people with cardiovascular disease or diabetes

	Known cardiovascular disease (stroke TIA, PVD, angina or after MI)	Diabetes	Diabetes and overt nephropathy, microalbuminuria or other renal disease
Total cholesterol	<4 mmol/L*		
LDL cholesterol	<2.5 mmol/L*		
HDL cholesterol	≥1 mmol/L		
TC/HDL ratio	<4.5		
Triglycerides	<1.7 mmol/L		

* Lower lipid targets are appropriate for people after CABG (TC <3.5 mmol/L or LDL-C <2.0 mmol/L).

Lipid Monitoring

Use LDL-C as the primary indicator of optimum lipid management and to monitor lipid-modifying treatment. HDL-C is a secondary indicator.

If on lipid-lowering drug treatment, monitor lipids every 3 months until levels are controlled, then every 6 months.

Starting Doses for Statins

- 🔑 For people with 5-year CVD risk 15–20%, start simvastatin 20 mg (or equivalent), and titrate if needed.
- 🔑 For people with known CVD or 5-year CVD risk >20%, a starting dose of 40 mg simvastatin (or equivalent) is an alternative, particularly if LDL is high (see Table 30).

Table 30: Doses of various statins required to reach a target*

	TC mmol/L	LDL-C mmol/L	% change needed	Fluvastatin [†] (mg/day)	Pravastatin [†] (mg/day)	Simvastatin (mg/day)	Atorvastatin (mg/day)	Rosuvastatin [†] (mg/day)		
Lipid profiles of subjects	4.5	3	15	20	10	5	2.5			
	5	3.5	25	40	20	10	5	1		
	5.5	4	35	80	40	20	10	2.5		
	6	4.5	45		80	40	20	5		
	6.5	5	50	Combination therapy needed					40	10
	7.5	6	60	Combination therapy needed					80	20

Doubling the statin dose gives a further 6–10% reduction of LDL

* Average daily doses (mg) of various statins needed to achieve a LDL-C target <2.6 mmol/L (100 mg/dL) in at least 50% of subjects with various baseline cholesterol and LDL-C levels (and normal range HDL-C and triglycerides).

† At the time of publication, rosuvastatin and pravastatin are not subsidised in New Zealand and fluvastatin is not available.

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Choice of Lipid Modifying Drugs

🔑 The lipid profile will determine the drug treatment selected to lower lipid levels (see Table 31).

Predominant hypercholesterolaemia

Statins are first-line therapy to lower total cholesterol or LDL-C when the triglyceride level is normal or only slightly elevated.

Low HDL-C on statin therapy

When HDL-C is <0.9 mmol/L and the person is on statin therapy, consider treatments that augment LDL-C and triglyceride lowering actions and raise HDL-C. Such people may need specialist review and those with very low HDL-C levels (<0.7 mmol/L) should be referred.

Predominant hypertriglyceridaemia and low HDL-C

Fibrates: should be used in people with predominant hyper-triglyceridaemia (≥ 3 mmol/L) and low HDL-C with a normal to slightly elevated LDL-C level (< 3 mmol/L), either as monotherapy or in combination with a statin).

Fish oil: if fibrate treatment is not tolerated or if additional triglyceride lowering effect is required, fish oil can be used with close monitoring of glycaemic control.

Acipimox: often useful in those not responding to fibrate agents.

Nicotinic acid: can be used, but will often require specialist review.

Statins: not usually useful if triglycerides are markedly elevated (≥ 5 mmol/L).

Combined dyslipidaemia

Consider treatment with a statin and a fibrate in people with moderate to marked elevation of both LDL-C and triglycerides. Because of the increased risk of myopathy with combinations (particularly those that include gemfibrozil), special care should be taken to fully inform and monitor people on combination treatments.

Table 31: The effect of various drug classes and plant sterols on lipid profiles

		Cholesterol	LDL Cholesterol	Triglycerides	HDL	IDL
Statins*	simvastatin atorvastatin 20–40 mg/day	↓↓↓	↓↓↓	↓→	→↑	Variable
Fibrates	gemfibrozil 1200 mg/day bezafibrate 600 mg/day or bezafibrate retard 400 mg/day	↓	↓→	↓↓	↑	↓↓
	ezetimibe 10 mg/day	↓↓	↓↓	↓	↓→	
Niacin	> 1.5 g/day	↓↓↓	↓↓	↓↓	↑	↓↓
Acipimox	750–1000 mg/day	↓	↓	↓	↑	?
Plant sterols	20 g/day of product	↓	↓	↓	↑	?

KEY	0	< 5%	5–10%	10–20%	20–30%	30+%
% change	→	↓→	↓	↓↓	↓↓↓	↓↓↓↓

* Statins on a mg dose basis: Atorvastatin > Simvastatin > Fluvastatin.

Statins are not usually useful when triglycerides are markedly elevated (≥ 5.0 mmol/L).

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Statin Monitoring

- 🔑 Perform alanine transaminase (ALT) at baseline and a creatine kinase (CK) for unexplained muscle symptoms (see Table 32).
- 🔑 Serious side effects are very rare and include hepatotoxicity, myopathy and rhabdomyolysis

Table 32: Statin monitoring

Blood test	Recommended action	
ALT	<ul style="list-style-type: none"> • Perform an ALT as a baseline prior to commencing a statin • Repeat an ALT at first follow-up, and thereafter if indicated • ALT rise <3 times normal: usually possible to continue with statin • ALT rise ≥3 times normal: increase monitoring, consider discussion with specialist 	
Perform CK for unexplained muscle pain, tenderness, weakness	<ul style="list-style-type: none"> • A clinical diagnosis of myopathy is made if muscle pain or weakness is accompanied by a CK rise ≥10 times upper limit of normal • Rhabdomyolysis can also occur (severe form of myopathy) • Discontinue statin if myopathy is diagnosed or suspected • The risk of myopathy is usually dose-related • Increased risk of myopathy: elderly, comorbidities (eg, diabetes, hypothyroidism, liver or renal disease), combination lipid therapy (statin and fibrate, statin and nicotinic acid) or if taking simvastatin/atorvastatin concomitantly with CYP3A4 inhibitors* 	
	Muscle pain without CK rise	Reduction in dose or temporary discontinuation may be appropriate
	CK rise 3–10 times upper limit normal plus symptoms	Monitor CK weekly Discuss with specialist Reduction in dose or temporary discontinuation may be appropriate
	CK ≥10 times upper limit normal	Discontinue statin immediately

* eg, diltiazem, macrolide antibiotics, azole antifungals and amiodarone

BLOOD PRESSURE LOWERING

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

Everyone with a BP \geq 170/100 mm Hg should have drug treatment and specific lifestyle advice to lower risk factor levels.

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.

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

- All medications have similar efficacy in lowering BP. However, certain medications are preferred in particular conditions (see CD Rom for details).
- A low-dose thiazide diuretic is first-line therapy in most people without contraindications.
- More than one drug is frequently required to lower BP to optimum levels.
- Low-dose combination therapies can maximise effectiveness and help minimise side effects.

After myocardial infarction

Treat ALL people post-MI with a beta-blocker (eg, metoprolol, propranolol or timolol) and an ACE inhibitor long-term regardless of BP level, unless contraindicated.

- Administer concurrently with intensive lifestyle advice and other appropriate medication, such as aspirin and a statin.
- Beta-blockers reduce total mortality, cardiovascular mortality and morbidity.

After stroke or transient ischaemic attack

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.

Secondary prevention post-acute ischaemic stroke or TIA

- Start or increase BP-lowering medication **irrespective** of the BP level (unless the person has symptomatic hypotension), as benefits are also seen in normotensive persons. 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. 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).
- 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.
- Older people tolerate BP-lowering medication as well as younger age groups, and in general, the range of BP treatments available are equally effective.
- Low-dose thiazide diuretics are generally the first drug of choice, but monitoring for electrolyte disturbance is recommended.

- Beta-blockers and ACE inhibitors can be used in this group of people.
- Caution is advised when prescribing alpha-blockers to people aged ≥ 75 years because of the tendency of these drugs to cause postural hypotension.
- People aged ≥ 75 years with isolated raised systolic hypertension (systolic BP ≥ 160 mm Hg with a diastolic BP < 90 mm Hg) have an increased risk of ischaemic stroke and should be managed vigorously.
- Long-acting dihydropyridine calcium channel blockers are a suitable alternative for people aged ≥ 75 years with isolated systolic hypertension when thiazides are contraindicated or poorly tolerated.

Diabetes

- BP target for all people with diabetes is $< 130/80$ mm Hg.
- Aggressive BP control ($< 130/80$ mm Hg, preferably less) is indicated in people with diabetes and overt nephropathy, or diabetes and microalbuminuria, or diabetes and other renal disease.
- ACE inhibitors, thiazide diuretics, beta-blockers, and calcium channel blockers are all effective on lowering BP and reducing the risk of CVD.
- 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.

LONG-TERM USE OF 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 33).

Table 33: Indications for long-term aspirin use

5-year CVD risk	Recommendation
Risk >20% clinically*	After angina or MI commence low-dose aspirin (75–50 mg) with a beta-blocker, a statin and an ACE inhibitor After ischaemic stroke or TIA commence low dose aspirin with a statin. Start or increase doses of BP-lowering drugs (two usually required)
Risk calculated >15%	Commence low-dose aspirin (75–150 mg/day) unless contraindicated
Risk assumed to be >15%: isolated high-risk factors TC ≥8 mmol/L TC:HDL ratio ≥8 BP ≥170/100 mm Hg	Low-dose aspirin is as effective as higher daily doses and may be associated with less bleeding
No clinical CVD and calculated 5-year CVD risk <15%	The risk of a significant bleed or major haemorrhage 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.

- Previous CVD event
- Some genetic lipid disorders
- Diabetes and overt nephropathy
- Diabetes and other renal disease

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.

DIABETES

People Known to have Diabetes

To calculate CVD risk in people with diabetes use the New Zealand cardiovascular risk charts and Tables 2 and 3.

- Measure and record clinical features including duration of diabetes, HbA1c and assessment of renal disease (serum creatinine, ACR and history of renal disease)
- Assume CVD risk >20% in very high risk groups
- Use the risk charts to calculate CVD risk in all other people
- Increase calculated CVD risk by 5% for:
 - 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 peoples from the Indian subcontinent
 - diabetes with microalbuminuria
 - type 2 diabetes for ≥ 10 years or HbA1c consistently $\geq 8\%$.

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

- Urinary albumin loss and serum creatinine are the best screening tests for renal disease.
- Glomerular filtration rate (GFR) is the best test for overall kidney function (see Table 34).

Table 34: Definitions of renal function

Renal condition	Test	Definition
Moderate renal impairment (referral indicated)	Serum creatinine or calculated GFR	≥ 0.15 mmol/L < 60 ml/min/1.73m ²
Microalbuminuria*	Sustained urinary albumin excretion	30–300 mg/day.
	Equivalent ACR for men	≥ 2.5 mg/mmol creatinine
	Equivalent ACR for women	≥ 3.5 mg/mmol creatinine
	Urinary albumin concentration	≥ 20 mg/L
Overt diabetic nephropathy represents a more severe and established form of diabetic nephropathy than microalbuminuria	Urinary albumin excretion	≥ 300 mg/day
	Equivalent ACR	≥ 30 mg/mmol
	Urinary albumin concentration	≥ 200 mg/L

* An adequate estimate of the daily albumin excretion is provided by the ACR.

 People with diabetes require intensive intervention (see Table 35).

Table 35: Summary of the interventions recommended for people with diabetes

Diabetic renal disease	
Screening/identification	ACR and serum creatinine at least annually from diagnosis More frequent monitoring of renal status may be prudent in high-risk ethnic groups (Māori, Pacific and Asian peoples)
Risk factors	Hyperglycaemia (level/duration), inappropriately high BP, albuminuria/proteinuria, retinopathy, ethnicity (Māori, Pacific, Asian), dyslipidemia, smoking
BP control	Aggressive BP control <130/80 (preferably lower) is indicated if diabetes and microalbuminuria, overt nephropathy or other renal disease. Most people require more than one BP-lowering agent
ACE inhibitor (or A2 receptor blocker if ACE not tolerated)	ACE inhibitor: recommended (irrespective of BP levels) if diabetes and microalbuminuria or overt nephropathy is present Note: ACE inhibitors can cause an initial rise in serum creatinine (check 1 week after starting ACE inhibitor). An increase <25% is not, by itself an indication to stop therapy. However, a continued rise may indicate renal stenosis, which should be investigated promptly
Referral	Refer to a specialist for an opinion/management if: <ul style="list-style-type: none"> • overt diabetic nephropathy • creatinine ≥ 0.15 mmol/L • calculated GFR <60ml/min/1.73m² • rapid increase in level of microalbuminuria or proteinuria • difficulty in achieving BP targets • non-diabetic renal disease may be present

Continued over...



See algorithm on CD Rom and in full guideline

Diabetic eye disease	
Screening/identification	<ul style="list-style-type: none"> • Screening with retinal photography (preferred) or slit lamp biomicroscopy should occur at least two yearly, but the number and severity of risk factors may indicate a shorter screening interval • Check and note corrected visual acuity on referral form • People with diabetes and symptomatic visual loss require review by an ophthalmologist, and are not candidates for screening
Risk factors	<ul style="list-style-type: none"> • Diabetes duration, poor glycaemic control, raised BP, dyslipidaemia microalbuminuria/proteinuria, pregnancy, anaemia, Māori and Pacific peoples
Referral	<ul style="list-style-type: none"> • All people with any degree of diabetic retinopathy should be under the supervision of an ophthalmologist, who can specify appropriate monitoring and treatment
Diabetic foot disease	
Screening/identification	<ul style="list-style-type: none"> • Screen at least annually from diagnosis if there are no features of a high risk foot • Screen 3–6 monthly if there are high risk foot features
Risk factors	Older age, males, Māori, Pacific peoples, longer duration of diabetes, poor vision, renal disease, impaired mobility, smoking, poor footwear, poor nutrition and social deprivation/isolation
Referral	Refer people with high risk feet to a specialist diabetic foot clinic or multidisciplinary foot care team (or a podiatrist if these are not available)
Glycaemic control	
Monitoring	Review HbA1c every 3–6 months
Prevention of complications	Tight glycaemic control reduces the risk of and slows the progression of microvascular and macrovascular complications
Targets	A stepwise approach is recommended to lower and maintain HbA1c to as close to physiological levels as possible, preferably <7%, without hypoglycaemia

Continued...



See algorithm on CD Rom and in full guideline

CVD risk assessment	
Screening/ identification	Annual cardiovascular risk assessment using the New Zealand cardiovascular risk charts is recommended for all people with diabetes People with diabetes and overt nephropathy or other renal disease are at high risk of CVD
Lipid targets	The goal for all people with diabetes is to achieve a 5-year CVD risk < 15% and where possible: <ul style="list-style-type: none"> • TC < 4, LDL-C < 2.5, HDL-C > 1 mmol/L • Triglycerides < 1.7 mmol/L
Blood pressure targets and medication	
BP targets	<ul style="list-style-type: none"> • All people with diabetes: BP < 130/80 • Diabetes and renal disease: BP < 130/80, preferably lower. Aggressive BP control is indicated
BP-lowering medication choice	<ul style="list-style-type: none"> • ACE inhibitors, thiazide diuretics, beta-blockers and calcium channel blockers are all effective in lowering BP and reducing CVD events • ACE inhibitors are first-line therapy in people with overt nephropathy or microalbuminuria and should be considered early in all other people with diabetes

Lifestyle
<p>Lifestyle change is central to the management of all people with diabetes and requires specific advice on energy intake and a cardioprotective dietary pattern (see Appendix C), physical activity and smoking cessation, where appropriate (Tables 15–20)</p> <p>Reduce foods rich in saturated fat, added sugars and white flour bakery products</p> <p>To control post-prandial hyperglycaemia, include high-fibre foods with a low to moderate GI at each meal, distribute carbohydrate foods evenly through the day and avoid a large volume of carbohydrate-rich foods at any one meal. See Chapter 2 of the diabetes guideline for a fuller explanation</p> <p>Refer to a dietitian and diabetes nurse specialist for intensive advice</p>

Note: Involving families in diabetes management planning is of particular importance to Māori and Pacific people with diabetes.

ATRIAL FIBRILLATION AND ATRIAL FLUTTER

Assessment of a First Episode of Atrial Fibrillation or Flutter

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 and a decision made on the appropriateness of lipid-modification and BP-lowering medication (see Figure 1).
- 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 36) and contraindications to warfarin (see Table 37) 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).

Table 36: Benefits and harms of treatment with warfarin compared to aspirin

5-year stroke risk %	Benefit of warfarin*		Benefit of aspirin †		Bleeding with warfarin ‡		ICH with aspirin§
	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	ICH per 100 people treated with warfarin 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 is fatal (the definition includes intracranial, respiratory or abdominal bleeds). The risk of ICH is 0.5 per 100 people per year on warfarin and 0.3 per 100 people per year on aspirin.

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

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

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

§ The population rate of ICH is approximately 0.75 per 100 people over 5 years. Therefore, the ICH risk attributable to aspirin is about half the tabulated value.

NNT = Number needed to treat

ICH = Intracranial haemorrhage

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

Table 37: Contraindications to treatment with warfarin

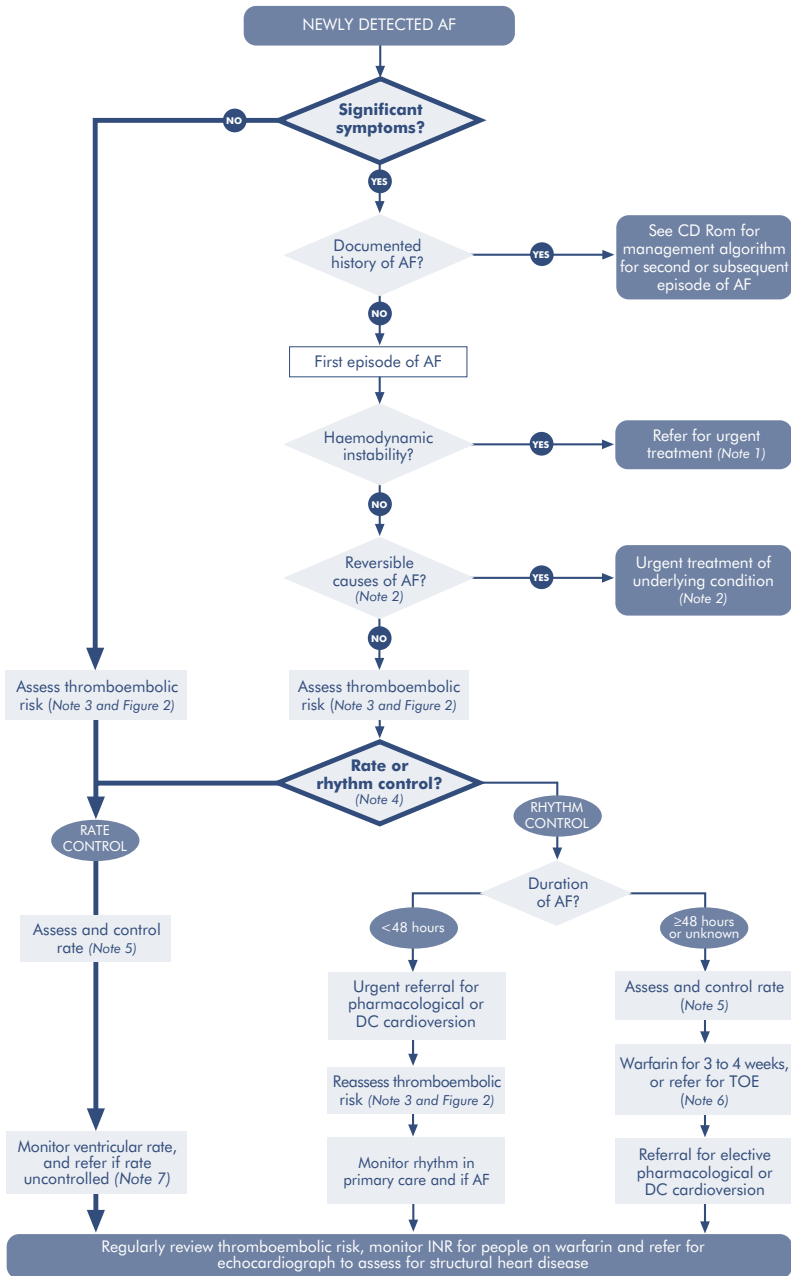
Absolute contraindications	Relative contraindications	NOT contraindications to receiving warfarin
<ul style="list-style-type: none">• Bleeding diathesis• Thrombocytopaenia• Poorly-controlled hypertension (BP consistently $\geq 160/90$ mm Hg)• Non-compliance with medication or INR monitoring• Previous intracranial bleed or retinal haemorrhage• Recent gastrointestinal/genitourinary bleeding• First trimester and last month of pregnancy	<ul style="list-style-type: none">• 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	<ul style="list-style-type: none">• 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 <i>Helicobacter pylori</i>• 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 and treated with warfarin or aspirin (see Figure 2).
- 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 to 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 full guideline, summary or CD Rom.

Figure 3: First episode of atrial fibrillation



Notes to the Atrial Fibrillation Algorithm

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

(See Figure 2)

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.

Note 4: Rhythm control; conversion of AF to sinus rhythm

(See Chapter 5 of full 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 full 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 full guideline)

Rate control is the recommended choice for most people with asymptomatic AF/AFL (see Table 38).

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...

Table 38: Selection of a rate-control agent for people with atrial fibrillation

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.

Note 6: Transoesophageal echocardiography (TOE) guided DC cardioversion

(See Section 8.1.3, Electrical cardioversion in full 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 3–6 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 full 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.

ANGINA AND MYOCARDIAL INFARCTION

Long-term Therapy after Myocardial Infarction or Angina

- Comprehensive cardiac rehabilitation should be considered in all people after MI, coronary artery bypass surgery or angioplasty.
- 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, a beta-blocker and an ACE inhibitor, unless contraindicated (see Table 39).

Table 39: Recommended medications after myocardial infarction or angina

Drug	Recommendation
Aspirin	Aspirin 75 to 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 to high-risk MI survivors including those with: <ul style="list-style-type: none">• 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)

Continued over...

Drug	Recommendation
Beta-blockers	<p>Beta-blockers should be considered for everyone following MI (eg, metoprolol, timolol, propranolol) unless contraindicated</p> <p>Beta-blockers are also recommended in those with left ventricular dysfunction and heart failure</p> <ul style="list-style-type: none"> • 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	<p>An ACE inhibitor should be prescribed for everyone after MI, regardless of left ventricular function</p> <p>Treatment should be started early and continued, especially in those with anterior infarction, LV dysfunction or heart failure</p> <p>Long-term ACE inhibitor therapy should be prescribed for all people with coronary heart disease</p>
Statins	<p>A statin equivalent to simvastatin 20–40 mg daily should be started after MI</p>
Calcium channel blockers	<p>Rate-limiting non-dihydropyridines calcium channel blockers (verapamil and diltiazem) may be considered for people with normal ventricular function where beta-blockers are contraindicated and treatment is required for concurrent angina or hypertension</p>
Nitrates	<p>Nitrates can be used after MI for controlling symptoms of angina, but are not indicated for reducing the risk of further events</p>

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

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

NEW STROKE

Transient Ischaemic Attack

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** already in appropriate institutional care **or** person/family prefer home care despite explanation of the benefits of hospital care.

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

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.

People Presenting with Transient Ischaemic Attacks

- By definition, a TIA lasts <24 hours; however, most resolve within the first hour. If symptoms last >1 hour, manage as if a stroke has occurred.
- People presenting following a TIA should be assessed in a specialist clinic as soon as possible and no later than 7 to 14 days after an attack.
- CT or MRI is recommended after a hemispheric TIA, especially if recurrent and stereotyped. Brain imaging is not routinely recommended after a vertebrobasilar TIA. Other investigations may be indicated.

🔑 All people after an ischaemic stroke or TIA should be on aspirin and a statin unless contraindicated (see Table 40). Start or increase BP-lowering therapy, irrespective of BP level.

Table 40: Recommended medication after ischaemic stroke or transient ischaemic attack

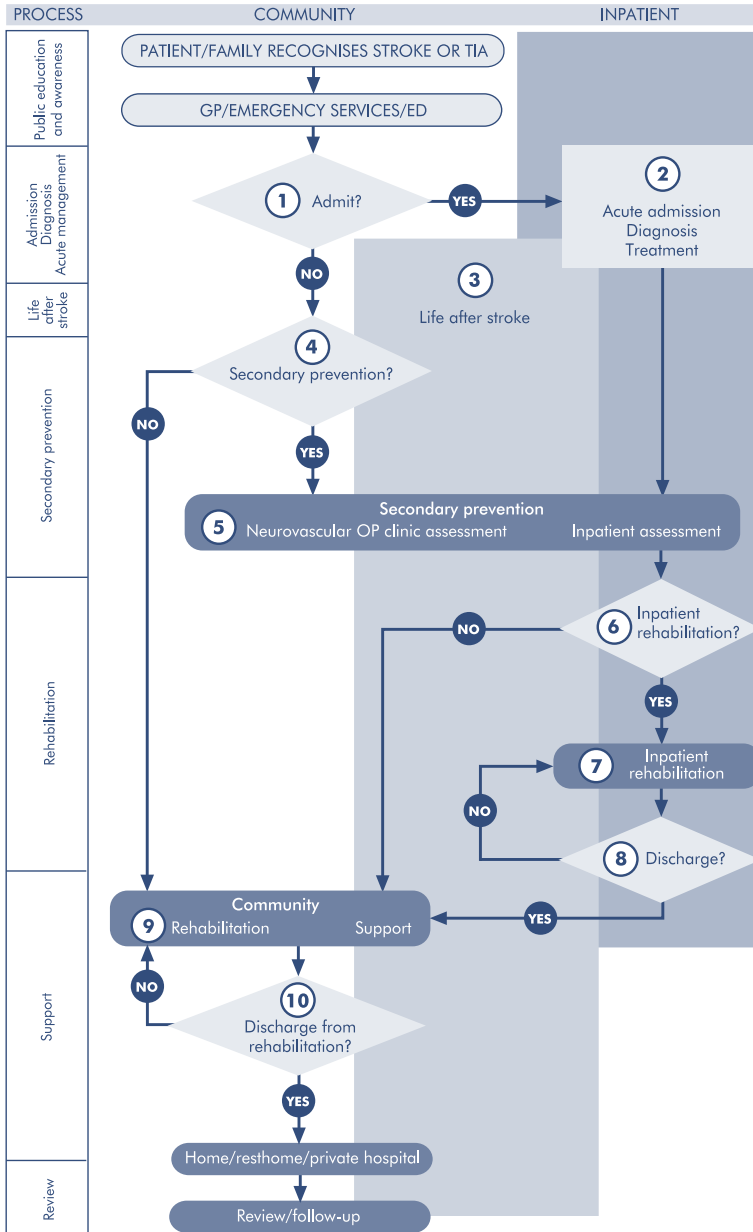
Drug	Recommendation
Aspirin	Aspirin 75–150 mg should be given routinely, long-term after ischaemic stroke or TIA, unless there is an indication for anticoagulation with warfarin. These doses are at least as effective as higher doses
Clopidogrel	Clopidogrel (75 mg/day) can be used as a safe and effective alternative to aspirin after stroke
Dipyridamole	<p>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</p> <p>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</p> <p>Monotherapy with modified-release dipyridamole is recommended for prevention of non-fatal stroke if aspirin is contraindicated and clopidogrel is unavailable</p>
Warfarin	<p>Warfarin should not be prescribed for people with TIA or minor strokes unless cardiac embolism is suspected</p> <p>Warfarin should be considered for people after ischaemic stroke associated with:</p> <ul style="list-style-type: none"> • AF unless contraindicated • mitral valve disease • prosthetic heart valves • MI in the previous 3 months. <p>Warfarin should ideally be started in hospital.</p> <ul style="list-style-type: none"> • 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 <p>The target INR should be 2.5 (range 2.0–3.0)</p>

Continued...

Drug	Recommendation
BP-lowering medication	<p>Acute BP-lowering therapy in ischaemic stroke</p> <ul style="list-style-type: none"> • 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 <p>Long-term BP-lowering in ischaemic stroke</p> <ul style="list-style-type: none"> • 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
Lipid modification	<p>A statin is recommended for most people following ischaemic stroke or TIA. Statin therapy should preferably be started in hospital</p>

LIFE AFTER STROKE

Figure 4: Organised Stroke Services



Key to Figure 4

① *Is admission required for this person?*

All people with stroke should expect to be admitted unless:

- No significant disability affecting functioning **and**
- Urgent outpatient assessment by specialist stroke service available **or**
- Already in appropriate institutional care **or**
- Person/family prefer home care despite explanation of benefits of hospital care

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

② *Acute admission*

- Admission to stroke unit or care of stroke team
- CT within 48 h
- Swallowing assessment within 24 h
- Multidisciplinary team (MDT) assessment within 48 h
- Aspirin initiation (if appropriate) within 48 h

③ *Life after stroke*

- Person has contact information for Stroke Foundation field officers or other support
- Caregiver support
- Cultural issues
- Ongoing education about stroke
- Appropriate advice and information on sexuality, mood, employment, driving

④ *Is diagnosis and secondary prevention an issue for this person?*

Typically appropriate if:

- Further stroke would have important clinical consequences **and**
- Person can cooperate and comply with investigations or antiplatelet drugs **and**
- If for carotid ultrasound, has significant functional recovery from an anterior circulation stroke and fit for surgery

Typically not appropriate if terminal illness, severe dementia/disability eg, in hospital-level care

⑤ *Outpatient clinic/review*

To confirm diagnosis, assess vascular risk factors and address secondary prevention

- Urgent outpatient assessment by clinicians knowledgeable about stroke
- ECG and bloods at GP or ED presentation
- Access within 1 to 2 weeks
- Review by physician with special interest or expertise in stroke management

Continued over...

⑥ *Is inpatient rehabilitation required?*

All people with stroke should expect inpatient rehabilitation by an MDT with expertise in stroke unless:

- No significant residual disability interfering with function on MDT assessment **or**
- Moderate disability (e.g. transfer with 1 person) and early supported discharge service available **or**
- Already in institutional care and community rehabilitation service available

⑦ *Inpatient rehabilitation*

- Admission to stroke unit or care by stroke team within a rehabilitation unit
- Stroke-expert MDT responsible for care
- Person-orientated goal setting
- Daily therapy input (Mon–Fri)
- Family and caregivers involved in rehabilitation
- Appropriate information and support available to person and family

⑧ *Is person ready for discharge to the community?*

Typically appropriate if:

- Medically stable **and**
- MDT has completed assessments of home situation and post-discharge requirements **and**
- An appropriate place for discharge has been identified **and**
- An appropriate plan has been agreed between MDT, person, caregivers and other agencies **and**
- All necessary equipment has been provided **and**
- All follow-up arrangements are in place (rehabilitation, social and GP/primary care)

⑨ *Community rehabilitation*

Can be provided with equal effectiveness in the community or a day hospital

⑩ *Is person ready for discharge from rehabilitation?*

Typically appropriate if:

- Person has achieved agreed therapy goals **and**
- No new goals are identified **and** agreed **and**
- Appropriate supports are in place

Adapted from: Stroke Foundation New Zealand Inc. *Life after stroke: New Zealand guideline for management of stroke*. 2003 p6–7

APPENDIX A

Genetic Causes of 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)	<p>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.</p> <p>People with FH usually have a family history of premature coronary heart disease compatible with autosomal dominant inheritance. Heterozygous FH has a prevalence in the general population of at least 1 in 500.</p> <p>Family tracing of the siblings and children of people with FH is recommended.</p> <p>Refer to a centre with expertise in management of lipid problems as mutation analysis allows more precise family tracing and screening. If referral is not possible these people should be discussed with an appropriate specialist.</p>
Familial defective ApoB (FDB)	These people with elevated ApoB levels 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. Refer 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 (see pages 28–35 of the CVD guideline)

Food	Healthy Servings (per day)	Serving Size Examples	Notes
Vegetables	At least 3–4 servings. Include at every meal	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> 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 tbsps 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	<ul style="list-style-type: none"> 1 medium slice of whole grain bread or ½ bread roll 30 g of other breads (eg, 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 1 small potato, ½ kumara, ⅓ cup yams, ½ cup corn, ½ parsnip, 1 small round of taro 	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 These replace bread/grain products. Limit for weight and diabetes control
Starchy vegetables			
Low-fat or fat-free milk products	2–3 servings or replace with soy products	<ul style="list-style-type: none"> 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 tbsps parmesan or 3 tbsps 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

Continued over...

Food	Healthy Servings (per day)	Serving Size Examples	Notes
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	
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 tbsps reduced-fat mayonnaise or dressing (10% fat or less) 1 tbsps 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 tbsps 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 tbsps sugar, jam, syrup or honey 2 tbsps 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

* 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 /day for people in the healthy weight range who are active with normal triglycerides and no diabetes.

Continued...



Food	Healthy Servings (per day)	Serving Size Examples	Notes
Minimise added salt	Limit high salt seasonings to 1/day	1 tsp seasoning paste 1/6 stock cube or 1/8 tsp stock powder 1/3 tsp gravy mix or 1 tbsp liquid seasoning	Use minimal salt in cooking Do not add salt to meals
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 1/2 cup canned/packet soup 50 g canned or smoked salmon/tuna 30 g other smoked fish/sardines	Choose breads and cereals with less than 450 mg/100 g sodium and spreads with less than 400 mg/100 g sodium Choose low or reduced salt/sodium canned foods, soups, sauces Seasonings, crispbreads, relishes and meals Check labels of cured, corned, pickled, smoked, marinated and canned foods
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	
Nonalcoholic 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

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

APPENDIX E

Land Transport Safety Authority Requirements for People with Heart Disease

Medical Condition	Class 1 or class 6 licence and a D, F, R, T or W licence endorsement in relation to vehicles of less than 4,500 kg GLW or GCW	Class 2, 3, 4 or 5 licence and P, V, I or O licence endorsement
Angina pectoris	Individuals with angina at rest or on minimal exertion despite medical therapy should not drive	Individuals with angina at rest or on minimal exertion despite medical therapy should not drive
Acute uncomplicated myocardial infarction	Should not drive for at least 2 weeks . Return to driving subject to specialist assessment	Should not drive for at least 4 weeks . Return to driving subject to specialist assessment
Coronary artery bypass surgery	Should not drive for at least 4 weeks . Return to driving subject to specialist assessment	Should not drive for at least 3 months . Return to driving subject to specialist assessment
Coronary angioplasty	Should not drive for at least 2 days . Return to driving subject to specialist assessment	Should not drive for at least 4 weeks . Return to driving subject to specialist assessment
Cardiac arrest	Should not drive for at least 2 months . Return to driving subject to specialist assessment	Normally considered permanently unfit to drive. For exceptions refer section 3.3.1 of Land Transport Safety Authority guide

Reproduced from: Land Transport Safety Authority 2002. *Medical Aspects of Fitness to Drive: a guide for medical practitioners*.

For more information see www.ltsa.govt.nz

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- Diabetes – identifying and managing diabetic renal disease
- Diabetes – identifying and preventing visual impairment and blindness
- Diabetes – preventing active foot problems and lower limb amputation
- Atrial fibrillation – warfarin initiation
- Atrial fibrillation – second or subsequent episode of atrial fibrillation
- Atrial fibrillation – antiarrhythmic therapy to maintain sinus rhythm
- Atrial fibrillation – direct current cardioversion for persistent atrial fibrillation

ABBREVIATIONS

A2	Angiotensin II	HDL	High density lipoprotein
ACE	Angiotensin converting enzyme	HDL-C	High density lipoprotein cholesterol
ACR	Albumin:creatinine ratio	HRT	Hormone replacement therapy
AF	Atrial fibrillation	ICH	Intracranial haemorrhage
AFL	Atrial flutter	IFG	Impaired fasting glycaemia
ALT	Alanine transaminase	IGT	Impaired glucose tolerance
ApoB	Apolipoprotein B	INR	International normalised ratio
BMI	Body mass index	IV	Intravenous
BP	Blood pressure	J	Joules
bpm	Beats per minute	kg	Kilogram
CABG	Coronary artery bypass graft	LDL	Low density lipoprotein
CK	Creatine kinase	LDL-C	Low density lipoprotein cholesterol
cm	Centimetres	LP(a)	Lipoprotein (a)
COX2-inhibitor	Cyclooxygenase-2 inhibitor	MDT	Multidisciplinary team
CT	Computed tomography	METs	Metabolic equivalents
CVD	Cardiovascular disease	mg	Milligram
CYP3A4	Cytochrome P4503A4	MI	Myocardial infarction
DBP	Diastolic blood pressure	ml	Millilitre
DC	Direct current	mm Hg	Millimetres of mercury
DHA	Docosahexaenoic acid	mmol/L	Millimole per litre
dL	Decilitre	NNT	Number needed to treat
dsp	Dessert spoon	NRT	Nicotine replacement therapy
ECG	Electrocardiogram	NSAID	Non-steroidal anti-inflammatory agents
ED	Emergency department	OGTT	Oral glucose tolerance test
EPA	Eicosapentaenoic acid	PTCA	Percutaneous transluminal coronary angioplasty
FCH	Familial combined dyslipidaemia	PVD	Peripheral vascular disease
FDB	Familial defective ApoB	SBP	Systolic blood pressure
FH	Familial hypercholesterolaemia	tbsp	Tablespoon
g	Gram	TC	Total cholesterol
GCW	Gross combined weight	TIA	Transient ischaemic attack
GFR	Glomerular filtration rate	TOE	Transoesophageal echocardiogram
GI	Glycaemic index	tsp	Teaspoon
GLW	Gross laden weight	UKPDS	United Kingdom Prospective Diabetes Study
GP	General practitioner	WPW	Wolf Parkinson White
h	Hour		
HbA1c	Haemoglobin type A1c		