RACGP

Royal Australian College *of* General Practitioners

diabetes australia

General practice management of type 2 diabetes

2016–18

Healthy Profession.
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General practice management of type 2 diabetes: 2016–18

Disclaimer

The information set out in this publication is current at the date of first publication and is intended for use as a guide of a general nature only and may or may not be relevant to particular patients or circumstances. Nor is this publication exhaustive of the subject matter. Persons implementing any recommendations contained in this publication must exercise their own independent skill or judgement or seek appropriate professional advice relevant to their own particular circumstances when so doing. Compliance with any recommendations cannot of itself guarantee discharge of the duty of care owed to patients and others coming into contact with the health professional and the premises from which the health professional operates.

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We recognise the traditional custodians of the land and sea on which we work and live.

Supporting the education programs of Diabetes Australia

Type 2 diabetes: Goals for optimum management

The table on the reverse lists goals for optimum management that all people with type 2 diabetes should be encouraged to reach.

This table has been specifically designed as a card for you to pull out and place on your desk or nearby for easy reference.

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Type 2 diabetes: Goals for optimum management

Encourage all people with type 2 diabetes to approach/reach these goals	
Diet	Advise eating according to *Australian dietary guidelines*, with attention to quantity and type of food If concerns are held regarding cardiovascular disease (CVD) risk, advise individual dietary review
Body mass index (BMI)	Therapeutic goal is 5–10% weight loss for people who are overweight or obese with type 2 diabetes Those with BMI >35 kg/m^2 and comorbidities, or BMI >40 kg/m^2, greater weight loss measures should be considered Note that BMI is a difficult parameter to standardise between different population groups
Physical activity	At least 30 minutes of moderate physical activity on most if not all days of the week (total ≥150 minutes/week)
Cigarette consumption	0 per day
Alcohol consumption	Advise ≤2 standard drinks (20 g) per day for men and women
Blood glucose level (BGL)	Advise 6–8 mmol/L fasting and 8–10 mmol/L postprandial Ongoing self-monitoring of blood glucose is recommended for people with diabetes using insulin, people using sulphonylureas or other medicines that may cause hypoglycaemia, hyperglycaemia arising from illness, with haemoglobinopathies, pregnancy or other conditions where data on glycaemic patterns is required Routine self-monitoring of blood glucose in low-risk patients who are using oral glucose-lowering drugs (with the exception of sulphonylureas) is not recommended
Glycated haemoglobin (HbA1c)	Needs individualisation according to patient circumstances. Generally: • ≤53 mmol/mol (48–58 mmol/mol) • ≤7% (6.5–7.5%) Allowing for normal variation in test accuracy, HbA1c results that range between 6.5% and 7.5% (48 and 58 mmol/mol) would reflect this goal.
Total cholesterol <4.0 mmol/L	Initiation of pharmacotherapy is dependent on the assessment of absolute CVD risk (refer to the Australian absolute CVD risk calculator at www.cvdcheck.org.au). This requires using multiple risk factors, which is considered more accurate than the use of individual parameters Once therapy is initiated, the specified targets apply; however, these targets should be used as a guide to treatment and not as a mandatory target
High-density lipoprotein-cholesterol (HDL-C) ≥1.0 mmol/L	
Low-density lipoprotein-cholesterol (LDL-C) <2.0 mmol/L	
Non–HDL-C <2.5 mmol/L	
Triglycerides <2.0 mmol/L	
Blood pressure (BP) ≤140/90 mmHg	Lower BP targets may be considered for younger people and for secondary prevention in those at high risk of stroke, as long as treatment burden does not increase risk The target BP for people with diabetes and albuminuria/proteinuria remains <130/80 mmHg. As always, treatment targets should be individualised and monitored for side effects from medications used to lower BP
Urine albumin excretion	Urine albumin-to-creatinine ratio (UACR): • women: <3.5 mg/mmol • men: <2.5 mg/mmol Timed overnight collection: <20 mcg/min Spot collection: <20 mg/L
Vaccination	Consider immunisation against influenza and pneumococcal disease, and the diphtheria-tetanus-acellular pertussis (dTpa) vaccine

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Acknowledgements

Reviewers

Diabetes Australia Medical Education and Scientific Committee

RACGP Expert Committee – Quality Care

Australian Diabetes Society

Australian Diabetes Educators Association

Australasian Podiatry Council

Exercise and Sports Science Australia

Kidney Health Australia

NPS MedicineWise

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About the RACGP

The RACGP is Australia's largest professional general practice organisation and represents urban and rural general practitioners (GPs). We represent more than 33,000 members working in or towards a career in general practice and are proud that more than 22,500 GPs in Australia have chosen to be a member of the RACGP.

The RACGP is responsible for defining the nature of the general practice discipline, setting the standards and curriculum for education and training, maintaining the standards for quality clinical practice, and supporting GPs in their pursuit of excellence in patient care and community service. We offer our members access to a vast suite of clinical resources, business support tools, education programs and are proud to advocate for the general practice profession on behalf of all GPs.

The RACGP advocates and promotes high-quality diabetes management and care through:

- regular articles in *Australian Family Physician* (*AFP*), the most widely read peer-reviewed general practice journal in Australia, available at www.racgp.org.au/publications/afp
- online general practice education provided by *gplearning* – the RACGP's online learning portal
- advocacy on key issues related to diabetes management
- partnership with Diabetes Australia in the production of this handbook
- giving members access to an extensive library collection, with many items available electronically
- the flagship products *Guidelines for preventive activities in general practice* (Red Book), *Putting prevention into practice: Guidelines for the implementation of prevention in the general practice setting* (Green Book) and *Smoking, nutrition, alcohol, physical activity* (*SNAP): A population health guide to behavioural risk factors in general practice*, available at www.racgp.org.au/your-practice/guidelines

About Diabetes Australia

Diabetes Australia is the national body for people affected by all types of diabetes and those at risk. We are committed to reducing the impact of diabetes.

Diabetes Australia combines the voice of consumers, health professionals and researchers dedicated to diabetes.

Diabetes Australia has four key activities:

- **National leadership** – National policy and advocacy, and raising of awareness of diabetes and its impact.
- **Management of diabetes** – Supporting and developing national self-management programs, and promoting the best possible management of diabetes to help prevent complications. These activities cover type 1, type 2 and gestational diabetes.
- **Prevention** – Supporting and developing prevention policies and programs for both the high-risk population (two million Australians at high risk) and the primary prevention at a whole-of-population level.
- **Research** – Supporting, funding and promoting the best diabetes research.

Diabetes Australia is the Australian member of the International Diabetes Federation (IDF), through which we work to reduce the impact of diabetes throughout the world, particularly in the Western Pacific region.

Working with general practice

Diabetes Australia publishes the *Diabetes Management Journal* quarterly, to inform GPs and health professionals in the field of diabetes management. This ensures that the latest information on the optimum care for people with diabetes, and the latest developments in diabetes management are delivered to frontline healthcare providers. The *Diabetes Management Journal* is available through Diabetes Australia, and professional membership of state and territory diabetes organisations at www.diabetesaustralia.com.au

National Diabetes Services Scheme

Diabetes Australia administers the National Diabetes Services Scheme (NDSS) in conjunction with state and territory diabetes organisations. The NDSS is an Australian Government initiative and has operated successfully for more than 28 years. The NDSS provides universal access for all Australians with diabetes to subsidised diabetes products, and education and support services. As at December 2015, there were more than 1.2 million Australians registered with the NDSS.

Through the NDSS, people with diabetes can receive telephone support via the National Helpline 1300 136 588, along with a range of diabetes information and educational resources and programs targeted for type 1, type 2 and gestational diabetes.

Educational resources from Diabetes Australia

Membership of state and territory diabetes organisations provides access to a wide range of educational resources and support for people with diabetes, their families and carers. To find out more, visit www.diabetesaustralia.com.au and click on your state or territory.

Acronyms

AACB	Australasian Association of Clinical Biochemists
ABI	ankle-brachial index
ACCORD	Action to Control Cardiovascular Risk in Diabetes
ACE	angiotensin converting enzyme
ACEI	angiotensin converting enzyme inhibitor
ACR	albumin-to-creatinine ratio
ADA	American Diabetes Association
ADEA	Australian Diabetes Educators Association
ADIPS	Australian Diabetes in Pregnancy Society
ADS	Australian Diabetes Society
ADVANCE	Advance in Diabetes and Vascular Disease
AEP	Accredited Exercise Physiologist
AHRQ	Agency for Healthcare Research and Quality
AIHW	Australian Institute of Health and Welfare
AN	acanthosis nigricans
APD	Accredited Practising Dietitian
ARA	angiotensin-receptor antagonist
ARB	angiotensin receptor blocker
AUSDRISK	Australian type 2 diabetes risk assessment tool
BGL	blood glucose level
BMI	body mass index
BP	blood pressure
CAD	coronary artery disease
CCM	Chronic Care Model
CDE	Credentialled Diabetes Educator
CDM	chronic disease management
CEITC	Centre for Excellence in Indigenous Tobacco Control
CI	confidence interval
CKD	chronic kidney disease
COI	conflict of interest
COPD	chronic obstructive pulmonary disease
CVD	cardiovascular disease
DBP	diastolic blood pressure
DE	diabetes educator

DKA	diabetic ketoacidosis
DR	diabetic retinopathy
DPP-4	dipeptidyl peptidase-4
DPP-4i	dipeptidyl peptidase-4 inhibitor
dTpa	diphtheria-tetanus-acellular pertussis
ECG	electrocardiogram
eGFR	estimated glomerular filtration rate
ELIXA	Evaluation of Lixisenatide in Acute Coronary Syndrome
EMPA-REG	Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes
EXAMINE	Examination of Cardiovascular Outcomes with Alogliptin versus Standard of Care
FBG	fasting blood glucose
FIELD	Fenofibrate Intervention and Event Lowering in Diabetes
FRE	Framingham risk evaluation
GAD	glutamic acid decarboxylase
GDM	gestational diabetes mellitus
GI	glycaemic index
GIP	glucose-dependent insulinotropic polypeptide
GLP-1	glucagon-like peptide-1
GLP-1 RA	glucagon-like peptide-1 receptor agonist
GP	general practitioner
GPMP	general practice management plan
HAPO	Hyperglycaemia and Adverse Pregnancy Outcome
HbA1c	glycated haemoglobin
HDL-C	high-density lipoprotein-cholesterol
HHS	hyperosmolar hyperglycaemic state
HOCM	hypertrophic obstructive cardiomyopathy
HONC	hyperosmolar nonketotic coma
IA-2	insulinoma antigen-2
IADPSG	International Association of the Diabetes and Pregnancy Study Groups
IBD	irritable bowel disease
IBS	irritable bowel syndrome
IDF	International Diabetes Federation
IFG	impaired fasting glucose
IGT	impaired glucose tolerance

IMPROVE-IT	Improved reduction of outcomes: Vytorin efficacy international trial
IUCD	intrauterine contraceptive device
LADA	latent autoimmune diabetes of adults
LDL-C	low-density lipoprotein-cholesterol
LEADER	Liraglutide Effect and Action in Diabetes: Evaluation of Cardiovascular Outcome Results
MACE	major adverse cardiovascular events
MBS	Medicare Benefits Schedule
MI	myocardial infarction
MODY	maturity onset diabetes of the young
MR	modified release
NDSS	National Diabetes Services Scheme
NHMRC	National Health and Medical Research Council
NICE	National Institute for Health and Care Excellence
NICEQOF	National Institute for Health and Clinical Excellence Quality and Outcomes Framework
NIH	National Institutes of Health
NPH	Neutral Protamine Hagedorn
NPS	National Prescribing Service
NSAID	non-steroidal anti-inflammatory drug
NVDPA	National Vascular Disease Prevention Alliance
OCP	oral contraceptive pill
OGTT	oral glucose tolerance test
OHA	oral hypoglycaemic agent
OR	odds ratio
ORIGIN	Outcome Reduction with Initial Glargine Intervention
PAD	peripheral arterial disease
PAID	problem areas in diabetes
PBAC	Pharmaceutical Benefits Advisory Committee
PBS	Pharmaceutical Benefits Scheme
PCOS	polycystic ovary syndrome
PHN	Primary Health Network
PHQ-2	Patient health questionnaire-2
PHQ-9	Patient health questionnaire-9
PIP	Practice Incentives Program
PROactive	Prospective pioglitazone clinical trial in macrovascular events

RACGP	The Royal Australian College of General Practitioners
RANZCOG	The Royal Australian and New Zealand College of Obstetricians and Gynaecologists
RBG	random blood glucose
RCPA	The Royal College of Pathologists of Australasia
RCT	randomised controlled trial
RR	relative risk
RRR	relative risk reduction
SAVOR-TIMI	Saxagliptin Assessment of Vascular Outcomes Recorded in Patients with Diabetes Mellitus Thrombolysis in Myocardial Infarction
SBP	systolic blood pressure
SGLT2	sodium glucose co-transporter 2
SGLT2i	sodium glucose co-transporter 2 inhibitor
SIGN	Scottish Intercollegiate Guidelines Network
SIP	Service Incentive Payments
SMBG	Self-monitoring of blood glucose
SNAP	Smoking, nutrition, alcohol, physical activity
SOE	statement of evidence
STOP-NIDDM	Study to Prevent Non-Insulin-Dependant Diabetes Mellitus
SU	sulphonylureas
TBI	toe-brachial index
TCA	team care arrangement
TECOS	Trial to Evaluate Cardiovascular Outcomes after Treatment with Sitagliptin
TGA	Therapeutic Goods Administration
TIA	transient ischaemic attack
TZD	thiazolidinedione
UACR	Urine albumin-to-creatinine ratio
UKPDS	UK Prospective Diabetes Study
USPSTF	US Preventive Services Task Force
WHO	World Health Organization

Contents

Summary, explanation and source of recommendations

The coding scheme for levels of evidence and grades of recommendation in this publication are provided in this summary. Refer to Section 1.3. How to use these guidelines for further explanation on how to use these recommendations.

National Health and Medical Research Council's levels of evidence and grades of recommendation

Levels of evidence	
Level	**Explanation**
I	Evidence obtained from a systematic review of level II studies
II	Evidence obtained from a randomised controlled trial (RCT)
III-1	Evidence obtained from a pseudo-RCT (ie alternate allocation or some other method)
III-2	Evidence obtained from a comparative study with concurrent controls: • non-randomised, experimental trial • cohort study • case-control study • interrupted time series with a control group
III-3	Evidence obtained from a comparative study without concurrent controls: • historical control study • two or more single arm study • interrupted time series without a parallel control group
IV	Case series with either post-test or pre-test/post-test outcomes
Practice Point	Opinions of respected authorities, based on clinical experience, descriptive studies or reports of expert committees
Grades of recommendations	
Grade	**Explanation**
A	Body of evidence can be trusted to guide practice
B	Body of evidence can be trusted to guide practice in most situations
C	Body of evidence provides some support for recommendation(s), but care should be taken in its application
D	Body of evidence is weak and recommendation must be applied with caution

Scottish Intercollegiate Guidelines Network's levels of evidence and grades of recommendations (1999–2012)

Levels of evidence	
Level	**Explanation**
1++	High-quality meta-analyses, systematic reviews of randomised controlled trial (RCTs), or RCTs with a very low risk of bias
1+	Well-conducted meta-analyses, systematic reviews or RCTs with a low risk of bias
1–	Meta-analyses, systematic reviews of RCTs or RCTs with a high risk of bias
2++	High-quality systematic reviews of case control or cohort or studies High-quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
2+	Well-conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2–	Case control or cohort studies with a high risk of confounding bias or chance and significant risk that the relationship is not causal
3	Non-analytic studies (eg case reports, case series)
4	Expert opinion
Grades of recommendations	
A	At least one meta-analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population; or A body of evidence consisting principally of studies rated as 1+, directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
C	A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
D	Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+
Good practice points	Recommended best practice based on the clinical experience of the guideline development group

American Diabetes Association's levels of evidence

Levels of evidence	Explanation
A	Clear evidence from well-conducted, generalisable randomised controlled trials (RCTs) that are adequately powered, including: • evidence from a well-conducted multicenter trial • evidence from a meta-analysis that incorporated quality ratings in the analysis Compelling non-experimental evidence (ie 'all or none' rule developed by the Centre for Evidence-Based Medicine at the University of Oxford) Supportive evidence from well-conducted RCTs that are adequately powered, including: • evidence from a well-conducted trial at one or more institutions • evidence from a meta-analysis that incorporated quality ratings in the analysis
B	Supportive evidence from well-conducted cohort studies: • evidence from a well-conducted prospective cohort study or registry • evidence from a well-conducted meta-analysis of cohort studies Supportive evidence from a well-conducted case-control study
C	Supportive evidence from poorly controlled or uncontrolled studies: • evidence from randomised clinical trials with one or more major or three or more minor methodological flaws that could invalidate the results • evidence from observational studies with high potential for bias (such as case series with comparison with historical controls) • evidence from case series or case reports Conflicting evidence with the weight of evidence supporting the recommendation
E	Expert consensus or clinical experience

Summary of recommendations

Please note that the asterisk (*) that appears next to 'Grade' in the following recommendations are explained in Summary, explanation and source of recommendations.

3.1 Identifying risk of diabetes in asymptomatic patients

Recommendations	Reference	Grade*
Individuals should be screened for risk of diabetes every three years from 40 years of age using AUSDRISK	25 NHMRC, 2009	C

Individuals at high risk with any one of following risk factors:
   – AUSDRISK score of 12 or more
   – all people with a history of a previous cardiovascular event (acute myocardial infarction or stroke)
   – women with a history of gestational diabetes mellitus
   – women with polycystic ovary syndrome
   – patients on antipsychotic drugs
	25
	NHMRC, 2009	

• should be screened with fasting blood glucose (or glycated haemoglobin [HbA1c]) • every three years		B
Individuals at high risk with impaired glucose tolerance test or fasting glucose (not limited by age) should be screened:	25 NHMRC, 2009	C
• with fasting blood glucose (or HbA1c) • every 12 months		B C
Risk assessment should begin from 18 years of age in Aboriginal and Torres Strait Islander peoples	25 NHMRC, 2009	Practice Point

4. Preventing type 2 diabetes

Recommendations	Reference	Grade*
Lifestyle modifications that focus on increased physical activity, dietary change and weight loss should be offered to all individuals at high risk of developing type 2 diabetes Structured diabetes prevention programs are available	42 NHMRC, 2009	A
Bariatric surgery can be considered in selected morbidly obese individuals (based on weight alone or the presence of comorbidities) who are at high risk of type 2 diabetes	42 NHMRC, 2009	C
Individuals who are at high risk of diabetes should be identified through the use of risk assessment tools	42 NHMRC, 2009	C

5.1 Patient-centred diabetes care

Recommendations	Reference	Grade*
A patient-centred communication style that incorporates patient preferences, assesses literacy and numeracy, and addresses cultural barriers to care should be used	19 American Diabetes Association, 2015	B

5.2 A structured diabetes care program consistent with the Chronic Care Model

Recommendations	Reference	Grade*
Care should be aligned with components of the Chronic Care Model (CCM) to ensure productive interactions between a prepared proactive practice team and an informed activated patient	56 American Diabetes Association, 2016	A
When feasible, care systems should support team-based care, community involvement, patient registries and embedded decision-support tools to meet patient needs	56 American Diabetes Association, 2016	B
Treatment decisions should be timely and based on evidence-based guidelines that are tailored to individual patient preferences, prognoses and comorbidities	56 American Diabetes Association, 2016	B

5.3 Patient education and self management

Recommendations	Reference	Grade*
All people with type 2 diabetes should be referred for structured diabetes patient education	42 NHMRC, 2009	A
Diabetes education should be delivered in groups or individually	42 NHMRC, 2009	A
Diabetes education should be culturally sensitive and tailored to the needs of socioeconomically disadvantaged populations	42 NHMRC, 2009	B

6.1 Physical activity

Recommendations	Reference	Grade*
People with type 2 diabetes of all ages benefit from accumulating 30 minutes or more of moderate physical activity on most if not all days of the week	64 Briffa T et al, 2006	B
Exercise and physical activity (involving aerobic and/or resistance exercise) should be performed on a regular basis	65 SIGN, 2014	D

6.2 Diet

Recommendations	Reference	Grade*
Consumption of cereal foods (especially three serves a day of wholegrains) is associated with reduced risk of type 2 diabetes	71 NHMRC, 2013	B
Consumption of at least one and a half serves of dairy foods (eg milk, yoghurt, cheese) per day is associated with reduced risk of type 2 diabetes	71 NHMRC, 2013	C

6.3 Weight

Recommendations	Reference	Grade*
Adults with impaired fasting glucose, impaired glucose tolerance or diabetes can be strongly advised that the health benefits of 5–10% weight loss include prevention, delayed progression or improved control of type 2 diabetes	78 NHMRC, 2013	A
For adults with body mass index (BMI) >40 kg/m², or adults with BMI >35 kg/m² and comorbidities that may improve with weight loss, bariatric surgery may be considered, taking into account the individual situation	78 NHMRC, 2013	A
Use BMI to classify overweight or obesity in adults	78 NHMRC, 2013	B
For adults, use waist circumference, in addition to BMI, to refine assessment of risk of obesity-related comorbidities	78 NHMRC, 2013	C

6.4 Smoking cessation

Recommendations	Reference	Grade*
Smoking cessation should be a major focus of the management of people with smoking-related diseases	86 RACGP, 2011	A
All smokers should be offered brief advice to quit smoking	86 RACGP, 2011	A

6.5 Alcohol consumption

Recommendations	Reference	Grade*
People with diabetes can take alcohol in moderation as part of a healthy lifestyle, but should aim to keep within the target consumption recommended for people without diabetes	65 SIGN, 2014	B

8.1 Glycaemic monitoring

Recommendations	Reference	Grade*
Glycated haemoglobin (HbA1c) measurement should be used to assess long-term blood glucose control	96 NHMRC, 2009	A
Self-monitoring of blood glucose is recommended for patients with type 2 diabetes who are using insulin, where patients have been educated in appropriate alterations in insulin dose (Refer to Self-monitoring of blood glucose under Section 8.2. Medication for examples of instances when self-monitoring of blood glucose may be considered)	65 SIGN, 2014	B
Routine self-monitoring of blood glucose in people with type 2 diabetes who are using oral glucose-lowering drugs (with the exception of sulphonylureas) is not recommended	65 SIGN, 2010	B

In practice

Recommendations	Reference	Grade*
Blood glucose control should be optimised because of its beneficial effects on the development and progression of microvascular complications	96 NHMRC, 2009	A
The potential harmful effects of optimising blood glucose control in people with type 2 diabetes should be considered when setting individual glycaemic targets	96 NHMRC, 2009	A
The general glycated haemoglobin (HbA1c) target in people with type 2 diabetes is ≤53 mmol/mol (≤7%). Adjustments to diabetes treatment should be considered when HbA1c is above this level	96 NHMRC, 2009	A
Targets for self-monitoring of blood glucose levels are 6–8 mmol/L for fasting and preprandial, and 6–10 mmol/L for two hour postprandial	96 NHMRC, 2009	C

8.2.1 General medication

Recommendations	Reference	Grade*
Care should be taken to address the potential harmful effects of optimising blood glucose control when setting individual glycaemic targets	96 NHMRC, 2009	A
Interventions to achieve target glycated haemoglobin (HbA1c) should begin with lifestyle modification followed by pharmacological options selected on the basis of individual clinical circumstances, side effects and contraindications	96 NHMRC, 2009	A
Blood glucose control should be optimised because of its beneficial effects on the development and progression of microvascular complications	96 NHMRC, 2009	A

9. Managing cardiovascular disease risk

Recommendations	Reference	Grade*
Patients with pre-existing cardiovascular disease (CVD) are at high risk	149 NVDPA, 2012	A
All adults with type 2 diabetes and known prior CVD (except haemorrhagic stroke) should receive the maximum tolerated dose of a statin, irrespective of their lipid levels Note: The maximum tolerated dose should not exceed the maximum available dose (eg 80 mg atorvastatin, 40 mg rosuvastatin)	150 Baker IDI, 2015	A
Adults with any of the following conditions do not require absolute CVD risk assessment using the Framingham Risk Equation because they are already known to be at clinically determined high risk of CVD: • Diabetes and aged >60 years • Diabetes with microalbuminuria (>20 mcg/min or urine albumin-to-creatinine ratio [UACR] >2.5 mg/mmol for men, and >3.5 mg/mmol for women) • Moderate or severe chronic kidney disease (CKD) (persistent proteinuria or estimated glomerular filtration rate [eGFR] <45 mL/min/1.73 m^2) • A previous diagnosis of familial hypercholesterolaemia • Systolic blood pressure ≥180 mmHg or diastolic blood pressure ≥110 mmHg • Serum total cholesterol >7.5 mmol/L	149 NVDPA, 2012	D

Calculate risk level using an evidence-based tool:
• National Vascular Disease Prevention Alliance charts,
  www.cvdcheck.org.au
• New Zealand Cardiovascular Risk charts,
  www.health.govt.nz/publications
• Heart Foundation NZ, www.knowyournumbers.co.nz
	149
	NVDPA, 2012
		B

Aboriginal and Torres Strait Islander peoples are generally assumed to be at higher risk	149 NVDPA, 2012	B

Antihypertensive medication to manage cardiovascular risk

Recommendations	Reference	Grade*
Blood pressure-lowering therapy in people with diabetes should preferentially include an angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB)	149 NVDPA, 2012	A
If monotherapy does not sufficiently reduce blood pressure, add one of the following:		
• Calcium channel blocker	149 NVDPA, 2012	B
• Low-dose thiazide or thiazide-like diuretic	149 NVDPA, 2012	C

Lipid medication to manage cardiovascular risk

Recommendations	Reference	Grade*
Use statins as first-line therapy	149 NVDPA, 2012	A

Antithrombotic therapy

Recommendations	Reference	Grade*
All adults with type 2 diabetes and known prior cardiovascular disease should receive long-term antiplatelet therapy unless there is a clear contraindication	150 Baker IDI, 2015	A
All adults with type 2 diabetes and a history of ischaemic stroke or transient ischaemic attack should receive:	150 Baker IDI, 2015	
• low-dose aspirin, or		A
• clopidogrel, or		A
• combination low-dose aspirin and extended-release dipyridamole		B
All adults with type 2 diabetes and recent acute coronary syndrome and/or coronary stent should receive, for 12 months after the event or procedure:	150 Baker IDI, 2015	
• combination low-dose aspirin and clopidogrel, or		B
• combination low-dose aspirin and prasugrel, or		B
• combination low-dose aspirin and ticagrelor		C
All adults with type 2 diabetes and a history of coronary artery disease, but no acute event in the past 12 months should receive:	150 Baker IDI, 2015	
• long-term low-dose aspirin, or		A
• long-term clopidogrel if intolerant to aspirin		B
In the presence of atrial fibrillation or other major risk factors for thromboembolism, there should be consideration of anticoagulant therapy according to other relevant guidelines	150 Baker IDI, 2015	Practice Point

10.1 Diabetic retinopathy

Recommendations	Reference	Grade*
Ensure that all people with diabetes have a dilated fundus examination and visual acuity assessment at the diagnosis of diabetes and at least every two years	158 NHMRC, 2008	None provided (Level I evidence)
Examine higher risk patients (eg longer duration of diabetes, or poor glycaemic control, blood pressure or blood lipid control) without diabetic retinopathy at least annually	158 NHMRC, 2008	None provided (Level I evidence)
Conduct annual screening for Aboriginal or Torres Strait Islander peoples with diabetes	158 NHMRC, 2008	None provided (Level IV evidence)

10.3 Neuropathy

Diabetic peripheral neuropathy

Recommendations	Reference	Grade*
All patients should be screened for distal symmetric polyneuropathy starting at diagnosis of type 2 diabetes and at least annually thereafter, using simple clinical tests	19 American Diabetes Association, 2015	B
Antidepressants, including tricyclics, duloxetine and venlafaxine should be considered for the treatment of patients with painful diabetic peripheral neuropathy	65 SIGN, 2014	A
Anticonvulsants, including pregabalin and gabapentin, should be considered for the treatment of patients with painful diabetic peripheral neuropathy	65 SIGN, 2014	A

10.4 Nephropathy

Recommendations	Reference	Grade*
Assessment Kidney status in people with type 2 diabetes should be assessed by:		
• annual screening for albuminuria (note that dipstick urine test is not adequate to identify albuminuria)	166 NHMRC, 2009	B
• annual estimated glomerular filtration rate (eGFR; in mL/min/1.73 m^2)	166 NHMRC, 2009	B
Management Reducing the risk or slowing the progression of nephropathy can be achieved by:		
• blood glucose control should be optimised aiming for a general glycated haemoglobin (HbA1c) target ≤7%	166 NMHRC 2009	A
• optimising blood pressure control	166 NMHRC 2009	A
In people with type 2 diabetes and microalbuminuria or macroalbuminuria, angiotensin receptor blocker (ARB) or angiotensin converting enzyme inhibitor (ACEI) antihypertensive should be used to protect against progression of kidney disease	166 NHMRC, 2009	A
People with type 2 diabetes should be informed that smoking increases the risk of chronic kidney disease	166 NHMRC, 2009	B
People with diabetes and microalbuminuria are considered at high cardiovascular disease risk, and should be treated with multifactorial interventions (refer to Chapter 9. Managing cardiovascular risk)	149 NVDPA, 2012	D

10.5 Foot complications

Recommendations	Reference	Grade*
Assess all people with diabetes and stratify their risk of developing foot complications	160 NHMRC, 2011	C
Assess risk stratification by inquiring about previous foot ulceration and amputation plus falls risk, visually inspecting the feet for structural abnormalities and ulceration, assessing for neuropathy using either the neuropathy disability score or a 10 g monofilament and palpating foot	160 NHMRC, 2011	C
People assessed as having intermediate-risk or high-risk feet should be offered a foot protection program. This includes foot care education, podiatry review and appropriate footwear	160 NHMRC, 2011	C
Pressure reduction, otherwise referred to as redistribution of pressure or offloading, is required to optimise the healing of plantar foot ulcers	160 NHMRC, 2011	B
Offloading of the wound can be achieved with the use of a total contact cast or other device rendered irremovable	160 NHMRC, 2011	B
People with diabetes-related foot ulceration are best managed by a multidisciplinary foot care team	160 NHMRC, 2011	C

11. Glycaemic emergencies

Recommendations	Reference	Grade*
The potential harmful effects of optimising blood glucose control in people with diabetes should be considered when setting individual glycaemic targets	96 NHMRC, 2009	A
Improving blood glucose control increases the risk of hypoglycaemia	96 NHMRC, 2009	None provided (Level I evidence)

13.2 Pregnancy with pre-existing diabetes

Recommendations	Reference	Grade*
Pre-pregnancy glycaemic control should be maintained as close to the non-diabetic range as possible, taking into account risk of maternal hypoglycaemia	65 SIGN, 2014	C
All women with diabetes should be prescribed *high-dose pre-pregnancy folate supplementation, continuing up to 12 weeks' gestation	65 SIGN, 2014	B
All women with pre-gestational diabetes should be encouraged to achieve excellent glycaemic control†	65 SIGN, 2014	D
Postprandial glucose monitoring should be carried out in pregnant women with type 1 or 2 diabetes Postprandial glucose monitoring should be carried out in pregnant women with gestational diabetes and may be considered in pregnant women with type 1 or 2 diabetes	65 SIGN, 2014	C
Pre-pregnancy care provided by a multidisciplinary team is strongly recommended for women with diabetes	65 SIGN, 2014	C

*5 mg of folate

†Glycated haemoglobin (HbA1c) <48 mmol/mol (6.5%) and consider stabilisation using metformin and/or insulin to achieve glycaemic targets. However, metformin has a category C rating in pregnancy. Continuation or initiation of metformin therapy should be considered only following full disclosure to the patient and under specialist supervision. Sulphonylureas may be associated with adverse neonatal outcomes and are thus best avoided[66,229–232]

Management

Recommendations	Reference	Grade*
Pregnant women with gestational diabetes mellitus should be offered dietary advice and blood glucose monitoring, and be treated with glucose-lowering therapy depending on target values for fasting and postprandial targets	65 SIGN, 2014	A

Follow-up of patients with a history of gestational diabetes mellitus

Recommendations	Reference	Grade*
Women with a history of gestational diabetes mellitus should receive a postpartum oral glucose tolerance test at 6–12 weeks	19 American Diabetes Association, 2015	E

14.1 Sick day management

Recommendations	Reference	Grade*
Patients should be educated to develop a sick day management plan after initial diagnosis. This plan should be reviewed at regular intervals	261 Australian Diabetes Educators Association, 2014	None provided
Assist in the development of a sick day care plan and preparation of a home sick day management kit for patients to use during episodes of sickness	261 Australian Diabetes Educators Association, 2014	None provided

15. Diabetes and end-of-life care

Recommendations	Reference	Grade*
To minimise the risks of hypoglycaemia and metabolic compensation, a blood glucose range of 6–15 mmol/L is appropriate for most palliative care patients	265 Diabetes UK, 2013	None provided
Maintain glycated haemoglobin (HbA1c) at no lower than 58 mmol/mol (7.5%) if on hypoglycaemic medication depending on the individual's life expectancy, as HbA1c will be less relevant in patients with months or days left to live	265 Diabetes UK, 2013	None provided

Updates in this edition

Chapter	
Summary of recommendations	This has been moved to the front of the handbook
4. Preventing type 2 diabetes	This chapter references interventions in clinical trials that may assist general practitioners (GPs) understand, and consider implementing with, patients at high risk of diabetes to prevent progress to type 2 diabetes
6. Lifestyle modification	This chapter has been reviewed and includes some practical updates for GPs to implement in patients diagnosed with type 2 diabetes
7. The person with diabetes – Assessment	Revision on practice guidelines on clinical assessment is included
8. Managing glycaemia	The new Australian blood glucose treatment algorithm developed in collaboration with the Australian Diabetes Society is now included (Figure 4) A table to guide clinical considerations of glucose-lowering agents is also newly embedded (Table 6) A revised insulin titration algorithm is included for premixed insulins
9. Managing cardiovascular risk	Updated information on antithrombotic therapy including aspirin
10. Managing microvascular and other complications	Expanded section on foot complications
11. Glycaemic emergencies	Expanded information on the management of diabetes glycaemic emergencies – including Appendix J. Detailed information on glycaemic emergencies
13. Diabetes and reproductive health	Revision of advice with emerging evidence, both in pregnancy with existing diabetes and gestational diabetes
16. Issues under debate	Revision of blood pressure targets is discussed, based upon new evidence particular to diabetes Possible new criteria for screening for diabetes in at-risk populations are discussed
Appendices The following appendices from the 2014–15 edition have been removed	Australian type 2 diabetes risk assessment tool (AUSDRISK) General outline of management of hyperosmolar nonketotic coma from glycaemic emergencies Potential drug interactions

1. Introduction

Diabetes is a national health priority. *The Australian National Diabetes Strategy 2016–2020* was released by the Australian Government in November 2013. The number of people with type 2 diabetes is growing, most likely the result of rising overweight and obesity rates, lifestyle and dietary changes, and an ageing population. Within 20 years, the number of people in Australia with type 2 diabetes may increase from an estimated 870,000 in 2014, to more than 2.5 million.[1] The most socially disadvantaged Australians are twice as likely to develop diabetes.

If left undiagnosed or poorly managed, type 2 diabetes can lead to coronary artery disease (CAD), stroke, kidney failure, limb amputations and blindness. The early identification and optimal management of people with type 2 diabetes is therefore critical. General practice has the central role in type 2 diabetes management across the spectrum, from identifying those at risk right through to caring for patients at the end of life. These guidelines give up-to-date, evidence-based information tailored for general practice to support general practitioners (GPs) and their teams in providing high-quality management.[1]

In the development of the 2016–18 edition of *General practice management of type 2 diabetes*, The Royal Australian College of General Practitioners (RACGP) has focused on factors relevant to current Australian clinical practice. The RACGP has used the skills and knowledge of your general practice peers who have an interest in diabetes management and are members of the RACGP Specific Interests Diabetes Network.

This publication has been produced in accordance with the rules and processes outlined in the RACGP's conflict of interest (COI) policy. The RACGP's COI policy is available at www.racgp.org.au/support/policies/organisational

This edition represents 19 years of a successful relationship between the RACGP and Diabetes Australia. We acknowledge the support of the RACGP Expert Committee – Quality Care, the Medical Education and Scientific Committee of Diabetes Australia, and RACGP staff in the development of these guidelines.

1.1 Defining type 2 diabetes

Diabetes is a group of disorders and the 10th leading cause of deaths in Australia. There are four clinical classes of diabetes:[1]

- **Type 1 diabetes** – Results from ß-cell destruction due to an autoimmune process usually leading to insulin deficiency

- **Type 2 diabetes** – Results from a progressive insulin secretory defect on the background of insulin resistance

- **Gestational diabetes mellitus** (GDM) – Defined as glucose intolerance with onset or first recognition during pregnancy

- **Other specific types of diabetes** (Section 3.3. Impaired fasting glucose or impaired glucose tolerance) – Due to other causes such as genetic defects in ß-cell function, genetic defects in insulin action, diseases of the exocrine pancreas (eg cystic fibrosis), and drug-induced or chemical-induced causes (eg treatment of human immunodeficiency virus/acquired immune deficiency syndrome [HIV/AIDS] or after organ transplantation)

Type 2 diabetes is a largely preventable, chronic and progressive medical condition that results from two major metabolic dysfunctions: insulin resistance and then pancreatic islet cell dysfunction causing a relative insulin deficiency. In an individual, these occur due to modifiable lifestyle-related risk factors interacting with non-modifiable and genetic risk factors.

The relative insulin deficiency leads to chronic hyperglycaemia and multiple disturbances in carbohydrate, protein and fat metabolism including:

- ß-islet cell dysfunction, failure of response to insulin signalling and increased islet cell apoptosis
- α-cell dysfunction with elevated glucagon levels
- resultant disorders of hepatic gluconeogenesis and insulin resistance with elevated glucose production
- muscle cell insulin resistance with decreased glucose uptake
- kidney adaptation with altered gluconeogenesis and increased glucose reabsorption via increased sodium glucose transporter protein activity
- diminished incretin hormonal production or resistance
- maladaptive cerebral hormonal responses to insulin and appetite
- increased lipolysis with elevated free fatty acids.

1.2 A patient-centred approach

Throughout these guidelines we refer to patient-centred care.

The concept of patient-centred care incorporates the patient's experience of care and patients as partners in their healthcare.[2] In practice, this means providing care that is 'respectful of and responsive to individual patient preferences, needs and values, and ensures that patient values guide all clinical decisions'[3] and supports self management.

Understanding a patient's diabetes-related (and comorbidity) experiences can improve practitioner–patient communication and help the GP understand their patient's priorities for education, resources and management. This is essential for building and adapting diabetes management plans to be consistent with an individual patient's needs.

1.3 How to use these guidelines

These guidelines have been designed to provide pragmatic, evidence-based recommendations for use in general practice, and adopt the most recent recommendations from organisations including the National Health and Medical Research Council (NHMRC), Scottish Intercollegiate Guidelines Network (SIGN), American Diabetes Association (ADA) and other relevant sources. The recommendations tables include the reference or source of each recommendation, and the grade of recommendation. In cases where these are not available or current, results of systematic reviews and primary research studies have been considered to formulate the overall recommendation. References to support these recommendations are included.

In each section, where possible, information is presented as:

• recommendations

• clinical context (or what you need to know)

• in practice (or what you can do).

Information specific to the Aboriginal and Torres Strait Islander population is highlighted in boxed text. Recommendations in some areas are different for Aboriginal and Torres Strait Islander patients. It is therefore important to identify, record and report the Aboriginal and Torres Strait Islander status of patients.

The RACGP has a position paper outlining the processes of identification, available at www.racgp.org.au/yourracgp/faculties/aboriginal

Refer to Summary, explanation and source of recommendations.

2. Clinical governance – Sustaining and improving high standards of care

The RACGP defines clinical governance as a framework through which clinicians and health service managers are jointly responsible and accountable for patient safety and quality care.[4]

Within this framework are embedded the principles of recognisably high standards of care, transparent responsibility and accountability for maintaining those standards, and a constant dynamic of quality improvement.[5]

2.1 Applying a clinical governance framework to diabetes care

Achieving equitable, safe, effective and high-quality care for patients across the spectrum of type 2 diabetes is no small task. It requires a coordinated interaction between patients, healthcare providers and the healthcare system, with a focus on improving the patient's experience and outcomes throughout the continuum of care.

Each of these elements requires systems and support. For example, comprehensive care for diabetes starts with prevention: through timely identification of at-risk individuals, education and support, it is possible to prevent or delay the onset of type 2 diabetes. The key is implementing risk assessment strategies and subsequently having the resources and communication strategies to effect change in patients' lifestyles. These need to operate and be supported at local and national levels.

Effective leadership is essential. Owners of a general practice (and others involved in its corporate governance) play an active role in developing these systems by cultivating a culture focused on clinical quality and patient-centred care.

In practice

Applying a clinical governance approach to your general practice means focusing on:

- **patients** – providing high-quality, effective and ongoing care, and ensuring good communication and support to enable patients to be informed and involved
- **healthcare teams** – ensuring adequate training and resources for the practice team and developing working relationships with all potential members of a diabetes team, including endocrinologists and Credentialled Diabetes Educators (CDEs)

- **quality improvement** – managing risk, ensuring high standards of care, using clinical audits, and creating and maintaining an environment that supports clinical excellence
- **information** – ensuring high-quality information systems, management and sharing that are the backbone to integrated care.

2.2 Models of high-quality healthcare

The medical home

Quality and safety in diabetes care start with coordinated, ongoing and comprehensive primary healthcare. Primary care is the central component of care across the spectrum of patients with diabetes: those dealing with a new diagnosis, managing (often multiple) medications, with complications of diabetes and multimorbidity, through to patients at the end of life.

A general practice chosen by a patient to provide ongoing, comprehensive, patient-centred care is known as a 'medical home'. The medical home is responsible for the patient's healthcare across their entire health journey, and this approach results in better health outcomes for patients and their families.[6] Australian general practice encapsulates the medical home model.[7]

This model has measurable benefits, including improved continuity of patient care,[8] and improved quality and cost effectiveness of care for patients with a chronic disease.[9]

Medical homes reduce disparities in access to quality care among traditionally difficult to reach groups,[10,11] which leads to improved overall population health[12] and lower overall healthcare spending.[13–15] The RACGP's *Vision for general practice and a sustainable healthcare system* (www.racgp.org.au/vision) is based on the patient-centred medical home model and is informed by RACGP's definition of quality general practice.

A model for chronic disease management

For patients with type 2 diabetes across the spectrum, structured care programs that are easy to implement, well supported and meet the needs of the individual are required. These programs bring together healthcare teams, evidence-based guidelines, useful support tools and good systems to support patients throughout their journey.

Refer to Chapter 5. Structured care and patient education for more information.

The Chronic Care Model (CCM), developed by the MacColl Institute
(www.improvingchroniccare.org/index.php?p=The_Chronic_Care_Model&s=2)
identifies the fundamental elements of a healthcare system that supports high-quality
chronic disease care:

- health system (organisation and mechanisms)
- delivery system design
- decision support
- clinical information systems
- self-management support
- the community.

The CCM has been shown to be an effective framework for improving the quality
of diabetes care.[16]

In practice

Health system

GPs can help create a health system that facilitates easy and appropriate access
to care for people with diabetes by reducing barriers associated with accessing,
and maintaining healthcare across primary care and other health tiers.

General practices can access the Australian Government system level incentives to
support diabetes care. This support is provided through Medicare Benefits Schedule
(MBS) payments to GPs, nurses, allied health professionals and general practices.
These include the Chronic Disease Management (CDM) items (formerly known as
Enhanced Primary Care), which provide support for developing management plans
and organising team care.

Patients have experienced improvements in process and clinical outcomes with these
management plans and team care arrangements.[17]

ABORIGINAL AND TORRES STRAIT ISLANDER POINT

It is recommended that all practices identify patients of Aboriginal or Torres Strait
Islander descent. Registering patients also allows access to the Closing the
Gap Pharmaceutical Benefits Scheme (PBS) co-payment, earlier interventions
(as determined by PBS criteria) and access to specific MBS item numbers.

Refer to Appendix A. Accessing government support for diabetes care in general
practice.

Delivery system design

Diabetes care requires a proactive preventive approach to keeping patients as healthy as possible rather than episodic or reactive intervention when complications arise. An effective system to achieve this will engage patients with a range of healthcare providers using good communication and information technology.[17]

Collaborative multidisciplinary teams are best suited to provide diabetes care, facilitate patient self management, identify those patients who require individualised support and coordinated case management.[18,19] Using multidisciplinary care and engaging the wider team have been shown to improve outcomes for people with diabetes.[20]

A team approach provides flexible and comprehensive care to meet individual patient needs. Roles within a general practice team are not mutually exclusive, and clear guidance is required to identify the team member primarily responsible for key activities. Teamwork success may be supported by workflow coordination and management of structured care programs (care planning).

Decision support

Accessible guidelines for diabetes management and associated issues (eg management and prevention guidelines for all types of diabetes; refer to Austroads and National Transport Commission [www.austroads.com.au/drivers-vehicles/assessing-fitness-to-drive] Therapeutic Guidelines Limited [TGL] antibiotic guidelines [https://tgldcdp.tg.org.au/etgAccess]) are required for GPs to make decisions about diabetes care that is consistent with evidence and meets regulations.

Having electronic records also facilitates decision to support this goal by ensuring prescription error checking against medication allergy, and drug–drug and drug–disease interactions, creating diabetes clinical measures and databases, facilitating risk assessment such as cardiovascular disease (CVD) risk scoring and thus providing a basis for data collection linked to quality improvement processes.

Clinical information and recall and reminder systems

Structured diabetes care programs are based upon good information management systems (eg registers, recalls and reminders) combined with risk factor, complication assessment management and comorbidity strategies. Management plans are most effective when they involve a team care arrangement and are reviewed regularly.[17]

Structured recall systems ensure a patient receives formal reviews at regular intervals. Several studies have shown that computerised recall systems, monitoring and reminding patients and practice team members about appointments, investigations and referrals, improve diabetes care.[21–24] Computerised systems can also provide automated reminders, generate mailing lists of those overdue for preventive activities and help minimise repeat data entries.

Combining a reminder system with a practice register ensures that the reminder system is systematic and targeted. This can prevent patients with diabetes missing out on basic care such as screening for retinopathy and foot care. Depending on the complexity of individual patient needs, structured recall may occur on a three-month to 12-month basis.

For example, during a consultation, the GP and patient make key management decisions or team care changes dependent on care plan progress. A recall is added in a time frame suited to the patient's needs. This cycle then repeats. Another example is where a structured recall may ensure that all necessary investigations are completed before the next practice visit by the patient.

Self-management support

The aim of self-management support is to facilitate skills-based learning and patient empowerment. Diabetes self-management, education and support programs improve understanding of, and belief in, the importance of those factors that influence diabetes (eg goals, complications, living with a chronic illness), and thus might be able to be modified by patients' behaviours and actions (eg self-monitoring of blood glucose [SMBG], dietary choices and physical activity). Other factors, such as medication(s), foot care, individual complication risk assessment and understanding of laboratory results, may need engagement with a health professional team.

The community

Community resources and policies can be harnessed to improve the quality of the practice service including:

- National Diabetes Services Scheme (NDSS)
- Diabetes Australia resources
- Primary Health Network (PHN) collaborations
- local endocrinology specialist services – private and tertiary-level care
- allied health support teams including CDEs, Accredited Practising Dietitians (APDs) and Accredited Exercise Physiologists (AEPs)
- partnerships with universities or hospitals in providing diabetes care

- medication advice at pharmacies
- diabetes support groups, diabetes support applications and online support groups
- community health centres.

Translated diabetes resources are available from the NDSS diabetes portal, which includes resources from Diabetes Australia, Diabetes Australia state and territory agents, and non-government organisations (http://multiculturalportal.ndss.com.au).

> **ABORIGINAL AND TORRES STRAIT ISLANDER POINT**
>
> Some PHNs run chronic care coordination programs for Aboriginal and Torres Strait Islander patients that can help access practical help in attending a range of specialist and allied health appointments.

2.3 A quality improvement program relevant to diabetes care

Accreditation against the RACGP's *Standards for general practices*, 4th edn, requires a commitment to quality improvement from a general practice. This can involve examining practice structures, systems and clinical care. Using practice data to identify areas in need of improvement is one way to achieve this.

Clinical audit software tools are widely available to assist practices to evaluate clinical outcomes for patients with diabetes. Audit information can be used to improve management of patients with diabetes on many levels. For example, patients can be identified on the basis of:

- incomplete information such as no smoking status, no recent blood pressure (BP) or glycated haemoglobin (HbA1c) reading
- Aboriginal and Torres Strait Islander status
- the presence of other risk factors such as HbA1c >75 mmol/mol (9%), microalbuminuria or comorbidities
- outstanding diabetes cycle of care items
- recent hospitalisation
- existing complications or comorbidities.

To help practices start quality improvement activities, the RACGP is developing a core set of clinical indicators to support care evaluation. This is supported with the RACGP's *Practice guides and tools for clinical indicators*, available at www.racgp.org.au/your-practice/business/tools/support

Four of these indicators are relevant to diabetes care:

Indicator number	Description
1	Practice infrastructure to support safety and quality of patient care
5	Assessment of absolute cardiovascular risk
12	Screening for retinopathy in patients with diabetes
13	Screening for nephropathy in high-risk patients (including diabetes)

Use of clinical indicators to assess care is advised but entirely voluntary.

3. Screening, risk assessment, case finding and diagnosis

3.1 Identifying risk of diabetes in asymptomatic patients

Recommendations	Reference	Grade*
Individuals should be screened for risk of diabetes every three years from 40 years of age using AUSDRISK	25 NHMRC, 2009	C

Individuals at high risk with any one of following risk factors:
  – AUSDRISK score of 12 or more
  – all people with a history of a previous cardiovascular event (acute myocardial infarction or stroke)
  – women with a history of gestational diabetes mellitus
  – women with polycystic ovary syndrome
  – patients on antipsychotic drugs
	25
	NHMRC, 2009	

• should be screened with fasting blood glucose (or glycated haemoglobin [HbA1c])		B
• every three years		C
Individuals at high risk with impaired glucose tolerance test or fasting glucose (not limited by age) should be screened:	25 NHMRC, 2009	
• with fasting blood glucose (or HbA1c)		B
• every 12 months		C
Risk assessment should begin from 18 years of age in Aboriginal and Torres Strait Islander peoples	25 NHMRC, 2009	Practice Point

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Type 2 diabetes is the most common form of diabetes in Australia, although many cases remain undiagnosed. Additionally, almost one in six adults is affected by impaired glucose tolerance (IGT) or impaired fasting glucose (IFG).[1] Earlier detection increases opportunities to reduce morbidity and mortality.

In practice

Patients should be screened for diabetes risk every three years from 40 years of age using the Australian type 2 diabetes risk assessment tool (AUSDRISK; www.health. gov.au/preventionoftype2diabetes). Aboriginal and Torres Strait Islander peoples should be screened from 18 years of age. Those with a risk score of 12 or more should have a blood examination for fasting blood glucose (FBG) or HbA1c.

Screen for undiagnosed diabetes in individuals at high risk[25,26] (Box 1).

Box 1. People considered to be at high risk of type 2 diabetes

- People of any age with impaired glucose tolerance (IGT) or impaired fasting glucose (IFG)*
- All patients with a history of a cardiovascular event (eg acute myocardial infarction, angina, peripheral vascular disease or stroke)
- People aged ≥35 years originating from the Pacific Islands, Indian subcontinent or China
- People aged ≥40 years with body mass index (BMI) ≥30 kg/m^2 or hypertension
- Women with a history of gestational diabetes mellitus (GDM)
- Women with polycystic ovary syndrome (PCOS)
- Patients taking antipsychotic medication

*Annual fasting blood glucose (FBG) or HbA1c is reserved for those people identified with IGT test or IFG (not limited by age)

Screening for risk of diabetes in specific or high-risk populations

There is a higher prevalence of type 2 diabetes in lower socioeconomic than higher socioeconomic Australians.[27] Certain ethnic groups are more at risk.[28] People with Pacific Islander, Southern European or Asian backgrounds are twice as likely as other Australians to have developed diabetes within five years.[29]

Aboriginal and Torres Strait Islander peoples are three times more likely to have diabetes than non-Indigenous Australians, and type 2 diabetes is a direct or indirect cause for 20% of Aboriginal and Torres Strait Islander peoples deaths.[30]

The AUSDRISK calculates the risk of developing diabetes over a five-year period. Patients with scores of 12 or more are considered at high risk (Table 1).

Table 1. Diabetes risk

AUSDRISK score	Risk of developing type 2 diabetes within five years*
≤5	1 in 100
6–8	1 in 50
9–11	1 in 30
12–15	1 in 14
16–19	1 in 7
≥20	1 in 3

*The overall score may overestimate the risk of diabetes in those aged <25 years and underestimate the risk in Aboriginal and Torres Strait Islander peoples

Practice points

- For Aboriginal and Torres Strait Islander peoples, AUSDRISK can be used from 18 years of age.
- Those considered at high risk (Box 1) should have an FBG or HbA1c test every three years.
- People with low individual risk or who are from a community with low prevalence (<5%) may be screened for risk with AUSDRISK every three years.[25]

Refer to Chapter 13. Diabetes and reproductive health for recommendations on screening in pregnancy.

3.2 Case finding in patients with symptoms suggestive of diabetes

Clinical context

Clinical suspicion for type 2 diabetes needs to remain high, as type 2 diabetes is often asymptomatic and developing in younger populations. Secondary causes of diabetes should also be considered in the presence of symptoms suggestive of diabetes.[31]

Clinical symptoms suggestive of diabetes

Symptoms of diabetes include:

- lethargy, polyuria, polydipsia
- frequent fungal or bacterial infections
- blurred vision
- loss of sensation (ie touch, vibration, cold)
- poor wound healing
- weight loss.

Clinical signs of insulin resistance

Signs of insulin resistance may include the following:

- Acanthosis nigricans (AN) – Typically characterised by hyperpigmentation (darkening of skin pigment) and usually accompanied by a velvety change in texture of the affected skin. Common sites are the neck and axillae.[32]
- Skin tags – Benign (non-cancerous) skin growths on the body or face. They can be smooth or wrinkled, skin-coloured or just slightly darker than skin colour and can vary in size.
- Central obesity – Defined by a high waist-to-hip ratio, waist-to-thigh ratio and waist circumference.
- Hirsutism – Excess facial and body hair, especially on women, which can indicate insulin resistance.

Box 2 provides examples clarifying when insulin levels may be useful.

> ### Box 2. When are insulin levels helpful?
>
> - There is no role for routinely testing insulin levels to assess insulin resistance in impaired glucose tolerance (IGT), impaired fasting glucose (IFG) or in the evaluation of type 2 diabetes
> - Patients with signs of insulin resistance should be screened for diabetes with fasting blood glucose (FBG) or glycated haemoglobin (HbA1c)

Tests to detect diabetes in symptomatic patients

In the presence of symptoms suggestive of hyperglycaemia or a clear clinical diagnosis (eg a patient presenting with hyperglycaemic crisis, a single elevated FBG ≥7.0 mmol/L or a random blood glucose ≥11.1 mmol/L), this is confirmatory of a diagnosis of diabetes. A second laboratory test is not required to confirm the diagnosis.

Diagnosis of diabetes in asymptomatic patients

Testing those at high-risk or with a clinical suspicion for diabetes involves three types of biochemical analyses (Box 3).

> ### Box 3. Diagnostic criteria for type 2 diabetes
>
> - Fasting blood glucose (FBG) ≥7.0 mmol/L or random blood glucose ≥11.1 mmol/L confirmed by a second abnormal FBG on a separate day
> - Oral glucose tolerance test (OGTT) before (fasting) and two hours after an oral 75 g glucose load is taken. Blood glucose is measured. Diabetes is diagnosed as FBG ≥7.0 mmol/L or two-hour blood glucose is ≥11.1 mmol/L
> - Glycated haemoglobin (HbA1c) ≥48 mmol/mol (6.5%; on two separate occasions)
>
> These are via venous sampling under laboratory methodology

Confirmatory testing for asymptomatic patients

A second laboratory result is required for confirmation of the diagnosis of diabetes in asymptomatic patients. It is recommended that the same laboratory result be repeated without delay using a new blood sample for confirmation because there will be a greater likelihood of concurrence.

Figure 1a. Screening and diagnosis algorithm – Fasting blood glucose

```
                    Fasting blood glucose

    <5.5 mmol/L          5.5–6.9 mmol/L           ≥7 mmol/L

  Diabetes unlikely      Diabetes possible       Diabetes likely

 Re-test every three   May need to perform oral   Confirm with a repeat
 years if indicated*   glucose tolerance test     test only in asymptomatic
                       (OGTT)†‡                    patients‡
```
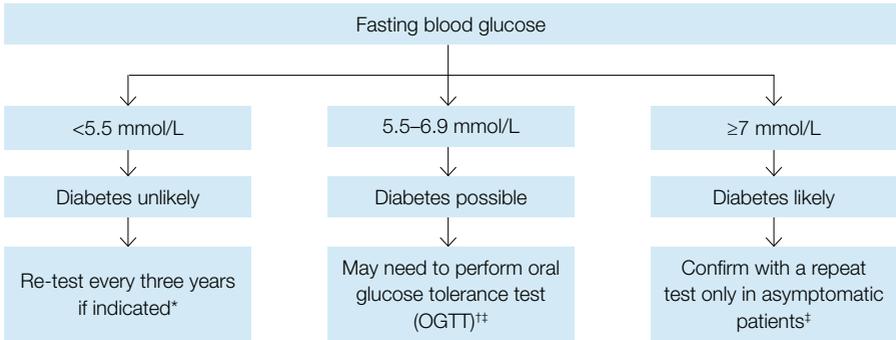
Figure 1b. Screening and diagnosis algorithm – Glycated haemoglobin

```
              Glycated haemoglobin (HbA1c)
    Non-fasting – No preparation required. Interpret with care
      for haemoglobinopathies or abnormal red cell lifespan

              ≥48 mmol/mol (≥6.5%)

                  Diabetes likely

            Confirm with follow up
               HbA1c test‡§||
```
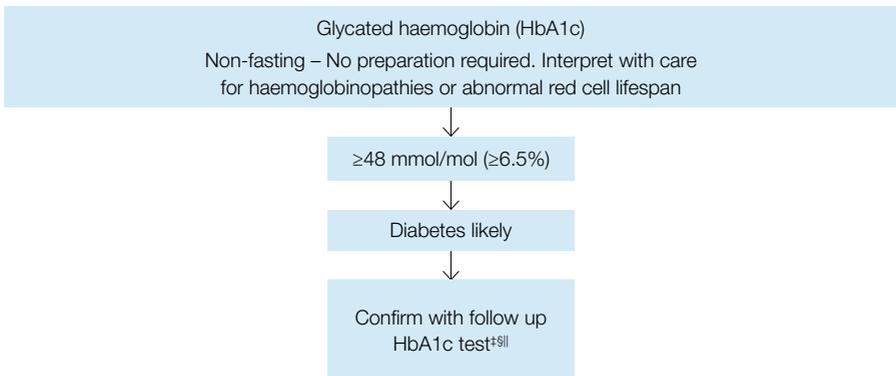
Figure 1c. Screening and diagnosis algorithm – Oral glucose tolerance test

Oral glucose tolerance test (OGTT)

Fasting glucose <6.1 mmol/L	Fasting glucose 6.1–6.9 mmol/L	Fasting glucose <7 mmol/L	Fasting glucose ≥7 mmol/L
Two-hour glucose <7.7 mmol/L	Two-hour glucose <7.8 mmol/L	Two-hour glucose ≥7.8 and ≤11 mmol/L	Two-hour glucose ≥11.1 mmol/L

Normal glucose tolerance	Impaired fasting glucose (IFG)*†	Impaired glucose tolerance (IGT)‡	Diabetes confirmed†

- lifestyle modification
- refer to allied health professionals
- blood pressure, body mass index, lipids
- waist circumference
- Problem areas in diabetes (PAID) tool
- general support
- yearly diabetes screen

- register with National Diabetes Services Scheme (NDSS)
- notify road traffic authority (RTA)
- assess modifiable risk factors
- assess cardiovascular disease (CVD) risk
- PAID tool
- refer to diabetes teams

*World Health Organization (WHO) criteria used[36]

Practice Points for diagnosed diabetes

†Monitor risk factors, and symptoms:

- Manage the individual patient by assessing modifiable risk factors and CVD risk
- Refer patients to a dietitian and a physical activity program
- Provide pre-conception advice to women in reproductive age with a history of gestational diabetes

‡Provide access to diabetes health teams, and commence lifestyle modifications and/or medication. Register with the NDSS and notify the RTA

§Medicare Benefits Schedule (MBS) item number 66841 allows for diagnostic use only once every 12 months. The request slip should be annotated as HbA1c for Service Incentive Payment (SIP) and Practice Incentives Program (PIP) purposes. However, a confirmatory HbA1c test (MBS item number 66551) should be ordered before treatment initiation[37]

‖A value of <48 mmol/mol (6.5%) does not exclude diabetes diagnosed using plasma glucose testing in symptomatic patients

NB: IGT and IFG cannot be diagnosed using HbA1c

3.3 Impaired fasting glucose or impaired glucose tolerance

The definition of diabetes is based on a collection of symptoms based on an agreed glycaemic measure. On glucose challenge testing, patients with elevated glucose not high enough to be diagnosed with type 2 diabetes, are considered to have either IFG or IGT (Figure 1). To apply the diagnostic algorithm, refer to Table 2.

These states are not considered to be benign and reflect a risk of developing diabetes in the future. In addition, as CVD risk is distributed across a continuum of post-challenge glucose levels, any degree of post-challenge hyperglycaemia may be associated with the development of premature CVD.[33]

Microvascular complications are commonly present at the time of diagnosis of type 2 diabetes in symptomatic and asymptomatic individuals.[25]

Table 2. Apply the diagnostic algorithm

Diagnostic method	Advantages/Disadvantages	
Fasting blood glucose (FBG) venous blood	**Fasting** (eight hours)	Additionally may be used to detect impaired fasting glucose (IFG)
Glycated haemoglobin (HbA1c) ≥48 mmol/mol or ≥6.5%	**Non-fasting** Note that HbA1c may lack accuracy (specificity and/or sensitivity) in: • acute onset glycaemic states such as post-traumatic type 2 diabetes (eg pancreatitis), rapid onset of glycaemia with sepsis and steroid use, etc • people with haemoglobinopathy or haemolysis, or advanced chronic kidney disease • people with iron deficiency (artificially elevated). In such cases, a fasting venous blood glucose or oral glucose tolerance test (OGTT) may assist diagnosis Abnormal HbA1c values generally should be repeated in asymptomatic patients and confirmed on a different day, unless two abnormal tests (eg FBG and HbA1c) are already available from the same day	Not useful for assessment of impaired glucose tolerance (IGT) Threshold of 48 mmol/mol is linked to escalating microvascular disease, and HbA1c is a better predictor of macrovascular disease than FBG and two-hour post-glucose[34,35] Lacks sensitivity with acute glycaemic states
OGTT	**Fasting** (eight hours) 75 g glucose administered orally Blood is collected from a fasting venous sample and two-hour post-glucose challenge venous sample	Only standard method used to detect IGT. May concurrently detect IFG

HbA1c as a diagnostic tool

HbA1c has recently been approved as a diagnostic test for diabetes under the Medicare Benefits Schedule (MBS) and by the World Health Organization (WHO). The Australian Diabetes Society (ADS), the Royal College of Pathologists of Australasia (RCPA), and the Australasian Association of Clinical Biochemists (AACB) have reviewed the available evidence and confirmed that HbA1c can be used to diagnose diabetes.[38]

Diagnostic dilemmas

Discordant testing

If diagnosing diabetes, due to the different physiological measures of glycaemia, confirmatory tests at times may give discordant results especially if you do not repeat the initial diagnostic test. For example, HbA1c levels may not be elevated in acute glycaemic states in newly diagnosed diabetes, such that a value of <48 mmol/mol (<6.5%) does not exclude diabetes in the presence of an elevated blood glucose testing ($\geq$7 mmol/L fasting or $\geq$11.1 mmol/L random). When the results of more than one test are discordant, the laboratory result that is above the diagnostic cut-off point should be repeated to make the diagnosis.

Alternative diagnoses

Alternative diagnoses to type 2 diabetes include unusual presentations of:

Type 1 diabetes

Consider type 1 diabetes if there is the presence of:

- ketosis/ketonuria (which may be absent)
- polyuria, polydipsia
- weight loss or BMI <25 kg/m^2
- <50 years of age
- personal and family history of autoimmune disease
- rapid onset of symptoms.

If suspicious of type 1 diabetes:

- Management of any hyperglycaemia should not be delayed and should include immediate assessment for possible ketosis and metabolic disorders such as hyperosmolar states. If blood ketone level is elevated seek help immediately. Blood ketones >0.6 mmol/L are abnormal and require investigation in the presence of hyperglycaemia.

- Non-urgent confirmatory tests for glutamic acid decarboxylase (GAD) and/or insulinoma antigen-2 (IA-2) antibodies. These will be present in 90% of patients with type 1 diabetes. When measuring antibodies, higher rates of false negative results occur early in the development of type 1 diabetes. However, false negative results decrease when two different antibody tests are measured.
- Test for plasma C-peptide level.[39] Levels <0.2 nmol/L on non-fasting sampling, support the diagnosis of type 1 diabetes. This will determine those patients with hyperglycaemia in the absence of or with minimal insulin production. This may not be low in acute early onset type 1 diabetes.

Latent autoimmune diabetes of adults

Latent autoimmune diabetes of adults (LADA) is diabetes with ß-islet cell antibodies more commonly occurring in adulthood, with a more rapid course of ß-cell destruction, a poorer metabolic response to sulphonylurea therapy and a more rapid progression to insulin requirement to control hyperglycaemia due to ß-cell failure.[40]

Monogenic diabetes

Monogenic diabetes is a disorder with the following characteristics:

- onset before 25 years of age
- non-ketotic diabetes mellitus often with a stable lower level hyperglycaemia
- autosomal dominant inheritance
- primary defect in the function of the pancreatic ß-cells.

Monogenic diabetes is genetically heterogeneous and all forms are dominantly inherited. There is variance among the forms with two main types – neonatal diabetes mellitus (rare) and maturity onset diabetes of the young (MODY). MODY subtypes may vary in the severity of hyperglycaemia, need for insulin and risk for future complications. Apart from MODY type 2, all other forms of monogenic diabetes are due to transcription factor gene mutations. Not all forms of the MODY phenotype have yet been defined. Suspected cases should be referred to a specialist endocrinologist, and management options and possible genetic diagnosis should be considered.[41]

Gestational diabetes mellitus

Refer to Section 13.3. Gestational diabetes mellitus.

4. Preventing type 2 diabetes

Recommendations	Reference	Grade*
Lifestyle modifications that focus on increased physical activity, dietary change and weight loss should be offered to all individuals at high risk of developing type 2 diabetes Structured diabetes prevention programs are available	42 NHMRC, 2009	A
Bariatric surgery can be considered in selected morbidly obese individuals (based on weight alone or the presence of comorbidities) who are at high risk of type 2 diabetes	42 NHMRC, 2009	C
Individuals who are at high risk of diabetes should be identified through the use of risk assessment tools	42 NHMRC, 2009	C

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

The dysglycaemic states (IFG, IGT) occur when blood glucose levels (BGLs) are elevated above normal, but are not high enough to be diagnosed as diabetes.

Intervention is warranted to prevent or delay progression to type 2 diabetes, and to reduce mortality associated with the metabolic condition itself.

Clinical trial evidence demonstrates that the metabolic progression to type 2 diabetes can be slowed or stopped with effective diet and lifestyle modification, as well as with some drug therapies. Studies demonstrating prevention of type 2 diabetes development by structured lifestyle behaviour change programs have been conducted in Finland, the US, China and India.[43–46] These had intensive programs supporting the intervention group, with the goals of intervention between 5% and 7% weight reduction using a low-kilojoule and low-fat diet, and moderate intensity physical activity (eg brisk walking) for at least 150 minutes/week.

In patients with dysglycaemic states, structured lifestyle interventions can achieve a relative risk reduction (RRR) of up to 58% in the development of type 2 diabetes. In the Indian study, the numbers needed to treat was 6.4, to prevent one incident case of diabetes over three years using a lifestyle modification program. Longer term follow-up has revealed a 43% reduction in the rate of diabetes at 20 years in the Da Qing study, 43% reduction at seven years in the Finnish study, and 34% reduction at 10 years in the US Diabetes Prevention program. Notably, in the US study, the strongest determinant of reduced diabetes incidence was weight reduction, with each 1 kg loss leading to a RRR of 16%. Of the therapies, pharmacotherapy with metformin

has been shown to achieve a RRR of 31%, particularly in obese individuals with BMI >35 kg/m^2. This is, however, still less effective than successful lifestyle change.[47] Of the trials that evaluated mortality benefits from interventions, lifestyle has not definitively shown clear benefits, except when mediated through diabetes prevention. Pharmacological interventions have not shown reduced CVD mortality benefit.[48]

Note: At the time of publication, metformin does not have Therapeutic Goods Administration (TGA) or Pharmaceutical Benefits Scheme (PBS) approval for this indication within Australia.

In practice

In addition to providing comprehensive risk assessment, screening, diagnosis and management for diabetes, GPs should consider systems for identifying and managing patients with IGT or IFG who are at high risk of diabetes. They should also include absolute CVD risk assessment, and chronic kidney disease risk assessment and management in this group. Referral to government-supported type 2 diabetes and CVD prevention programs should be considered where these exist. Programs and strategies for educating patients about diabetes to encourage lifestyle modification should also be considered.

Lifestyle modification

Lifestyle modification programs (refer to Chapter 6. Lifestyle modification) should be developed using a patient-centred approach. These should be individualised with realistic goals based on what the patient can and wants to achieve. Each plan should: focus on physical activity, dietary modification and weight control; be long term; and involve partners and other family members.

The main element of the programs was an intensive lifestyle modification aimed at helping participants achieve and maintain 7% weight loss and ≥150 minutes per week of moderate-intensity physical activity. The US study included 75 minutes per week of strength training, while the Finnish study provided strength training twice weekly. All studies with success had individual goal setting, and provided individualised and group counselling, predominantly on behavioural change and nutrition.[49]

Plans could involve other practice team members and may include referral to allied health professionals such as, APDs, CDEs and AEPs, physiotherapists and clinical psychologists. Consider structured goal-oriented programs and community resources.

Diabetes prevention programs are supported by different state and local health authorities. General practices may seek advice on local patient referral pathways for those with IGT or IFG, or those identified by risk assessment tools such as AUSDRISK by contacting their local Diabetes Australia state or territory organisation on 1300 136 588 or at www.diabetesaustralia.com.au/prevention

5. Structured care and patient education

5.1 Patient-centred diabetes care

Recommendations	Reference	Grade*
A patient-centred communication style that incorporates patient preferences, assesses literacy and numeracy, and addresses cultural barriers to care should be used	19 American Diabetes Association, 2015	B

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

How well a patient can read and use numbers has a significant impact on their ability to self-manage. Patients with diabetes and lower literacy or numeracy skills are at greater risk for poor diabetes outcomes.[50]

Many factors influence a person's literacy and numeracy (eg socioeconomic status, cognitive function, culture). Health literacy is defined as an individual's ability to read, perform basic numeracy skills, and understand and use healthcare information to make decisions and follow instructions for treatment.[51] In 2006, the Australian Bureau of Statistics (ABS) identified that half of healthcare clients lacked sufficient health literacy to navigate the system.[52] Importantly, this means that simply providing brochures and written information is not health education, and is unlikely to change health behaviour.

A patient's health literacy typically improves through self-education and contact with health providers.[53]

In practice

A patient-centred consultation involves assessing a patient's clinical signs and symptoms, as well as their thoughts, fears, preferences and expectations, and their social context. This ensures a complete understanding of the individual who is living with type 2 diabetes.

From a position of mutual understanding, management plans can then be developed with the patient, and tailored to specifically meet their needs, values and choices. Studies show that people with diabetes are more likely to engage actively in self management and achieve optimal health outcomes if plans are person-centred.[54]

Many of the assessments discussed in these guidelines are performed informally during a routine consultation. However, systems should be developed within the practice to allow appropriate assessment, review and management of individual patients.

Assess health literacy status

A patient's literacy and numeracy skills affect their capacity for self management and what resources they will need. Literacy and numeracy skills are not always obvious, and GPs may worry that attempting to evaluate them will be uncomfortable for patients. The evidence in the literature does not support this concern.[55]

Organisations such as Diabetes Australia provide self-management education and support programs, peer support programs, mental health and diabetes programs, culturally and linguistically appropriate education, and information in several languages. They also have resources to help patients with low literacy skills.

Determine priorities for management

Discover what areas are affecting the patient's quality of life in the context of comorbidities and life expectancy. Determine the management priorities, focusing on specific interventions (including those chosen by the patient) that have the most impact on the individual and will form the basis of their continuing care.

Encourage enrolment in structured programs

Structured diabetes care programs and evidence-based structured self-management training programs are available.

Refer to Appendix B. Structured patient-centred care plan – Examples of a General practice management and Patient care plan for a template of a General practice management plan (GPMP; structured patient-centred care plan).

5.2 A structured diabetes care program consistent with the Chronic Care Model

Recommendations	Reference	Grade*
Care should be aligned with components of the chromic care model (CCM) to ensure productive interactions between a prepared proactive practice team and an informed activated patient	56 American Diabetes Association, 2016	A
When feasible, care systems should support team-based care, community involvement, patient registries and embedded decision-support tools to meet patient needs	56 American Diabetes Association, 2016	B
Treatment decisions should be timely, and based on evidence-based guidelines that are tailored to individual patient preferences, prognoses and comorbidities	56 American Diabetes Association, 2016	B

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

The goal of a structured care program is to increase the quality of life for people with diabetes. Structured care means having all the necessary aspects of the required care in place.

The structure of each diabetes care program will vary depending on the local circumstances and the needs of the patient. There is good evidence to support patient access to a variety of healthcare providers.[17]

Access to, and care delivery by, different healthcare providers allows the patient to benefit from a broad perspective on their health and wellbeing. There are some team roles that fit into most patients' programs, but whatever the composition of the team, care needs to be organised and delivered systematically.

Multidisciplinary care (Figure 2) also covers gaps in care that may be apparent to one healthcare provider, but go unnoticed by another. For example, recognising a patient's social difficulties may be detected during an educator evaluation or by a practice nurse rather than during a routine medical consultation.

Figure 2. Potential members of the multidisciplinary diabetes care team
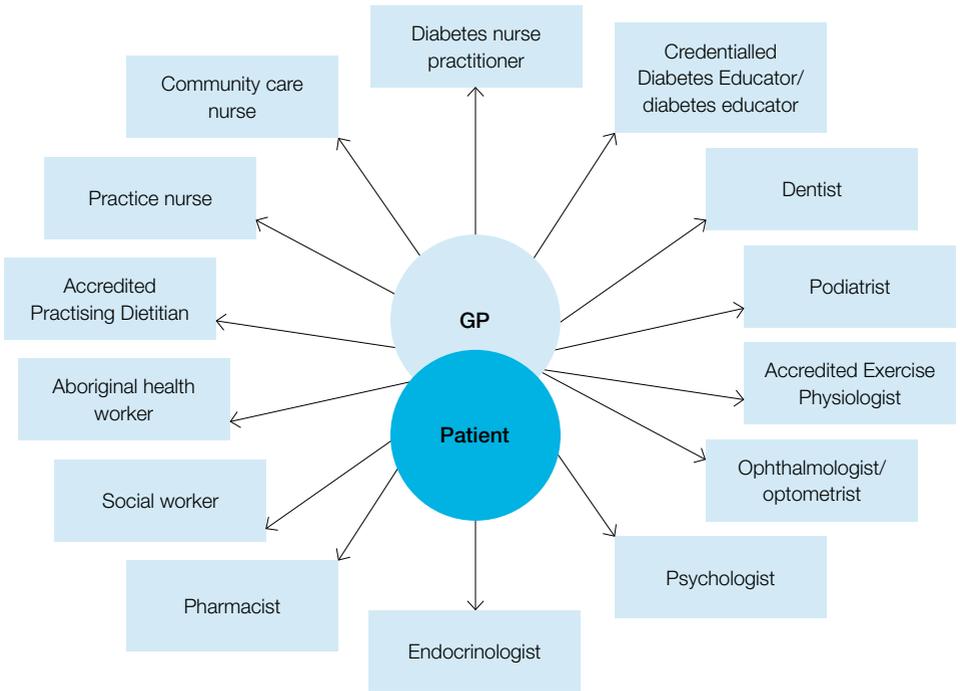
Registered and practice nurses within a general practice can provide an administrative and a clinical role. Practice nurses often manage the diabetes register, structured care-and-recall system, as well as provide a clinical assessment before the GP sees the patient. Practice nurses have an important role in team-based care processes, including motivational interviewing, education activities and support for lifestyle modification. These can be facilitated and enabled upon GP assessment and recommendation. This enables healthcare efficiency and allows the GP to focus on any identified problems. Practice nurses can also act as practice liaison, and facilitate rapid access to GP care in the event of a clinical problem.

ABORIGINAL AND TORRES STRAIT ISLANDER POINT

Involvement of an Aboriginal health worker, or an Aboriginal liaison officer, or Indigenous outreach worker or care coordinator is essential in the care of Aboriginal and Torres Strait Islander peoples.

5.3 Patient education and self management

Recommendations	Reference	Grade*
All people with type 2 diabetes should be referred for structured diabetes patient education	42 NHMRC, 2009	A
Diabetes education should be delivered in groups or individually	42 NHMRC, 2009	A
Diabetes education should be culturally sensitive and tailored to the needs of socioeconomically disadvantaged populations	42 NHMRC, 2009	B

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Education to support self management is an integral part of diabetes care. Patients and their carers should be offered a structured, evidence-based education program at the time of diagnosis, with an annual update and review.[57]

Educating people with diabetes about their condition and its treatment will assist in self management.[58–60]

In addition to the team members mentioned in Figure 2, patients can obtain further education and support through Diabetes Australia and the NDSS, and their state or territory diabetes organisation. More information is available at www.diabetesaustralia.com.au and www.ndss.com.au

Multiple online support and education programs may be available for patients who are unable to access face-to-face group meetings. However, there are few studies on the individual effectiveness of these programs. More information is available at www.diabetesaustralia.com.au and www.adea.com.au/members/adea-website

Self management

Self management involves the person with diabetes working in partnership with their carers and health professionals so they can:

- understand their condition and various treatment options
- contribute to, review and monitor a plan of care (eg care plan)
- engage in activities that protect and promote health
- monitor and manage the symptoms and signs of the condition
- manage the impact of the condition on physical functioning, emotions and interpersonal relationships.

Identifying barriers to self management is important when developing a management plan with the patient. Issues around cognition, physical disability, mental health, health literacy, socioeconomic constraints, location and access to services can have an impact on the ability of the person to self-manage their diabetes.[61]

6. *Lifestyle modification*

Type 2 diabetes after initial diagnosis in some patients can usually be managed with diet and exercise alone.[62,63] Lifestyle modification continues to play an important role in glycaemic control and managing CVD risk in more advanced stages of type 2 diabetes, and may be supported by allied health and specialist support services.

6.1 Physical activity

Recommendations	Reference	Grade*
People with type 2 diabetes of all ages benefit from accumulating 30 minutes or more of moderate physical activity on most, if not all, days of the week	64 Briffa T et al, 2006	B
Exercise and physical activity (involving aerobic and/or resistance exercise) should be performed on a regular basis	65 SIGN, 2014	D

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Exercise is regarded as one of the cornerstones for diabetes management, and while generic physical activity can have a favourable impact on improving glycaemic control, reducing CVD risk and reducing overall mortality,[66, 67] more specialised and individualised exercise prescription can achieve superior benefits.[66] Aerobic exercise has been shown to achieve a similar reduction on HbA1c as either metformin or a sulphonylurea (ie a reduction of 0.73% with exercise and 0.9% with single medication). Resistance training has a smaller effect. For more information on exercise and type 2 diabetes, visit www.racgp.org.au/your-practice/guidelines/handi/interventions/cardiovascular/exercise-for-type-2-diabetes

Regular physical activity improves metabolic control, reduces CVD risks and can reduce the risk of developing type 2 diabetes.[68] Low-level aerobic exercise (eg brisk walking for half an hour per day) and physical resistance training improves glucose tolerance, energy expenditure, feeling of wellbeing and work capacity, and improves BP, lipid profiles and mood.

In practice

The goal is for patients to undertake aerobic training that brings the heart rate up to 55–69% of maximum (208 – 0.7 x age [years] = maximum beats per minute)[69,70] for a minimum of 30 minutes on most, if not all days of the week (≥150 minutes/week). This establishes and maintains fitness and aerobic capacity.

Evidence indicates that people with diabetes, IFG and/or IGT should aim for a minimum of 210 minutes per week of moderate intensity exercise or 125 minutes per week of vigorous intensity exercise, and no more than two consecutive days without training. For more information on resistance training, visit www.racgp.org.au/your-practice/guidelines/handi/interventions/cardiovascular/exercise-for-type-2-diabetes

Note that setting short-term, gradually increasing goals may assist the patient in achieving goals. Evidence suggests that multidisciplinary and integrated care teams are best practice in the management of type 2 diabetes, preventing and decreasing the impact of complications and comorbidities, resulting in healthcare cost savings.[67] Group-based training that facilitates self management in people with type 2 diabetes is effective in improving FBG levels, HbA1c and diabetes knowledge, and in reducing systolic blood pressure (SBP) levels, body weight and the requirement for diabetes medication.[66]

People requiring insulin or those treated with sulphonylureas need to be aware of potential delayed effects of physical activity on BGLs – in particular delayed hypoglycaemia 6–12 hours after cessation of the activity.

People with diabetes need to be advised to moderate or cease their activity if they develop cardiovascular symptoms or feel unwell. Patients with claudication need to be encouraged to continue physical activity under appropriate clinical supervision.

When advising on physical activity, the GP should explain the:

- risks and benefits of physical activity for the individual
- importance of varying intensity of exercise levels
- importance of following the chest pain/discomfort and/or diabetes symptom management plan.[64]

Clinical advice should be given to stop physical activity if the patient experiences symptoms of hypoglycaemia and to discontinue further physical activity until reviewed by their GP.

General physical activity safety advice for people with diabetes:

- Instruct patients to check their BGL before, during and after prolonged physical activity if using insulin or sulphonylureas. Additional carbohydrate foods and medications adjustments may be required depending on the patient's BGLs.

Advise patients that if their pre-exercise BGL is <5 mmol/L, they are at risk of a hypoglycaemic episode during or after exercise, and to have access to additional carbohydrates as per the advice of their CDE or APD. Delayed hypoglycaemia may occur up to 24 hours post-exercise.

- Advise patients on how to recognise, prevent or manage hypoglycaemic events, including potential post-exercise hypoglycaemia (ie need for carbohydrates or medication adjustment).

- Advise patients to carry a rapid-acting glucose source at all times (eg jelly beans, or glucose gel/drink).

- Advise patients to wear correct supportive footwear – especially if there is neuropathy, vascular disease, abnormal foot structure or previous foot ulcer(s), in which case the advice of a podiatrist with an interest in high-risk feet should be sought. This advice would also include the appropriateness of 'jolting' exercises such as running, skipping and jumping.

- Advise patients to check their feet daily and after physical activity for blisters, warm areas or redness.

When prescribing a physical activity program, the GP should be aware of the following:

- A careful history should be taken.

- Special attention needs to be paid to exertion-induced symptoms, chest or abdominal discomfort, claudication or syncope.

- People with type 2 diabetes frequently have silent macrovascular disease.

- For patients with hypertrophic obstructive cardiomyopathy (HOCM), heavy weight-lifting and high-intensity aerobic exercise are not recommended.*

- For patients with long QT syndrome, exercise may trigger a cardiac arrhythmic event.*

- Vigorous exercise is contraindicated for those with proliferative retinopathy, and for three months after laser retinal treatment.*

- Exercise may be relatively contraindicated in patients with peripheral neuropathy, a history of recurrent falls or uncontrolled hypertension.*

- Recommendation for referral to an AEP should be considered.

*From The Royal Australian College of General Practitioners. HANDI interventions – Exercise: Type 2 diabetes. East Melbourne, Vic: RACGP, 2015. Available at www.racgp.org.au/your-practice/guidelines/handi/interventions/cardiovascular/exercise-for-type-2-diabetes

Screening with a stress electrocardiogram (ECG) is not indicated in asymptomatic individuals, but any symptoms suggestive of CVD need to be actively investigated.

ABORIGINAL AND TORRES STRAIT ISLANDER POINT

Many Aboriginal and Torres Strait Islander peoples are involved in physically demanding sporting and cultural activities, although, overall, Aboriginal and Torres Strait Islander peoples are not more physically active than non-Indigenous Australians.

GPs should be aware of activities that are affordable, appropriate and accessible for their Aboriginal and Torres Strait Islander patients, which may be run by local community groups.

Aboriginal and Torres Strait Islander peoples may prefer exercise and physical activities that are culturally, socially and economically meaningful. Such activities or other cultural activities may not be described as 'exercise' or 'physical activity', yet be of significant health and social benefit. A careful history in the context of a trusting doctor–patient relationship may bring about better understanding and opportunity.

6.2 Diet

Recommendations	Reference	Grade*
Consumption of cereal foods (especially three serves a day of wholegrains) is associated with reduced risk of type 2 diabetes	71 NHMRC, 2013	B
Consumption of at least one and a half serves of dairy foods (eg milk, yoghurt, cheese) per day is associated with reduced risk of type 2 diabetes	71 NHMRC, 2013	C

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Most of the burden of disease due to poor nutrition in Australia is associated with eating too much energy-dense and relatively nutrient-poor foods, and eating too few micronutrient-dense foods, including vegetables, fruit and wholegrain cereals.

Composition of a healthy diet

The *Australian dietary guidelines* promotes healthy eating patterns, emphasising a range of nutrient-dense foods in appropriate portion sizes.[71]

- Enjoy a wide variety of nutritious foods from these core food groups every day:
  - plenty of vegetables, including different types and colours, and legumes/beans
  - fruit (consumption of fruit is not associated with risks of type 2 diabetes)
  - grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties, such as breads, cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa and barley
  - lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans
  - milk, yoghurt, cheese and/or their alternatives, mostly reduced fat (reduced fat milks are not suitable for children under two years of age).
- Drink plenty of water.
- Limit intake of foods containing saturated fat, added salt, added sugars and alcohol:
  - Restrict foods high in saturated fat such as many biscuits, cakes, pastries, pies, processed meats, commercial burgers, pizza, fried foods, potato chips, crisps and other savoury snacks.
  - Limit foods and drinks containing added salt.
  - Avoid foods and drinks containing added sugars such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks.
  - Alcohol intake should be as per the Australian dietary guidelines. For women who are pregnant, planning a pregnancy or breastfeeding, not drinking alcohol is the safest option.

Reproduced with permission from the National Health and Medical Research Council. Australian dietary guidelines. Canberra: NHMRC, 2013. Available at www.nhmrc.gov.au/guidelines-publications/n55 [Accessed 29 August 2016].

Two other key themes are eating for cardiovascular protection, and glycaemic management and meal planning.

Eating for cardiovascular protection

Consultation with an APD will allow individualised advice on CVD risk reduction with healthy food choices to be provided. A variety of eating patterns are acceptable for the management of metabolic control. However, personal preferences for cultural, religious and economic preferences should also be considered.[72] One such dietary choice is the Mediterranean diet, which is associated with a lowering of morbidity and mortality for some chronic diseases, including CVD.[73] In persons with high CVD risk, the Mediterranean diet reduced CVD events when compared to a low-fat diet.[74]

Glycaemic management and meal planning

Evaluation of current dietary intake and the eating patterns of an individual is an initial critical step and should be recommended in all people to support the management of type 2 diabetes. All patients should be offered and encouraged to seek advice on medical nutrition therapy by referral to an APD. The APD can help support a person living with diabetes to address the core themes around nutrition such as promoting healthy eating patterns and, where appropriate, healthy body weight (loss) with reduction in energy intake (portion control and type of food).

To influence the glycaemic response after eating, the amount and quality of the carbohydrate eaten may be the most important factor. The amount of carbohydrate eaten within a meal should therefore be considered when meal planning. Eating low-glycaemic-load foods instead of higher glycaemic-load foods may modestly improve glycaemic control.[75] Low glycaemic index (GI) foods include dense wholegrain breads, steel-cut oats, lower fat milk and yoghurt, minimally processed (eg wholegrain, low GI) breakfast cereals, pasta, Doongara rice, legumes and most fruits. Intake of high carbohydrate, low-nutrient-dense foods such as soft drinks, cakes and lollies should be confined to infrequent, small amounts to reduce the risk of weight gain and a worsening cardiometabolic profile.[75] The total amount of carbohydrate consumed (compared with other macronutrients or GI of the meal) may be the major dietary factor contributing to high post-prandial BGLs.[76]

There is evidence that nutrition education may be particularly important for the prevention of hypoglycaemia in people with type 2 diabetes on insulin or oral glucose lowering medications that may cause hypoglycaemia (eg sulphonylureas). Consistency in carbohydrate intake, and spacing and regularity of meal consumption may help some patients manage BGL and weight. Inclusion of snacks as part of a person's meal plan should be individualised and should be balanced against the potential risk of weight gain and/or hypoglycaemia.[77]

In practice

Dietary habit changes are often slow and incremental. There is no need for a 'special' diet for diabetes, just the requirement to follow a sensible, balanced eating plan.

Keep advice simple and educate patients about healthy food choices rather than on unhealthy food choices or what they should not eat. All sugars do not need to be eliminated. A small amount of sugar as part of a mixed meal or food (eg one teaspoon of sugar/honey added to breakfast cereal) may not adversely affect the blood glucose level. Small amounts of added sugar as part of a high-fibre, modified-fat meal plan increases the choice of food available and may aid adherence. Foods naturally high in sugars such as fruit and dairy do not need to be avoided.

Consider recommending/referring to the Eat for Health website (www.eatforhealth. gov.au), which is easy to access and its recommendations easy to implement. Referral to an APD or a CDE will support implementation/reinforcement of these recommendations. To find an APD, visit www.daa.asn.au

For basic dietary advice, visit www.diabetesaustralia.com.au/eating-well

ABORIGINAL AND TORRES STRAIT ISLANDER POINT

There is evidence that Aboriginal and Torres Strait Islander communities in urban and remote regions face significant access barriers to nutritious and affordable food. Nutritious food tends to cost more, and requires refrigeration and preparation, so lack of food security may affect the choice of glucose-lowering medications. Food choices can be significantly altered when people have access to appropriate foods and education about nutrition.

GPs should make themselves aware of local community initiatives for the supply of fresh fruit and vegetables at affordable prices. In some areas, these include arrangements with farmers' markets or local community gardens. Information in regards to Aboriginal and Torres Strait Islander peoples nutrition is available at:

• Australian Bureau of Statistics, www.abs.gov.au/ausstats/abs@.nsf/Lookup/ by%20Subject/4727.0.55.005~2012-13~Main%20Features~Key%20 Findings~1

• Australian Institute of Health and Welfare, www.aihw.gov.au/uploadedFiles/ ClosingTheGap/Content/Publications/2012/ctgc-rs09.pdf

• Department of Health, www.health.gov.au/internet/publications/publishing. nsf/Content/oatsih-evidence-socialhealth-toc~nutrition

6.3 Weight

Recommendations	Reference	Grade*
Adults with impaired fasting glucose, impaired glucose tolerance or diabetes can be strongly advised that the health benefits of 5–10% weight loss include prevention, delayed progression or improved control of type 2 diabetes	78 NHMRC, 2013	A
For adults with body mass index (BMI) >40 kg/m^2, or BMI >35 kg/m^2 and comorbidities that may improve with weight loss, bariatric surgery may be considered, taking into account the individual situation	78 NHMRC, 2013	A
Use BMI to classify overweight or obesity in adults	78 NHMRC, 2013	B
For adults, use waist circumference, in addition to BMI, to refine assessment of risk of obesity-related comorbidities	78 NHMRC, 2013	C

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Excess weight in individuals usually results from a prolonged period of energy imbalance. However, the causes of overweight and obesity are complex. Older people with diabetes may also be at risk of malnutrition. Diet and physical activity are central to the energy balance equation, but are directly and indirectly influenced by a wide range of social, environmental, behavioural, genetic and physiological factors, the relationships between which are not yet fully understood. Increasing physical activity regardless of weight loss may reduce CVD risk factors, improve functional mobility in older people and reduce HbA1c by ~0.6% in adults with type 2 diabetes.[78] Table 4.4 in *Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia*[78] (www.nhmrc.gov.au/guidelines-publications/n57) lists common medications associated with weight gain at 12 weeks of commencement of a weight management diet.[79]

It is important to encourage some degree of healthy weight loss, except where there are other associated risks (eg frail and elderly, or those with psychologically related eating disorders). A healthy body weight is often not achievable and setting this as a goal discourages patients from attempting any dietary change. Many studies suggest that weight loss of 5–10% will improve glycaemic control.[80, 81] Section 2.2.3 in the above clinical practice guidelines discusses the importance of psychological issues influencing weight management.[78]

A recent multi-centre, randomised clinical trial (Action for Health in Diabetes [Look AHEAD]) provided evidence that intensive lifestyle intervention focusing on weight loss did not result in a significant reduction in cardiovascular events in overweight or obese adults with established type 2 diabetes.[82] This was despite greater reductions in HbA1c and greater initial improvement in fitness and all CVD risk factors, except for low-density lipoprotein cholesterol levels.

In practice

Weight assessment

Assessing weight is typically done using BMI. Note that BMI is a difficult parameter to standardise between different population groups.

For those of European descent, a healthy BMI is 18.5–24.9 kg/m^2, overweight is 25–29.9 kg/m^2 and obese is ≥30 kg/m^2.[78] Different classification criteria may apply to other population groups. Some groups may have equivalent levels of risk at a lower BMI (eg people of Asian origin) or higher BMI (eg Torres Strait Islander and Maori peoples).[83]

It is advisable to also assess waist circumference (in cm) as this is a good indicator of total body fat and useful predictor of visceral fat. Waist circumference of ≥94 cm in men and ≥80 cm in women conveys increased risk; ≥102 cm in males and ≥88 cm in females conveys high risk.[84] As with BMI, these values may differ for other population groups.[78] Measuring waist circumference in patients with a BMI >35 kg/m^2 may not add any further to predictive disease risk classification.[85]

Weight management

Modest weight loss (5–10%) may provide clinical benefits for those with type 2 diabetes, especially early in the disease process.[75] Loss of body weight often results in improved glycaemic control, BP and lipid profiles. Sustained weight reduction of approximately 5 kg is associated with a reduction in HbA1c of approximately 0.5–1%.[78] In adults with BMI <35 kg/m^2 with dysglycaemic states or hypertension, weight loss of at least 2–3 kg achieved with lifestyle interventions may result in a clinically meaningful reduction in BP (an average SBP of 4.5 mmHg and DBP of 3–3.5 mmHg).[78]

In overweight or obese people with diabetes, a nutritionally balanced, energy-reduced diet should be recommended if a lower, healthier body weight is to be achieved and maintained as part of a multicomponent lifestyle intervention (including healthy eating, physical activity and support for behavioural change).

Very low energy diets are a useful intensive medical therapy for supporting rapid weight loss when used under medical supervision.[78] This involves regular appointments with appropriate health professionals aimed at supporting the progress of the individual. These diets may be considered in adults with a BMI >30 kg/m^2, or with BMI >27 kg/m^2 and obesity-related comorbidities, taking into account each individual situation.

Pharmacotherapy is licensed by the TGA for weight management, including for patients with diabetes, but is currently not PBS reimbursed. All agents are to be used as adjuncts to dietary changes and physical activity improvement. Agents available include phentermine (a sympathomimetic amine), orlistat (an inhibitor of intestinal lipase) and liraglutide (a glucagon-like peptide-1 receptor agonist [GLP-1 RA]). Each agent has the potential for significant clinical side effects and contraindications associated with its use, and require careful clinical risk–benefit assessment when applied in practice. Refer to the TGA website for more information (www.tga.gov.au).

Taking into account each individual situation, bariatric surgery may be considered for people with a BMI >30 kg/m^2 who have suboptimal blood glucose levels and are at increased CVD risk and who are not achieving recommended targets with medical therapy.[78] GPs should assess the appropriateness of surgery for each individual patient and provide information on the risks, benefits and appropriateness of the type of procedure. Bariatric surgery performed in a high-volume specialist centre with an experienced surgical team may have the lowest risks and GPs should liaise with a specialised surgical team if there are concerns.[78] Bariatric surgery, when indicated, should be included as part of an overall clinical pathway for adult weight management that is delivered by a multidisciplinary team (including surgeons, APDs, nurses, psychologists and physicians), and includes planning for surgery, and continuing follow-up.[78]

6.4 Smoking cessation

Recommendations	Reference	Grade*
Smoking cessation should be a major focus of the management of people with smoking-related diseases	86 RACGP, 2011	A
All smokers should be offered brief advice to quit smoking	86 RACGP, 2011	A

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Smoking is associated with an increased risk of type 2 diabetes in men and women,[87] and smoking negatively affects glycaemic control (eg smokers with type 2 diabetes need larger doses of insulin to achieve control similar to that of those who do not smoke).[88]

Patients with diabetes who smoke also further increase their risk of CVD, peripheral vascular disease and neuropathy (and progression of neuropathy). Smoking also increases the risk associated with hospitalisation for surgery.[86]

In practice

The importance of smoking cessation in those with or at risk of type 2 diabetes cannot be overstated.

Guidelines for smoking cessation and a pharmacotherapy treatment algorithm are available for download from the RACGP website (www.racgp.org.au) and in the RACGP's *Supporting smoking cessation: A guide for health professionals*.[86]

ABORIGINAL AND TORRES STRAIT ISLANDER POINT

Resources and strategies for smoking cessation for Aboriginal and Torres Strait Islander peoples are available at the Centre for Excellence in Indigenous Tobacco Control (CEITC; www.ceitc.org.au). Specific support for Aboriginal and Torres Strait Islander peoples is also provided by Quitline.

In the absence of contraindications, smokers who have evidence of nicotine dependence should be offered pharmacotherapy if they are motivated to stop smoking. The choice of pharmacotherapy is based on clinical suitability and patient choice.

There is a lack of safety data on the use of varenicline or bupropion in diabetes. However, if diabetes is well controlled with insulin or oral glucose-lowering medication, 150 mg of bupropion once daily may be prescribed. If the diabetes is poorly controlled, nicotine replacement therapy is considered preferable.[86]

6.5 Alcohol consumption

Recommendations	Reference	Grade*
People with diabetes can take alcohol in moderation as part of a healthy lifestyle, but should aim to keep within the target consumption recommended for people without diabetes	65 SIGN, 2014	B

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Alcohol affects the management of type 2 diabetes through its effects on diet and control of BGLs. Alcohol interferes with the action of insulin, insulin secretagogues and glucagon, thereby increasing the risk of hypoglycaemia in people with type 2 diabetes who take these medications.[71] Alcohol can lower BGLs and reduce awareness of hypoglycaemia. Alcohol and hypoglycaemia have independent but additive adverse effects on cognitive function.[65]

Reduction in energy intake, which may involve reducing alcohol intake, may be important for managing weight in the overweight or obese person as part of diabetes management.[89]

In practice

Patients should be educated on how to avoid hypoglycaemia when drinking alcohol. The current *Australian guidelines to reduce health risks from drinking alcohol* (www. nhmrc.gov.au/_files_nhmrc/publications/attachments/ds10-alcohol.pdf) recommends ≤2 standard drinks (20 g) per day for men and women. Low-alcohol beers are a better choice than ordinary or diet beers.[89] The carbohydrate content of low-carbohydrate beer is not significantly less than full-carbohydrate beers and the alcohol content is often full strength.

For healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury. Drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion.[89] It is recommended that people with diabetes abstain from alcohol if they plan to drive.[90]

7. The person with diabetes – Assessment

7.1 Understanding the person – Initial assessment

The aim of the initial assessment is to provide a whole-person evaluation, and determine and understand which factors are affecting the patient's health and quality of life. Many people with diabetes are dealing with multiple medical conditions (not necessarily related to diabetes) and family, work or financial stresses. Some are also dealing with other factors including poor sleep, smoking, lack of exercise and pain that affects their priorities for management.[91] This can have an impact on the individualised approach to diabetes management and outcomes.

A detailed assessment, including appraisal of CVD risk and end-organ damage, should be made at first diagnosis.

ABORIGINAL AND TORRES STRAIT ISLANDER POINT

In Aboriginal and Torres Strait Islander patients, the development of rapport may take precedence over a detailed assessment in a single consultation. An assessment could be done over several visits.[92]

Developing a doctor–patient (or patient–healthcare worker) relationship based on trust and respect is the best way of overcoming cultural barriers and ensuring effective care in the long term.

History

Ascertain symptoms supportive of a diagnosis of diabetes, including a history regarding the onset of symptoms (including obstetric history).

Factors in a specific diabetes enquiry:

- Symptoms of hyperglycaemia: polyuria, polydipsia, polyphagia, weight loss, nocturia.
- Sequelae of hyperglycaemia and complications of diabetes: malaise/fatigue, cardiovascular symptoms, neurological and autonomic symptoms, altered vision, bladder and sexual function, foot and toe numbness and pain, and any recurrent infections (especially urinary and skin with delayed wound healing) and gastrointestinal dysfunction (such as gastroparesis and nausea). Include specific enquiry about dental hygiene and gingivitis.

Enquire about specific issues that may provide the aetiology or predisposition
to diabetes, including:

- age, family history, cultural group, overweight, physical inactivity, hypertension
- obstetric history of macrosomic babies or gestational diabetes
- medication causing hyperglycaemia (refer to Chapter 8. Managing glycaemia)
- personal or family history of haemochromatosis
- personal or family history of other autoimmune diseases (eg hypothyroidism
  or hyperthyroidism)
- pancreatic disease, Cushing's disease
- obstructive sleep apnoea.

Perform a general health enquiry for:

- presence of risk factors for diabetes complications or known comorbidities
  (including personal or family history of CVD), smoking, hypertension, dyslipidaemia,
  and also include a history of past or current mental health problems such as
  depression
- health literacy and knowledge about diabetes and related complications
- emotional and mental health (use the Patient Health Questionnaire-2 [PHQ-2]
  tool to assess depressive symptoms and problem areas in diabetes [PAID] tool to
  assess diabetes-specific distress – refer to Appendix C. Problem areas in diabetes
  questionnaire and Appendix D. Patient health questionnaire-2 tool)
- living situation (eg alone/with family, employment, financial worries).

It is important to confirm the patient's immunisation currency. The following
vaccinations are recommended for patients with type 2 diabetes:

- Influenza – once per year
- Pneumococcal
  - Non-Indigenous Australians: <65 years of age – single dose and revaccinate at
    65 years of age or after 10 years, whichever is later; >65 years of age – single
    dose and revaccinate after five years
  - Aboriginal and Torres Strait Islander peoples: <50 years of age – single dose
    and revaccinate at 50 years of age or after 10 years, whichever is later; >50
    years of age – single dose and revaccinate after five years
- Tetanus – booster at 50 years of age (unless booster has been given within 10
  years). Tetanus vaccination in adults is best given with a multivalent vaccine such
  as dTpa.

- Other vaccinations as required on an individual basis according to the *Australian immunisation handbook,* 10th edn (www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home).

Full physical assessment

This should be focused on determining current overall health status to provide information that helps establish management strategies including treatment options.

Assess cardiovascular status and risks

Visceral fat accumulation and obesity increase the risks of developing diabetes, and complications arising from diabetes and comorbidities such as hypertension, dyslipidaemia and pancreatitis.

Hypertension is more common in diabetes, but autonomic neuropathy conversely may arise and lead to postural hypotension. Macrovascular disease may be evident on peripheral arterial examination including the presence of carotid bruits. Dysrhythmias may indicate the presence of existing CVD.

Assess:

- weight: BMI = weight (kg) divided by height^2 (m^2)
- waist: waist circumference (cm)
- BP, central and peripheral vascular systems
- absolute CVD risk assessment (this may require calculation and investigations)
- for symptoms of ischaemic disease or dysrhythmia, in which case an ECG may be considered.

Assess for the presence or absence of diabetes complications

Eyes: Visual acuity (with correction); screen for retinopathy (retinal photography or examine with pupil dilation and ophthalmoscope) – it is prudent to assess for retinopathy and maculopathy with diabetes, as they are the leading causes of blindness and may occur asymptomatically.

Feet: Stratify the risk of developing foot complications (refer to Section 10.5. Foot complications) – sensation and circulation, skin condition, pressure areas, interdigital problems, abnormal bone architecture.

Peripheral nerves: Tendon reflexes, sensation – touch (eg 10 g monofilament) and vibration (eg 128 Hz tuning fork) – existence of peripheral neuropathic changes indicates the onset of microvascular diabetes complications.

Urinalysis: Testing for albumin, ketones, nitrites and/or leucocytes. The presence of proteinuria on clinic testing may necessitate further albumin-to-creatinine ratio (ACR) investigation to exclude existing diabetes nephropathy.

- Microalbuminuria (http://patient.info/doctor/microalbuminuria) ACR ≥2.5 mg/mmol (men) or ≥3.5 mg/mmol (women), or albumin concentration ≥20 mg/L.
- Proteinuria (http://patient.info/doctor/proteinuria-pro) ACR ≥25 mg/mmol (men) or ≥35 mg/mmol (women; refer to Figure 5).
- Elevated leucocytes and nitrites may indicate genitourinary infection, which occurs at a higher prevalence and severity in diabetes.

Investigations

To help determine CVD risks, and as clinically indicated, obtain levels of:

- urine microalbumin, calculated estimated glomerular filtration rate (eGFR)
- lipids – low-density lipoprotein-cholesterol (LDL-C), high-density lipoprotein-cholesterol (HDL-C), total cholesterol, triglycerides
- HbA1c (mmol/mol or %).

7.2 What needs ongoing assessment?

The purpose of ongoing structured evaluation is to determine the impact of care and diabetes on the life of the person with diabetes. It is also to individually assess the impact of clinical management by assessing the person's diabetes goals and risk factors. Review the use of medication every three or six months using the principles of the 'Stop rule' (refer to What if medication is not working – The 'Stop rule' under Section 8.2.2. Glucose-lowering agents).

History

Review the patient's overall sense of wellbeing, ability to cope and self-manage with the diagnosis of diabetes, and what effect this is having on the person's life. Establish their level of health literacy about their diabetes, and what to do in the event of problems arising.

Review lifestyle interventions, particularly SNAP profiles: (S)moking persistence or relapse, (N)utrition and diet, (A)lcohol intake and (P)hysical activity.

Enquire about possible diabetes complications as well as known comorbid conditions including psychological stress and/or depression (refer to Appendix C. Problem areas in diabetes questionnaire and Appendix D. Patient health questionnaire-2 tool).

Enquire about intercurrent illnesses (eg urinary tract infections, influenza, thyroid disease) that may alter the degree of control. Urinary tract infections are common in patients with diabetes, especially in females.

Enquire about symptoms of hypoglycaemia if the patient is on insulin and/or oral agents that can cause hypoglycaemia.

Enquire about the burdens of care/self management. Has the person been referred or received structured diabetes self-management education?[42] Does the person experience any problems with medication taking, including side effects, forgetting or sometimes intentionally not taking medications as recommended? Consider alternative regimens or problem-solving with the patient if problems are significant.

Examination

To help re-evaluate therapeutic goals and assess for complications, check weight and waist, height (children and adolescents), BP, feet examination (refer to Section 10.5. Foot complications) if new symptoms or at risk (eg neuropathy ± peripheral vascular disease). Assess a patient's record of SMBG testing (if utilised). Individually assess the need for further re-examination dependent on individual risk factors. For example, BP may need re-evaluation in two months if elevated at systolic 140–159 mmHg/diastolic 90–99 mmHg, whereas BP at 160–179 mmHg systolic/100–109 mmHg diastolic may need reassessment within one month.[93]

Routine investigations are best organised before the review appointment.

To determine measurable diabetes goals for the individual patient:

- Measure HbA1c as needed on an individual basis – this may be up to every third month (maximum) in a newly diagnosed patient, patients undergoing therapeutic changes or those outside of recommended ranges; stable patients at agreed targets may need less-frequent interval testing.
- Base further investigations on re-evaluated clinical symptoms and history (eg a urine assessment may be considered, or investigation of emotional issues including depressive symptoms, diabetes-specific distress, or other diabetes-related issues suspected or identified in earlier consultations).

Refining the management plan

Review the goals and individual targets with the patient to identify specific areas for ongoing or interval therapeutic review.

Patient support – refer to structured self-management education (eg CDE). Does other allied health intervention need to be considered (eg psychologist, APD)?

Medication/therapy choices – adjustment of agent, dose, combinations, enquire about symptoms of hypoglycaemia. Review use of medication using the principles of the 'Stop rule' every three or six months.

Complication management – specific intervention/support/referral when indicated.

7.3 What should be evaluated yearly?

The annual review is a time for more detailed assessment, updating the problem priority list, re-establishing goals and checking agreed arrangements for management.

As there is an increasing trend towards involving specialist allied health professionals, the yearly visit is a good opportunity to coordinate follow-up.

Annual review

A full system review checking for vascular, renal, eye, nerve and podiatric problems is required. An annual review or cycle of care may address the following:

History

- Review issues specific to diabetes:
  – including symptoms of hyperglycaemia, hypoglycaemia and diabetes complications.
- Preventive health issues:
  – smoking
  – nutrition (last contact with APD or CDE)
  – alcohol intake
  – physical activity.
- Adequacy of, or problems with, team care arrangements.
- Patients with diabetes can be assessed for perspectives on adequacy of treatment, quality of life, medication burden, and concerns regarding diabetes such as mental health issues, social isolation/networks and family or work stress. Daily diabetes self care and management can place a considerable burden upon people with diabetes. It is common for people with diabetes, at times, to feel overwhelmed, frustrated, guilty, or to worry about their current and/or future diabetes management and health outcomes.
- Immunisations.

Clinical examination

- Visual acuity. Retinal screening – every two years with no retinopathy, more frequently if abnormal.
- Cardiovascular system, including postural BP, and central and peripheral vascular systems. Calculate or re-evaluate absolute CVD risk assessment.

- Weight, waist, height (children and adolescents).
- Feet examination without shoes – pulses, monofilament check, any foot discomfort.
- Consider assessment of diabetes distress through the use of the PAID[94] questionnaire and depression with the PHQ-2[95] (refer to Appendix C. Problem areas in diabetes questionnaire and Appendix D. Patient health questionnaire-2 tool).

Routine investigations

- Re-evaluate lipid parameters. If the patient has low CVD risk, these tests can be performed every three years. More frequent testing can be justified if the clinical situation varies or if therapeutic changes have been instituted. Some guidelines suggest yearly testing of lipids when the patient is deemed to be at clinically high risk.
- Re-evaluate urine microalbumin annually, unless existing pathology necessitates more frequent testing.
- Based upon a clinical risk assessment, individually assess the need for further investigations such as liver enzyme abnormalities for hepatic steatosis.

Evaluation and management

- Shared decision making – Identify specific clinical areas for focus within the consultation and re-establish patient-specific goals for support and re-evaluation.
- Renew team care planning with identified specific interventions.
- Identify therapeutic management changes and additional education goals with patient involvement.
- Organise appropriate referral where clinically necessary. Some patients may require ongoing specialist or other allied health review. Others will have changed priorities; hence, it is sometimes wise not to commit to referrals too early.

Table 3. Suggested actions and health professionals providing treatment or service

Suggested actions	Team resource – Who?
Ask	
Symptoms	General practitioner (GP)
Goal setting supporting self management	GP
Cardiovascular issues (eg blood pressure [BP] measurement)	GP/practice nurse
Glycaemic control	GP/practice nurse/Credentialled Diabetes Educator (CDE)/diabetes educator (DE)
Assess (inclusive within an annual cycle of care)	
Risk factors for modification	GP/practice nurse/CDE/DE
Weight, height	GP/practice nurse
Cardiovascular disease risk assessment	GP/practice nurse
Foot examination	GP/podiatrist/practice nurse
Presence of other complications, especially hypoglycaemia risk with insulin or sulphonylureas	GP/practice nurse/CDE/DE/ endocrinologist
Psychological status	GP/psychologist
Eye examination	GP/optometrist/ophthalmologist
Dental review	GP/dentist
Consider other assessments where appropriate (eg cognitive impairment, obstructive sleep apnoea)	GP/endocrinologist/specialist (where indicated)
Advise	
Review smoking, nutrition, alcohol, physical activity (SNAP) profiles, including specific issues	GP/registered nurse/CDE/DE
Nutrition	GP/Accredited Practising Dietitian (APD)
Physical activity levels	GP/Accredited Exercise Physiologist (AEP)/physiotherapy
Driving	GP/practice nurse/CDE
Immunisation	GP/practice nurse
Sick day management	GP/practice nurse/CDE
Medication issues	GP/pharmacist/CDE/endocrinologist
Self-monitoring blood glucose	GP/CDE/DE/practice nurse
Insulin/injectable management	GP/CDE/registered nurse/accredited nurse practitioner/endocrinologist
Psychological issues	GP/practice nurse/CDE/DE/psychologist

Assist	
Register for National Diabetes Services Scheme (NDSS)	GP/CDE/nurse practitioner
General practice management plan (GPMP) and Chronic disease management plan	GP/practice nurse
Cultural, psychosocial issues	GP/Aboriginal health worker/social worker/CDE/DE/psychologist
Arrange	
Addition to the practice's diabetes register and recall	GP/practice nurse/practice staff
Organise reviews including pathology and annual cycle of care	GP/practice nurse
Driver's licence assessment	GP/practice nurse/endocrinologist (when indicated)

8. Managing glycaemia

The aim of glycaemic control is to assess each individual patient and balance the role of prevention of glycaemic complications of diabetes while avoiding hyperglycaemia and hypoglycaemia. Overall, managing CVD risk in each patient remains a higher priority than strict glycaemic control.

Refer to Chapter 9. Managing cardiovascular risk.

8.1 Glycaemic monitoring

Recommendations	Reference	Grade*
Glycated haemoglobin (HbA1c) measurement should be used to assess long-term blood glucose control	96 NHMRC, 2009	A
Self-monitoring of blood glucose is recommended for patients with type 2 diabetes who are using insulin where patients have been educated in appropriate alterations in insulin dose (Refer to Self-monitoring of blood glucose under Section 8.2. Medication for examples of instances when self-monitoring of blood glucose may be considered)	65 SIGN, 2014	B
Routine self-monitoring of blood glucose in people with type 2 diabetes who are using oral glucose-lowering drugs (with the exception of sulphonylureas) is not recommended	65 SIGN, 2014	B

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Accuracy and limitations of the HbA1c test

Koenig first proposed the measurement of HbA1c in patients with diabetes as a marker for evaluating long-term control of diabetes in 1976.[97] Over time, this has become a gold standard.

There are potential pitfalls of HbA1c as a measure of long-term diabetes management, and all diabetes clinicians should be aware of natural test variations and conditions that affect HbA1c results.

HbA1c measurement and natural test variation

HbA1c can be measured and reported using two different standards: the National Glycohemoglobin Standardization Program method reported as a per cent of units

(eg 7%) and the newer International Federation of Clinical Chemistry standardisation reported as mmol/mol (eg 53 mmol/mol).

The variability of HbA1c values within Australia is now acceptably low. In a recent Australian study, more than 90% of HbA1c results fell within 6% of the median. A true level of 53 mmol/mol or 7.0% may be reported as anywhere between 49 mmol/mol or 6.6% and 57 mmol/mol or 7.4%.[38]

This variation needs to be considered when monitoring long-term glucose control.

Conditions affecting the HbA1c result

Any condition that shortens erythrocyte survival or decreases mean erythrocyte age will falsely lower HbA1c test results regardless of the assay method used (Table 4).

The presence of abnormal haemoglobin variants can cause unusually high HbA1c (eg HbF, HbE, HbD, HbJ Capetown, Hb Raleigh) or unusually low HbA1c readings (eg HbS, HbC, HbJ, HbG, Hb Ramadan).

Table 4. Other causes of variances to HbA1c

Abnormally low glycated haemoglobin (HbA1c)
- Haemolytic anaemia: congenital (eg spherocytosis, elliptocytosis), haemoglobinopathies, acquired haemolytic anaemias (eg drug-induced such as with dapsone, methyldopa)
- Recovery from acute blood loss
- Chronic blood loss
- Chronic renal failure (variable)

Abnormally high HbA1c
- Iron deficiency anaemia[98]
- Splenectomy
- Alcoholism
- Steroid therapy, stress, surgery or illness in the past three months

Several situations may indicate the presence of a haemoglobinopathy, including when:

- results of SMBG have a low correlation with HbA1c results
- an HbA1c result is discordant with measured laboratory glycaemia
- an HbA1c result is more than 15%
- a patient's HbA1c test result is radically different from a previous test result following a change in laboratory HbA1c measurement methods.

If a haemoglobinopathy is suspected, then a haemoglobin electrophoresis is suggested. Schnedl et al reported a prevalence of abnormal haemoglobin variants of 0.6% among 15,000 HbA1c estimations in a period of over six years.[99]

Reliable HbA1c tests, in which haemoglobin variants do not cause interference, are available. Otherwise, alternative forms of diabetes monitoring such as continual blood glucose estimations, SMBG and fructosamine should be considered for these patients.

Self-monitoring of blood glucose

Self-monitoring in patients with type 2 diabetes is usually recommended:

- for patients on insulin and glucose lowering agents that can cause hypoglycaemia
- when monitoring hyperglycaemia arising from illness (Chapter 11. Glycaemic emergencies and Appendix J. Detailed information on glycaemic emergencies)
- with pregnancy and pre-pregnancy planning
- when changes in treatment, lifestyle or other conditions requires data on glycaemic patterns
- when HbA1c estimations are unreliable (eg haemoglobinopathies).

The method and frequency of monitoring need to reflect individual circumstances and therapeutic aims, and where the person with diabetes and their healthcare providers have the knowledge, skills and willingness to incorporate SMBG and therapy adjustments into diabetes care plans.

A 2012 Cochrane review on the effect of SMBG in patients with type 2 diabetes not using insulin found limited clinical benefit as measured by HbA1c from SMBG. Therefore, routine SMBG for people with type 2 diabetes who are considered low risk and using oral glucose lowering drugs (with the exception of sulphonylureas) is not recommended.[100–104]

In practice

Recommendations	Reference	Grade*
Blood glucose control should be optimised because of its beneficial effects on the development and progression of microvascular complications	96 NHMRC, 2009	A
The potential harmful effects of optimising blood glucose control in people with type 2 diabetes should be considered when setting individual glycaemic targets	96 NHMRC, 2009	A
The general glycated haemoglobin (HbA1c) target in people with type 2 diabetes is ≤53 mmol/mol (≤7%). Adjustments to diabetes treatment should be considered when HbA1c is above this level	96 NHMRC, 2009	A
Targets for self-monitoring of blood glucose levels are 6–8 mmol/L for fasting and preprandial, and 6–10 mmol/L for two hour postprandial	96 NHMRC, 2009	C

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

HbA1c targets and individualisation

The general HbA1c target in people with type 2 diabetes is HbA1c ≤53 mmol/mol (≤7%). Due to the natural variation of HbA1c test results, a target HbA1c of 53 mmol/mol would be achieved by laboratory results being in a range of 48–58 mmol/mol (6.5–7.5%).

All patients with diabetes need to optimise their blood glucose control to improve short-term and long-term health outcomes. However, what is 'optimal' will vary depending on the balance between benefits and risks, and the patient's priorities (Figure 3).

Given the range of diabetes presentations to general practice, there is no single glycaemic target that suits all patients. Targets need be individualised and balanced against patient capabilities and the risk of severe hypoglycaemia, especially among older people.

Control of diabetes symptoms (eg polydipsia, polyuria) can usually be achieved around a HbA1c level of 64 mmol/mol (8%). This does not necessarily mean optimum metabolic control.

More stringent HbA1c targets might be considered in selected patients (eg those with short disease duration, long life expectancy, no significant CVD) if this can be easily and safely achieved without significant hypoglycaemia or other adverse effects of treatment.

Figure 3. Approach to management of hyperglycaemia

Patient/disease features	More stringent	← HbA1c → 7%	Less stringent
Risks potentially associated with hypoglycaemia, other adverse events	Low		High
Disease duration	Newly diagnosed		Longstanding
Life expectancy	Long		Short
Important comorbidities	Absent	Few/mild	Severe
Established vascular complications	Absent	Few/mild	Severe
Patient attitude and expected treatment efforts	Highly motivated, adherent, excellent self-care capacities		Less motivated, non-adherent, poor self-care capacities
Resources, support system	Readily available		Limited

Usually not modifiable   Potentially modifiable

Reproduced with permission from the American Diabetes Association from Inzucchi SE, Bergenstal RM, Buse JB, et al. Management of hyperglycemia in type 2 diabetes: A patient-centered approach: Position statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care 2015;38(1):140–49.

Not everyone benefits from long-term intensive glucose management.

Less stringent HbA1c goals (eg 58–64 mmol/mol [7.5–8.0%] or even slightly higher) are appropriate for patients with a history of severe hypoglycaemia, limited life expectancy, advanced complications, extensive comorbid conditions, and those for whom the target is difficult to attain despite intensive self-management education, repeated counselling, and effective doses of multiple glucose-lowering agents, including insulin.[105]

Targets for self-monitored glycaemic control in type 2 diabetes are shown in Table 5.

Table 5. Targets for self-monitored glycaemic control in type 2 diabetes

Fasting blood glucose (FBG; mmol/L)	Preprandial blood glucose (mmol/L)	Postprandial blood glucose (mmol/L)	Comment
6.0–8.0	6.0–8.0	6.0–10.0	NHMRC values[96]

8.2 Medication

8.2.1 General medication

Recommendations	Reference	Grade*
Care should be taken to address the potential harmful effects of optimising blood glucose control when setting individual glycaemic targets	96 NHMRC,2009	A
Interventions to achieve target glycated haemoglobin (HbA1c) should begin with lifestyle modification followed by pharmacological options selected on the basis of individual clinical circumstances, side effects and contraindications	96 NHMRC,2009	A
Blood glucose control should be optimised because of its beneficial effects on the development and progression of microvascular complications	96 NHMRC,2009	A

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

In addition to lifestyle modification, people with type 2 diabetes may require pharmacotherapy to achieve long-term glycaemic control and to prevent complications of diabetes. There may be a need to commence medication without delay in patients who are symptomatic of hyperglycaemia or having accompanying metabolic dysfunction (eg ketosis), while providing ongoing lifestyle support.

The benefits of management of hyperglycaemia for the prevention of microvascular complications have been demonstrated in randomised clinical trials. BP and lipid-lowering therapy have also been demonstrated in clinical trials to show clear benefits in preventing cardiovascular events and reducing premature mortality.

The choice, order and combination of medications used are based on evidence of improved clinical outcomes, risk of side effects and patient choice/capacity.

In practice

Use of these therapies is associated with risks and other negative effects. These should be taken into consideration when deciding the appropriateness of implementing the treatment recommendations contained in these guidelines. These therapies may be contraindicated in some situations and their use may result in troublesome side effects.

8.2.2 Glucose-lowering agents

Clinical context

Multiple glucose-lowering pharmacotherapies are available (Appendix E. Available glucose-lowering agents).

Algorithms have been designed to help navigate choice. However, applying the principles of patient-centred care may mean that choices made by algorithm are not always appropriate.

The *Australian blood glucose treatment algorithm for type 2 diabetes* (Figure 4) is an evidence-based algorithm developed by the Australian Diabetes Society (ADS) in consultation with all key stakeholders including the RACGP.

Additionally, when analysing combination therapies used in current suggested algorithms for the management of hyperglycaemia, high-quality trials of clinical outcomes may be lacking. The most studied agents include metformin and sulphonylureas. However, randomised controlled trials (RCTs) investigating the safety

of newer agents such as the incretins and sodium glucose co-transporter 2 (SGLT2) inhibitors in patients with high CVD risk are now being reported.[106–108]

Prescribing algorithms suggest multiple ways of combining agents. Always consult the PBS when combining therapy as restrictions and reimbursement may change. Table 6 provides a guide for clinical considerations when choosing diabetes medications.

Appendix F. Table of evidence and properties of glucose-lowering agents provides the evidence and properties of glucose-lowering agents for this algorithm.

What if medication is not working – The 'Stop rule'

At each visit, ask about the patient's understanding of their diabetes and the role of medication in management. Specific enquiry may help decisions on medication efficacy and choices. Symptoms suggestive of hypoglycaemia or other side effects of medication should trigger a clinical examination and review. Symptoms of hyperglycaemia (eg polyuria, polydipsia), fatigue and visual changes may warn of poor adherence to medication by the patient, or indicate that the medication(s) is/are not effective in glycaemic management. Confirmation with an assessment of HbA1c (usually every three months) in relation to the individual's goals may be appropriate. Consideration of the effect of comorbidities (refer Chapter 12. Diabetes multimorbidity and medication complications) or other medication affecting glycaemic control may need to be assessed and managed. Consideration of a home medicines review by the treating pharmacist may also assist assessment of clinical reasons for problems of persistence, side effects with medication or patient concerns.

Practice point

The 'Stop rule' emphasises that before advancing through additional glycaemic-lowering combinations, after evaluating each patient's HbA1c response after three to six months, support the patient to engage in healthy lifestyle choices, assess for comorbidities and complications (eg CVD risk or distress) and then evaluate the need for additional or altered medication/combination therapy.

Figure 4. Australian diabetes algorithm and clinical medication table

First line: Metformin is the usual first-line therapy unless contraindicated or not tolerated

Metformin	Sulphonylureas (SU)	Dipeptidyl peptidase inhibitor (DPP-4i)	Sodium glucose co-transporter 2 inhibitor (SGLT2i)	Insulin	Acarbose	Thiazolidinedione (TZD)

If glycated haemoglobin (HbA1c) target not achieved in three months:

- check and review current therapies, stop any that fail to improve glycaemic control
- check patient's understanding and self-management

- review use of therapies
- exclude other comorbidities/therapies impacting on glycaemic control

Second line: If metformin was not used first line, add it now, if not contraindicated

SU are the usual initial agent to add to metformin. If SU are contraindicated or not tolerated, another agent may be used

SU	DPP-4i	SGLT2i	Glucagon-like peptide-1 receptor agonist (GLP-1RA)	Insulin*	Acarbose	TZD

If HbA1c target not achieved in three months:

- check and review current therapies, stop any that fail to improve glycaemic control
- check patient understanding and self-management

- review use of therapies
- exclude other comorbidities/therapies impacting on glycaemic control

Third line: Consider triple oral therapy or addition of GLP-1 RA or insulin

| SU | DPP-4i | SGLT2i | GLP-1RA | Insulin* | Acarbose | TZD |

If HbA1c target not achieved in three months:

- check and review current therapies, stop any that fail to improve glycaemic control
- check patient understanding and self-management
- review use of therapies
- exclude other comorbidities/therapies impacting on glycaemic control

THEN

If on triple oral therapy

Switch ≥1 oral agent to GLP-1 RA or insulin* or another oral agent†   OR   Change to basal or premixed insulin*

If on GLP-1 RA

Add basal or premixed insulin*

If on basal insulin*

Add SGLT2 inhibitor or GLP-1 RA or basal bolus or basal plus insulin

OR

Australian blood glucose treatment algorithm for type 2 diabetes

Determine the individual's HbA1c target – this will commonly be ≤53 mmol/mol (7%). If not at target, or if an HbA1c reduction of ≥0.5% is not achieved after three months, move down the algorithm

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Dark blue boxes indicate usual therapeutic strategy (order is not meant to denote any specific preference) (usual refers to commonly available, evidence-based, cost-effective therapy). White boxes indicate alternate approaches (order is not meant to denote any specific preference). Black outlines indicate the classes of glucose-lowering agent that include PBS-subsidised products
*Unless metformin is contraindicated, or not tolerated, it is often therapeutically useful to continue it in combination with insulin in people with type 2 diabetes
†Switching an oral agent is likely to have the smallest impact on glycaemia

Table 6. Clinical considerations when choosing diabetes medications

Clinical outcome	Metformin	Sulphonylurea (SU)	Dipeptidyl peptidase-4 inhibitor (DPP-4i)	
Patients with established or high risk cardiovascular disease (CVD)	Neutral[109]	Increase risk when SU compared to metformin monotherapy (excluding gliclazide), but neutral when used in combination with metformin[110]	Neutral[107,111–114] Refer to Note A	
Patients with risk from hypoglycaemia	Lower rates compared to SU[110]	Higher clinical risks as monotherapy and combination with other agents[110] Gliclazide – 50% fewer hypoglycaemia episodes versus glimepiride[122]	Lower rates compared to SU[110]	
Patients at risk of gastrointestinal conditions (eg irritable bowel syndrome [IBS], inflammatory bowel disease [IBD] and gastroparesis)	Known intolerance as monotherapy or combination therapy – diarrhoea[110,124]	Neutral effect	Neutral effect	
Patients in whom stabilisation of body mass index [BMI] or weight loss is desired	Neutral effect	Neutral effect (gliclazide)[125] Modest gain (other SUs) compared to metformin monotherapy[110]	Less weight gain (when added to metformin versus metformin and SU)[110]	
Patients with renal dysfunction (eg lowered estimated glomerular filtration rate [eGFR])	Reduce dose eGFR 30–60 Contraindication with eGFR <30[124]	Hypoglycaemia risk increases	Safe with dose reduction# Refer to Note D	
Unique/class-specific pharmacological effects	Monotherapy or combination with other agents (DPP-4i or SGLT2i) is available to reduce 'pill burden'	The Australian algorithm (refer to Figure 4) suggests they may be used as monotherapy or combined with other agents	Contraindication – do not use with a GLP–1 RA	

	Acarbose	Thiazolidinedione (TZD)	Sodium glucose co-transporter 2 inhibitor (SGLT2i)	Glucagon-like peptide-1 receptor agonist (GLP-1 RA)	Insulin
	Not yet known[115]	Contraindication if symptomatic heart failure*[116]	Selective benefit/not yet known[117] Refer to Note B	Selective benefit/[118,119] not yet known Refer to Note C	Neutral[109,120,121]
	Neutral	Lower rates compared to SU[110]	Lower rates compared to SU[110]	Lower rates compared to SU[110]	Lower hypoglycaemia when metformin is added to basal versus premixed insulin (consider risks greater with prandial insulin)[123]
	Known Intolerance – bloating and flatulence[†]	Neutral effect	Neutral effect	Known intolerance – nausea and vomiting, and diarrhoea[‡ 110]	Neutral
	Neutral effect	Modest gain compared with other dual combination therapies[110]	Modest weight loss (in monotherapy, plus in combination with metformin versus metformin with alternate dual oral drug combinations)[§ 110]	Weight loss (in monotherapy, plus in combination with metformin versus metformin with alternate dual oral drug combinations)[‖ 110,126]	Modest gain – risk greater with prandial insulin[110,123]
	Contraindication in severe renal impairment[**]	Neutral	Efficacy decreases, thus contraindication with moderate renal impairment[††]	Contraindication eGFR<30[‡]	Hypoglycaemia risk increases
		Increased atypical fractures relative risk (RR) 1.57[127] in women* Rare – Pioglitazone has been associated with an overall 63% increased risk of bladder cancer, with the risk increasing with increasing duration of use and dose[128]	Modest lowering of blood pressure (BP)[110] Increased genitourinary (especially females) Refer to Note E Rare – euglycaemic ketoacidosis[‡‡]	Once weekly formulations are available[‡] Contraindication – do not combine with a DPP-4i	Dose required to be titrated to glycaemic goals

DPP-4i, dipeptidyl peptidase-4 inhibitor; eGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon like peptide-1 receptor agonist; IBD, Inflammatory bowel disease; IBS, Irritable bowel syndrome; insulin, basal, basal analogue, NPH and rapid-acting or prandial insulins including mixed insulins; PI, product information; SGLT2i, sodium glucose co-transporter 2 inhibitor; SU, sulphonylurea; TZD, thiazolidinedione

Important to note

Clinical considerations have been derived from the attached main references: Bennet W et al,[127] Qaseem A et al[129] and Bolen T et al[110]

Careful interpretation of results is recommended due to medication in combination studies having variance from Australian prescribing algorithms

Due consideration to each individual patient, diabetes and metabolic goals, disease and complication burden, medication side effects, and costs and psychosocial factors need to be incorporated in decision making, not just the medication factors (Table 6)

Notes

SU: Statements of evidence (SOEs) strengths[110] are related to the common forms available in the US – but are not inclusive of gliclazide, the most commonly used SU agent in Australia

Note A. DPP-4i and heart failure: In the SAVOR-TIMI 53 trial[107] – hospitalisations for heart failure (a secondary outcome) increased with saxagliptin. Despite this, a recent meta-analysis found difficulty in drawing firm negative conclusions on the comparative safety of this class of drugs in heart failure

Note B. SGLT2i: The EMPA-REG outcome trial[117] found that patients with diabetes at high CVD risk had reduced all-cause mortality and death from cardiovascular causes with empagliflozin. There have been no reported clinical trials for other drugs in this class

Note C. GLP-1 RA – The LEADER trial, a prospective cardiovascular safety outcomes trial for liraglutide, found a 13% reduction in major adverse cardiac events and a 22% reduction in cardiovascular death in high-risk cardiovascular patients.[130] This effect has not been replicated in similar trials for lixisenatide and no trials on exenatide have yet been reported

Note D. DPP-4i: All except linagliptin (no dose reduction) as this is hepatically metabolised

Note E. All classes: The recent Agency for Healthcare Research and Quality (AHRQ) US review[110] determined no moderate to high levels of evidence for the following adverse events (this does not mean no risk):

- Lactic acidosis (metformin)
- Urinary tract infections/fractures/volume depletion (SGLT2i)
- pancreatitis (DPP-4i and GLP-1 RA)
- bladder cancer risks (pioglitazones)
- thyroid cancer (GLP-1 RA)

*Pioglitazone product information is available at www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent&id=CP-2015-PI-01220-1&d=20160601161114622483

†Acarbose product information is available at www.bayerresources.com.au/resources/uploads/PI/file9350.pdf

‡Exenatide product information is available at www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent&id=CP-2013-PI-01780-1

§Dapagliflozin product information (–2.14 kg) as an add-on to metformin versus placebo at 104 weeks; empagliflozin (–1.63 kg) and (–2.03 kg) at doses 10 mg and 25 mg respectively as an add-on to metformin at 24 weeks

‖Exenatide –1 kg to 3.9 kg in comparative trials with combinations including metformin and SU and TZD

#All except linigliptin (no dose reduction)

**Exenatide (Bydureon) product information is available at www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent&id=CP-2013-PI-01764-1

††Dapagliflozin contraindicated of eGFR <60; empagliflozin/canagliflozin contraindicated of eGFR <45

‡‡Safety advisory – risk of diabetic ketoacidosis available at www.tga.gov.au/alert/sodium-glucose-co-transporter-2-inhibitors-used-treat-type-2-diabetes

In practice

Beginning glucose-lowering therapy

Healthy eating, physical activity and education remain the foundation of all type 2 diabetes treatment programs.

If lifestyle modification is not effective in controlling hyperglycaemia, metformin is the first choice unless contraindicated or not tolerated.

Second-line agents (added to existing metformin) may be necessary and should be chosen using an individualised approach, noting that agents work in different ways and are chosen to work synergistically.

While these guidelines recommend a stepwise approach to the management of type 2 diabetes, glycaemic management has become more complex with an increasing range of medications now available. There are a number of trials exploring the effects of various therapies on major cardiovascular events such as myocardial infarction (MI) and stroke. These trials include short-term trials of dipeptidyl peptidase-4 inhibitors (DPP-4i; oral: saxagliptin, alogliptin and sitagliptin) and glucagon like peptide-1 receptor agonists (GLP-1 RA; injectables: lixisenatide and liraglutide) and a sodium glucose co-transporter 2 inhibitor (SGLT2i; empagliflozin). However, there is a lack of data regarding long-term outcomes as well as potentially serious adverse outcomes associated with some of the newer agents. Unfortunately, a simple stepwise algorithm cannot match all individual patient's needs. The Australian Diabetes Society position statement[131] provides patient options based on consideration of efficacy, side effects, and costs.

Start with the correct dose of each medication and review on an individual basis at least every three to six months, keeping in mind the patient's individual HbA1c target.[132]

Safety

Each different class of glycaemic medications may have common and uncommon side effects that impact quality of life and require careful clinical re-assessment. Examples include some weight gain with sulphonylureas,[110] or mycotic infections and the rare euglycaemic diabetic ketoacidosis with SGLT2i.

Some patient groups (eg elderly and those with multiple comorbidities) may not be represented in the published clinical outcome trials of newer diabetes agents, so caution should be exercised when considering choice of agents for these patients.

When used as monotherapy, metformin, acarbose, glitazones, GLP-1 RA, DPP-4i and SGLT2i should not cause hypoglycaemia.

Long-acting sulphonylureas (eg glimepiride) or sulphonylureas with renally excreted active metabolites (eg glibenclamide) are more likely to cause hypoglycaemia than shorter-acting sulphonylureas (eg gliclazide).[133,134] Special care needs to be taken with those at increased risk of hypoglycaemia, especially the elderly. People taking sulphonylureas or insulin may need to notify motor vehicle licensing authorities as these medications can affect driving performance, as well as increase the patient's burden by requiring glucose self monitoring, especially on initiation or dose titrations. It is important that patients inform their insurance agents or companies (also refer to Chapter 14. Management of other impacts of diabetes).

8.3 Insulin

Clinical context

The impact of insulin on microvascular and cardiovascular outcomes has been partly evaluated in a comparative prospective outcome trial (eg the United Kingdom Prospective Diabetes Study [UKPDS]).

In terms of glycaemic control in type 2 diabetes, rapid-acting insulin and long-acting insulin analogues offer little benefit relative to conventional insulins.[135] However, a meta-analysis has demonstrated reduced hypoglycaemia with glargine insulin when compared to isophane insulin.[136] The Outcome Reduction with Initial Glargine Intervention (ORIGIN) trial demonstrated no beneficial or detrimental effect of glargine insulin on cardiovascular events.[121]

The use of insulin can improve glycaemic control in most people, but the increased risk of hypoglycaemia and weight gain (especially prandial insulins) must be considered.

Further long-term, high-quality prospective outcome studies of all glucose-lowering agents including insulin analogues are required. Improvements in surrogate markers such as HbA1c may not necessarily equate with longer term, clinically significant benefits.

Side effects of insulin therapy

Rare adverse events associated with the use of insulin have been reported in observational studies. Such events include congestive heart failure, oedema, lipodystrophy, allergic reactions, reversible transaminitis, reversible nephrotic syndrome and ß-cell destruction.[61]

Common side effects include hypoglycaemia and weight gain.

Risk factors for hypoglycaemia include:

- inappropriate dose
- timing or type of insulin (refer to below)
- missing meals
- alcohol intake
- exercise
- weight loss
- treatment with agents potentiating hypoglycaemia (eg sulphonylureas)
- decreased insulin clearance (eg renal failure).

Strategies for preventing hypoglycaemia in patients involve educating them about hypoglycaemic symptoms, structured SMBG, discussing and individualising glycaemic goals, and continued team-based support.[61]

Weight gain is variable on initiation of insulin and may accompany initial titration such that weight gain may eventually level off. Slower titration has been accompanied by slower weight gain. Strategies to address weight include: referral to an APD, management of any accompanying depression, a review of medications that may contribute to weight gain and advice on increased physical activity.

Early insulin intervention

Guidelines outlining the use of insulin in acute hyperglycaemic emergencies (including ketosis inducing and hyperosmolar crises) are available.[137–139] The use of insulin in these cases may be life saving and re-assessment of long-term use can occur on metabolic stabilisation.

Insulin types

Refer to Appendix G. Types of insulin available.

Insulin delivery options

A range of devices are available to deliver insulin, including insulin injectors (pens), insulin syringes and insulin pumps. Choice will depend on patient preference and capability. A CDE or a diabetes nurse practitioner can assist in the provision of patient support.

Insulin injectors (pens) are the most common way of administering insulin as they make multiple daily injection schedules much easier and allow people to be more flexible in their self management. Some people may find the 'InnoLet' injector easier to use because it is larger and has more visible markings; however, there are limited forms of insulin available with this device.

Insulin pumps have traditionally only been used in the management of type 1 diabetes. There is no evidence of the beneficial effects of using insulin pumps in people with type 2 diabetes.

Single use of pen needles and syringes is recommended.

In practice

Insulin therapy can be well managed in general practice.

GPs should anticipate and proactively address the patient's (and their own) reluctance to starting insulin therapy. Many patient concerns can be easily alleviated.[140]

Insulin is still the most effective glucose-lowering agent for type 2 diabetes, and can be titrated to suit the individual patient's requirements. Commencement should not be delayed if hyperglycaemia and symptoms cannot be adequately controlled by a patient's existing treatments. Insulin is not the end of the road for the person with diabetes, nor does it represent therapeutic or patient failure. The potential need for insulin should be broached early with patients.

When should patients start insulin?

It is important to discuss with patients with diabetes that insulin may be used at some point in their illness. Insulin should be initiated in patients with type 2 diabetes who are taking maximal doses of oral glucose-lowering agents and who have suboptimal glycaemic control (HbA1c or blood glucose above individualised target) whether they are asymptomatic[96,105,141,142] or symptomatic.[96]

Insulin therapy may remain an alternative for elderly or nursing home patients with HbA1c >75 mmol/mol (9%), especially if control of symptomatic hyperglycaemia is difficult.

Before starting insulin

Ensure that other possible causes of hyperglycaemia have been addressed (eg lifestyle, non-adherence to oral glucose lowering agents, taking other medication or medical conditions).[141]

Discuss with patients the benefits and costs of using insulin for better glycaemic control (Box 4).

Insulin initiation requires planning for patients (or their carers) with a focus on self management through education about dose adjustment, insulin delivery techniques, and SMBG and sick day management. Referral is recommended to a CDE prior to commencing insulin therapy. Insulin therapy requires initial education and skills training, with regular structured follow-up on such topics as: dose adjustment, insulin needle length and rotation techniques, hypoglycaemia management, exercise, illness and travel considerations, sharps disposal, identification, roads and maritime services notifications, and sick day management.

Box 4. Benefits and costs

Benefits:
- complications
- quality of life

Costs:
- weight
- hypoglycaemic risk
- GP visits

Initiating insulin

All insulins work effectively[140] and there is no wrong choice when commencing insulin. At the same time as selecting the insulin preparation, consider which injecting device is most suitable for the patient.

Select one of two insulin schedules:

- Basal insulin (eg glargine, isophane) once daily (refer to Appendix H.1. Guide to starting and adjusting basal insulin).

- Premixed (biphasic) insulin (eg lispro/lispro protamine or aspart/protamine insulin) once daily before the largest meal of the day[143] (refer to Appendix H.2. Guide to starting and adjusting premixed insulin).

One way to commence insulin therapy is with basal insulin, which has a slightly lower risk of nocturnal hypoglycaemia, especially if the fasting glucose is consistently above target.[105,141,143] Alternatively, premixed insulin may be more appropriate and simpler for a patient where fasting and postprandial glucose are consistently elevated.

Dosage adjustment can be more complex with premixed insulins as both insulin components are adjusted simultaneously, increasing the risk of hypoglycaemia and weight gain compared with basal insulin.[143–145]

Oral glucose lowering medications (eg metformin, sulphonylureas) may be continued as:[141]

- early cessation before blood glucose targets are achieved can result in significant hyperglycaemia

- ongoing use can reduce weight gain

- ongoing use allows a smaller insulin dose and can reduce the risk of hypoglycaemia or hyperglycaemia.

Titrating insulin

Set an individualised target (refer to Section 8.1. Glycaemic monitoring), then 'start low and go slow' to gain patient confidence and reduce the risk of hypoglycaemia.[141]

If insulin is commenced early, HbA1c targets can often be achieved with a once-daily insulin dose. Blood glucose control may be achieved before the HbA1c is at target because HbA1c measures the BGL over the preceding three months. Careful discussion of the impact of insulin on weight management needs to accompany dose titration, as well as the effect of exercise, carbohydrate intake and timing of meals with insulin dosing.

Check HbA1c (three months):

- Generally, if HbA1c is within target, then continue with the current schedule.[132]

- If HbA1c is outside the target, further action may be required (Box 5).

Box 5. If HbA1c is outside target[142]

Look for hidden hyperglycaemia by checking BGL before and 2 hours after lunch and dinner. Structured blood glucose monitoring may assist with this

If postprandial hyperglycaemia is identified, consider:[141]

- changing preceding meal size or composition
- increasing activity after meals
- adding an oral agent (eg acarbose, glucagon-like peptide-1 receptor agonist [GLP-1 RA], sodium glucose co-transporter 2 inhibitor [SGLT2i] or dipeptidyl peptidase-4 inhibitor [DPP-4i]) if the patient is not already taking one
- adding a prandial insulin dose (refer to 'Insulin intensification' below)
- switching to a premixed insulin (if on basal insulin alone).

Insulin intensification – Choosing a second-line insulin schedule

If HbA1c is elevated despite achieving appropriate BGL, a second-line insulin schedule (insulin intensification) should be implemented based on the individual patient's needs (Table 7).

Table 7. Patient considerations with insulin intensification

Considerations when setting targets	Lower target	Higher target	
Willing to monitor blood glucose level (BGL) several times per day	+	–	
Support from family and general practitioner (GP)	+	–	
Physically and cognitively capable	+	–	
Considerations when selecting a schedule	**Basal plus/Basal + glucagon-like-peptide-1 receptor agonist (GLP-1 RA)/Basal + sodium glucose co-transporter 2 (SGLT2) inhibitor/Basal + dipeptidyl peptidase-4 inhibitor (DPP-4i)**	**Basal bolus**	**Premixed**
Patient preference for fewest injections	+		+
Variable meal pattern	+	+	
Variable daily routine	+	+	
Better postprandial control required	+	+	±
Limited capacity (eg dexterity)			+

The preferred schedule is not the one with the most '+', but one which best meets the specific patient needs.[146,147]

There are newer insulin-intensification strategies[146] that can be implemented.

Basal insulin may be intensified in the following ways:

• Commence with the addition of a GLP-1 RA (eg twice daily exenatide). This strategy has the potential to improve HbA1c and postprandial BGL, while controlling weight gain and not markedly increasing the risk of hypoglycaemia.[148] Introduction of the GLP-1 RA requires instruction on injection technique – this is usually slowly titrated from a starting dose over several weeks, to a stable twice daily or once daily routine depending on the choice of agents. Only twice daily exenatide is currently TGA and PBS listed for use in combination with insulin. Liraglutide, an alternative GLP-1 RA, is currently TGA but not PBS approved. Long-acting (weekly) exenatide is now PBS-approved for use in combination with oral agents but not insulin.

- Use with a single daily oral dose of an SGLT2i. If commenced – improved glycaemia may require dose reduction of insulin at initiation by 10–20% of the total daily dose.
- Use with a daily dose of an oral DPP-4i – some agents have TGA approval and Pharmaceutical Benefits Advisory Committee (PBAC) recommendations, and will soon be PBS-approved, for this indication.

Complex regimes of additions to premixed insulin and basal bolus insulin may require additional specialist endocrinology support and education because of insulin dose adjustment complexity.

Insulin intensification regimes:

- Basal plus – where additional preprandial injection of short-acting insulin is added to basal insulin (Appendix H.3. Guide to basal plus insulin intensification schedules).
- Premixed – where additional injections of premixed are added to meals – either two or three times a day, or, alternatively, basal insulin is switched to premixed insulins (Appendix H.2. Guide to starting and adjusting premixed insulin).
- Basal bolus – where short-acting insulin injections are used before each meal in addition to basal insulin (Appendix H.3. Guide to basal plus insulin intensification schedules).

As Basal bolus involves the most number of injections and monitoring, it is typically the final strategy implemented.

When insulin intensification is initiated (eg multiple daily doses of insulin), metformin should be continued, but any remaining insulin secretagogues may need to be reviewed to minimise risk of hypoglycaemia.

Follow-up

The insulin schedule and dosing should be reviewed at each consultation. The insulin dosage may need to be reduced if the person adopts a healthier lifestyle and/or loses weight.

9. Managing cardiovascular risk

Recommendations	Reference	Grade*
Patients with pre-existing cardiovascular disease (CVD) are at high risk	149 NVDPA, 2012	A
All adults with type 2 diabetes and known prior CVD (except haemorrhagic stroke) should receive the maximum tolerated dose of a statin, irrespective of their lipid levels Note: The maximum tolerated dose should not exceed the maximum available dose (eg 80 mg atorvastatin, 40 mg rosuvastatin)	150 Baker IDI, 2015	A
Adults with any of the following conditions do not require absolute CVD risk assessment using the Framingham risk equation because they are already known to be at clinically determined high risk of CVD: • Diabetes and aged >60 years • Diabetes with microalbuminuria (>20 mcg/min or urine albumin-to-creatinine ratio [UACR] >2.5 mg/mmol for men and >3.5 mg/mmol for women) • Moderate or severe chronic kidney disease (persistent proteinuria or estimated glomerular filtration rate [eGFR] <45 mL/min/1.73 m^2) • A previous diagnosis of familial hypercholesterolaemia • Systolic blood pressure ≥180 mmHg or diastolic blood pressure ≥110 mmHg • Serum total cholesterol >7.5 mmol/L	149 NVDPA, 2012	D
Calculate risk level using an evidence-based tool: • National Vascular Disease Prevention Alliance charts, www.cvdcheck.org.au • New Zealand Cardiovascular Risk charts, www.health.govt.nz/publications • Heart Foundation NZ, www.knowyournumbers.co.nz	149 NVDPA, 2012	B
Aboriginal and Torres Strait Islander peoples are generally assumed to be at higher risk	149 NVDPA, 2012	B

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

CVD is the leading cause of death in people with diabetes, making assessment of CVD risk a vital part of diabetes care.

Assessment of combined multiple risk factors (absolute CVD risk) is more accurate than the use of individual risk factors.[149]

As with glycaemic targets, treatment targets in CVD need to be made on an individual basis, balancing the benefits and risks of interventions. For example, the CVD risk associated with lipid and BP levels is continuous; hence, specific targets are somewhat arbitrary and should be used as a guide to treatment, and not as a mandatory requirement. Table 8 can be used when developing a management plan for patients.

The risks associated with the effort required to reach a particular target, as opposed to achieving a near-target value, may outweigh any small absolute benefit. Any reduction in risk factor values will be associated with some benefit.[149]

Recommendations are summarised from the Natural Vascular Disease Prevention Alliance's *Guidelines for the management of absolute CVD risk*.[149]

Table 8. Risk management summary

CVD risk	Lifestyle	Pharmacotherapy	Targets	Monitoring
High risk: Clinically determined or calculated using Framingham risk evaluation (FRE) as >15% absolute risk of cardiovascular disease events over five years	Frequent and sustained specific advice and support regarding diet and physical activity Appropriate advice, support and pharmacotherapy for smoking cessation Advice given simultaneously with blood pressure (BP) and lipid-lowering drug treatment	Treat simultaneously with lipid-lowering and BP-lowering unless contraindicated or clinically inappropriate Aspirin not routinely recommended Consider withdrawal of therapy for people who make profound lifestyle changes	BP: • ≤140/90 mmHg in general or people with CKD • ≤130/80 mmHg in all people with diabetes* • ≤130/80 mmHg if microalbuminuria or macroalbuminuria (UACR >2.5 mg/mmol in men and >3.5 mg/mmol in women)	Review response 6–12 weekly until sufficient improvement or maximum tolerated dose achieved Adjust medication as required Review of absolute risk according to clinical context

CVD risk	Lifestyle	Pharmacotherapy	Targets	Monitoring
Moderate risk: Calculated using FRE as 10–15% absolute CVD risk events over five years	Appropriate, specific advice and support regarding diet and physical activity Appropriate advice, support and pharmacotherapy for smoking cessation Lifestyle advice given in preference to drug therapy	Not routinely recommended Consider BP-lowering and/or lipid-lowering in addition to lifestyle advice if three to six months of lifestyle intervention does not reduce risk or: • BP persistently ≥160/100 mmHg • Family history of premature CVD • Specific population where the FRE underestimates risk e.g. Aboriginal and Torres Strait Islander, South Asian, Maori, Pacific Islander and Middle Eastern peoples Consider withdrawal of therapy for people who make profound lifestyle changes	Lipids: Total cholesterol <4.0 mmol/L; high-density lipoprotein-cholesterol ≥1.0 mmol/L; low-density lipoprotein-cholesterol <2.0 mmol/L; Non-HDL-C <2.5 mmol/L; triglycerides <2.0 mmol/L. Lifestyle: • Smoking cessation (if smoker) • Consume diet rich in vegetables and fruit, low in salt and saturated and trans fats • At least 30 min physical activity on most or preferably every day of the week • Limit alcohol intake	Review response 6–12 weekly until sufficient improvement or maximum tolerated dose achieved Adjust medication as required Review absolute risk every 6–12 months
Low risk: Calculated using FRE as <10% absolute CVD risk events over five years	Brief, general lifestyle advice regarding diet and physical activity Appropriate advice, support and pharmacotherapy for smoking cessation	Not routinely recommended Consider BP-lowering therapy in addition to specific lifestyle advice if BP persistently ≥160/100 mmHg. Consider withdrawal of therapy for people who make profound lifestyle changes		Review response 6–12 weekly until sufficient improvement or maximum tolerated dose achieved Adjust medication as required Review absolute risk every two years Blood test results within five years can be used

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*Refer to the 'Antihypertensive medication to manage cardiovascular risk' section opposite for further discussion of BP targets

In practice

There are several interventions for managing CVD risk.

Lifestyle interventions to reduce CVD risk

Lifestyle changes in nutrition, physical activity and smoking status fundamentally underpin a comprehensive approach to CVD risk minimisation. Lifestyle changes show excellent cost-effectiveness in lowering the burden of disease and remain the basis for the management of all CVD risk levels.[151,152] However, the Look AHEAD study, showed improved HbA1c and quality of life benefit, but no reduction in risk of cardiovascular morbidity or mortality in people with type 2 diabetes who were obese (average BMI 36 kg/m^2;[82] refer to Chapter 6. Lifestyle modification).

Antihypertensive medication to manage cardiovascular risk

This section on antihypertensive interventions to reduce cardiovascular risk provides further discussion of BP targets.

Recommendations	Reference	Grade*
BP-lowering therapy in people with diabetes should preferentially include an angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB) If monotherapy does not sufficiently reduce blood pressure (BP) add one of the following:	149 NVDPA, 2012	A
• Calcium channel blocker	149 NVDPA, 2012	B
• Low-dose thiazide or thiazide-like diuretic	149 NVDPA, 2012	C
*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence		

Lowering BP reduces cardiovascular events and all-cause mortality in people with type 2 diabetes in the same manner as for the general population.

While no difference is noted between different classes of BP-lowering therapy for CVD outcomes, there is clear evidence that in people with type 2 diabetes, antihypertensive therapy with an angiotensin receptor blocker (ARB) or angiotensin-converting enzyme inhibitor (ACEI) decreases the rate of progression of albuminuria, promotes regression to normoalbuminuria and may reduce the risk of decline in renal function. Combining an ARB and an ACEI is not recommended.[149]

The target level for optimum BP is controversial.

A number of international guidelines have changed their blood pressure targets to <140/90 mmHg, while others remain at <130/80 mmHg. The target levels for BP therapy have been based on little direct trial evidence. A number of meta-analyses have demonstrated that the benefits of intensive BP control needs to be balanced with the risks. One meta-analysis demonstrated that more intensive BP control (SBP ≤130 mmHg) compared to usual (<140/90 mmHg) was associated with further reduction in stroke but a 40% increase in serious adverse events.[153] Two additional meta-analyses have recently been published. The analysis by Emdin at al[48] found that risk reduction was attenuated below an SBP of 140mmHg. However, there did appear to be a lower risk of stroke, retinopathy and albuminuria when blood pressure was reduced to <130 mmHg. Another meta-analysis, however, found that treatment of an SBP <140 mmHg was associated with increased CVD death.[154] This may in part be related to the selection of trials in this analysis which included patients with comorbidites such as CKD, heart failure and CVD.[155]

In line with these findings, it would be reasonable for GPs to shift the BP target to <140/90 mmHg for people with diabetes, with lower targets considered for younger people and those at high risk of stroke (secondary prevention), as long as the treatment burden is not high. The target BP for people with diabetes and microalbuminuria or proteinuria remains <130/80 mmHg. As always, treatment targets should be individualised and people with diabetes monitored for side effects from the use of medications to achieve lower targets.

Lipid medication to manage cardiovascular risk

Recommendations	Reference	Grade*
Use statins as first-line therapy	149 NVDPA, 2012	A

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

GPs should consider treatable secondary causes of raised blood lipids before commencing drug therapy.

When commencing drug therapy, statins remain the clear first-line choice. The results from several systematic reviews are consistent, and suggest that people with diabetes gain at least similar benefits from statin therapy as people without diabetes. The data clearly demonstrate that statin therapy results in a significant decrease in CAD morbidity and mortality in type 2 diabetes for those at high CVD risk. This benefit is in contrast to the contentious effects of improved glycaemic control in CVD.

Apart from statins, the evidence for other lipid-lowering therapy in decreasing the risk of CAD is still accumulating. Ezetimibe has been studied in IMPROVE-IT (Improved

reduction of outcomes: Vytorin efficacy international trial).[156] Patients with existing acute coronary syndrome and diabetes had a 14% reduction in death from CVD, a major coronary event (eg nonfatal MI, documented unstable angina requiring hospital admission, or coronary revascularisation occurring at least 30 days after randomisation), or nonfatal stroke. Among the patients with diabetes, the rates of MI and stroke were reduced by approximately 21% and 42% respectively.

Nicotinic acid, bile-acid resins, and fibrates (fenofibrate, gemfibrozil) have been suggested as alternatives for people who cannot tolerate statins. Nicotinic acid has been shown in one trial to reduce CVD outcomes, although the study was done in a cohort of people without diabetes.[157] More recent trials have not confirmed this initial result. The use of nicotinic acid, in particular, as well as gemfibrozil and cholestyramine is limited by a high rate of adverse effects.

The role of fibrates to decrease CVD is contentious. All large prospective randomised clinical trials of fibric acids have failed to decrease the primary cardiovascular end point. However, in all studies, predominantly post-hoc analyses, have shown that sub-groups having a low HDL-C level <0.9 mmol/L together with a raised triglyceride (generally >2.3 mmol/L) derive a significant benefit from fibrate therapy. Given these results, it may be reasonable to consider the introduction of fenofibrate in high-risk patients on statin therapy whose LDL-C is not at target and who have low HDL-C and raised triglyceride levels. Newer injectable lipid lowering agents called PCSK9 i (inactivate proprotein convertase subtilisin–kexin type 9 inhibitors) has TGA approval for combination with other lipid lowering agents in select high-risk patients. Mortality or morbidity outcomes are not yet established. Refer to the TGA website (www.ebs.tga.gov.au) for more information.

Antithrombotic therapy

Recommendations	Reference	Grade*
All adults with type 2 diabetes and known prior cardiovascular disease should receive long-term antiplatelet therapy unless there is a clear contraindication	150 Baker IDI, 2015	A
All adults with type 2 diabetes and a history of ischaemic stroke or transient ischaemic attack should receive:	150 Baker IDI, 2015	
• low-dose aspirin or		A
• clopidogrel or		A
• combination low-dose aspirin and extended-release dipyridamole		B
All adults with type 2 diabetes and recent acute coronary syndrome and/or coronary stent should receive, for 12 months after the event or procedure:	150 Baker IDI, 2015	
• combination low-dose aspirin and clopidogrel or		B
• combination low-dose aspirin and prasugrel or		B
• combination low-dose aspirin and ticagrelor		C
All adults with type 2 diabetes and a history of coronary artery disease, but no acute event in the past 12 months should receive	150 Baker IDI, 2015	
• long-term low-dose aspirin, or		A
• long-term clopidogrel if intolerant to aspirin		B
In the presence of atrial fibrillation or other major risk factors for thromboembolism, there should be consideration of anticoagulant therapy according to other relevant guidelines	150 Baker IDI, 2015	Practice Point

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Prescription of antiplatelet therapy (eg aspirin, clopidogrel) is not usually recommended in primary prevention, but for secondary prevention, the strong positive effects in the following conditions need to be weighed against individual patient risks.

10. Managing microvascular and other complications

10.1 Diabetic retinopathy

Recommendations	Reference	Grade*
Ensure that all people with diabetes have a dilated fundus examination and visual acuity assessment at the diagnosis of diabetes and at least every two years	158 NHMRC, 2008	None provided (Level I evidence)
Examine higher risk patients (eg longer duration of diabetes, or poor glycaemic control, blood pressure or blood lipid control) without diabetic retinopathy at least annually	158 NHMRC, 2008	None provided (Level I evidence)
Conduct annual screening for Aboriginal or Torres Strait Islander peoples with diabetes	158 NHMRC, 2008	None provided (Level IV evidence)

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Diabetic retinopathy (DR) occurs as a result of microvascular disease of the retina, and causes visual impairment and blindness, and affects up to one in three people with diabetes.

DR is categorised as either:

- non-proliferative DR
- proliferative DR.

Non-proliferative DR affects 19.3% of people with diabetes, while 2.1% may have proliferative DR and 3.3% may suffer macular oedema.

Non-proliferative DR may be asymptomatic and is characterised by retinal haemorrhages and exudates. Proliferative DR is characterised by new blood vessel growth (neovascularisation), which may lead to severe complications and blindness. Diabetes-related macular oedema is the leading cause of vision impairment with diabetes and occurs when exudates impact the macula. Tight control of blood glucose and blood pressure reduces the risk of onset and progression of diabetic eye disease in type 2 diabetes.

With good screening and care, visual impairment due to diabetes can be avoided for the vast majority of patients.

Two studies have prospectively assessed the effect of fenofibrate on microvascular disease, principally retinopathy. In both the Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) and Action to Control Cardiovascular Risk in Diabetes (ACCORD) studies, patients randomised to fenofibrate therapy had a significant reduction in retinopathy and need for laser surgery.[159] Additionally, in FIELD, there was a reduction in peripheral neuropathy complications and an improvement in proteinuria, suggesting a more generalised effect on microvascular disease. In Australia, the TGA has approved the use of fenofibrate for the treatment of diabetic retinopathy. Its use in patients with diabetes with evidence of retinopathy should now be considered. The benefits on retinopathy were not dependent on the patient having dyslipidaemia.[159]

Retinal photography

Retinal photography is technically simple and now usually performed within the Australian community by GPs, optometrists and ophthalmologists. Training is required to ensure quality of image interpretation. Some isolated general practices and Aboriginal health services are providing their own retinal photography services with support through telemedicine.

People whose retinal images suggest they may be at increased risk of having, or at some point developing, sight-threatening retinopathy should be referred for ophthalmology.

NB: A new item on the MBS for retinal photography with a non-mydriatic retinal camera will be available for general practice use from November 2016. The listing is expected to benefit Aboriginal and Torres Strait Islander peoples and communities in rural and remote locations where there is limited access to optometric and ophthalmic services to diagnose DR.

In practice

Assess all patients with type 2 diabetes for risk factors (refer to Box 6).

Box 6. Risk factors for development and progression of diabetic retinopathy

- Existing diabetic retinopathy
- Poor glycaemic control
- Raised blood pressure
- Duration of diabetes >10 years
- Microalbuminuria
- Dyslipidaemia
- Anaemia
- Pregnancy

The aim is to prevent vision loss and this is best done with regular review of fundi, early detection and optimisation of therapy.

GPs can monitor patients for diabetic eye disease if they are confident of their technique and examine the eyes through dilated pupils or have their own retinal camera. Where practitioners are not comfortable with fundoscopy and assessment of the retina, referral to an ophthalmologist or optometrist is recommended.

Monitoring involves:

- visual acuity (with correction)
- cataracts (refer to Section 10.2. Other ophthalmological)
- retinopathy (examine with pupil dilation or retinal camera, or refer to an optometrist or ophthalmologist).

Initial and then intermittent ongoing referral to an ophthalmologist or optometrist is still recommended for DR or peripheral retinopathy, which can be treated with laser photocoagulation therapy to prevent visual loss secondary to retinal haemorrhage.

Patients should be reviewed at least every two years and more frequently if problems exist.

10.2 Other ophthalmological effects

Refractive errors

Refractive errors occur as the lens' shape alters with changes in blood glucose concentrations and result in blurred vision. Detection is done with pinhole test – blurred vision due purely to refractive error corrects with the pinhole test.

Correction of refractive errors should be postponed until blood glucose levels are stabilised.

Cataracts

Cataracts occur prematurely in people with diabetes. Patients present with blurred vision and glare intolerance, and may find night vision a particular problem. Over time, interpretation of colours becomes more difficult.

Clinically, the light reflex is reduced and fundus may be difficult to see.

Surgical treatment is recommended when reduced acuity is affecting lifestyle and independence.

Maculopathy

Maculopathy is difficult to see ophthalmoscopically, but is the most common cause of visual loss in people with diabetes. Assessment is by direct ophthalmoscopy (with dilated pupils), retinal photography and fluorescein angiography, depending on the state of the patient's fundi.

Intra-vitreous injectable ranibizumab and aflibercept have recently been approved under the PBS as monotherapy or in combination with laser photocoagulation for diabetic macular oedema under management by an ophthalmologist. Refer to the PBS for further information at www.pbs.gov.au/pbs/home

Sudden blindness

Sudden loss of vision is an emergency and may be due to:

- central retinal artery occlusion
- retinal detachment
- vitreous haemorrhage.

These conditions can occur independently of diabetes. Urgent contact with an ophthalmologist or timely assessment by a specialist team is indicated.

10.3 Neuropathy

Clinical context

Pain and paraesthesia are common peripheral neuropathic symptoms, and if the autonomic nervous system is involved, gastrointestinal, bladder and sexual problems arise.

Diabetic neuropathic complications increase the patient's burden of self care and overall management.

The clinical focus is on prevention via good glycaemic control, and early recognition facilitated by good history and routine sensory testing. New modalities are arriving to assist in the management of diabetic neuropathies.

Before any treatment is instigated, exclusion of non-diabetic causes of neuropathy is suggested. This includes assessment for vitamin B12 deficiency, hypothyroidism, renal disease and a review of neurotoxic drugs including excessive alcohol consumption.

Autonomic neuropathy

Autonomic neuropathy may result in:

- orthostatic hypotension with >20 mmHg drop
- impaired and unpredictable gastric emptying (gastroparesis), which can cause a person's blood glucose levels to be erratic and difficult to control. Pro-kinetic agents such as metoclopramide, domperidone or erythromycin may improve symptoms
- diarrhoea
- delayed/incomplete bladder emptying
- erectile dysfunction and retrograde ejaculation in males
- reduced vaginal lubrication with arousal in women
- loss of cardiac pain, 'silent' ischaemia or myocardial infarction
- sudden, unexpected cardiorespiratory arrest, especially under anaesthetic or treatment with respiratory-depressant medications
- difficulty recognising hypoglycaemia (hypoglycaemic unawareness)
- unexplained ankle oedema.

Cardiovascular autonomic neuropathy should be suspected by resting tachycardia (>100 bpm) or orthostatic reduction in BP (a fall in SBP >20 mmHg on standing without an appropriate heart rate response). This applies to patients not currently on antihypertensive agents that may cause variations in BP responsiveness such as β-blockers. It is associated with increased cardiac event rates.

Diabetic peripheral neuropathy

Recommendations	Reference	Grade*
All patients should be screened for distal symmetric polyneuropathy starting at diagnosis of type 2 diabetes and at least annually thereafter, using simple clinical tests	19 American Diabetes Association, 2015	B
Antidepressants, including tricyclics, duloxetine and venlafaxine should be considered for the treatment of patients with painful diabetic peripheral neuropathy	65 SIGN, 2014	A
Anticonvulsants, including pregabalin and gabapentin, should be considered for the treatment of patients with painful diabetic peripheral neuropathy	65 SIGN, 2014	A

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Diabetic neuropathies increase with age, duration of diabetes and level of control of diabetes. They are heterogeneous with diverse clinical manifestations and may be focal or diffuse.

In practice

Early recognition and appropriate management is important. People with type 2 diabetes should have an annual check for diabetic peripheral neuropathy.

The appearance of peripheral neuropathy should prompt review and consideration of improved glycaemic control.

The Diabetes Neuropathy Score may be used to confirm diagnosis and assess severity (refer to Table 9).[160,161]

Table 9. Diabetic neuropathy symptom score[162]

1.	Are you experiencing unsteadiness in walking (need for visual control, increase in the dark, walk like a drunk man, lack of contact with floor)?
2.	Do you have a burning, aching pain or tenderness at your legs or feet (occurring at rest or at night, not related to exercise, excluding intermittent claudication)?
3.	Do you have prickling sensations on your legs and feet (occurring at rest or at night, distal>proximal, stocking glove distribution)?
4.	Do you have places of numbness on your legs or feet (distal>proximal, stocking glove distribution)?

Score:

- 1 point if a symptom occurred several times a week during the past two weeks
- No points if this does not occur

0 points, polyneuropathy absent; 1–4 points, polyneuropathy present

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The pain of peripheral neuropathy can be difficult to manage, although there is evidence that several agents can improve symptom control and quality of life. Tricyclic medications should be used as a first-line treatment, although side effects are relatively uncommon.

Gabapentin provides pain relief of a high level in approximately one-third of people who take this medication for painful neuropathic pain. Side effects are common (66%).[163] Pregabalin at daily oral doses of 300–600 mg provides high levels of benefit for a minority of patients experiencing neuropathic pain including painful diabetic neuropathy.[164]

Motor neuropathy sometimes occurs with muscle wasting, weakness and abnormalities of gait. This can contribute to foot problems by altering the biomechanics of the ankle and foot.

Combinations of more than one test have >87% sensitivity in detecting diabetic peripheral neuropathy (refer to Box 7). Loss of 10 g monofilament perception and reduced vibration perception predict foot ulcers.[165]

Box 7. Tests to assess for peripheral neuropathy

- Pinprick sensation
- Vibration perception (using a 128-hz tuning fork)
- 10 g monofilament pressure sensation at the distal plantar aspect of both great toes and metatarsal joints
- Assessment of ankle reflexes.

10.4 Nephropathy

Recommendations	Reference	Grade*
Assessment		
Kidney status in people with type 2 diabetes should be assessed by:		
• annual screening for albuminuria (note that dipstick urine test is not adequate to identify albuminuria)	166 NHMRC, 2009	B
• annual estimated glomerular filtration rate (eGFR; in mL/min/1.73 m^2)	166 NHMRC, 2009	B
Management		
Reducing the risk or slowing the progression of nephropathy can be achieved by:		
• blood glucose control should be optimised aiming for a general glycated haemoglobin (HbA1c) target ≤7%	166 NMHRC 2009	A
• optimising blood pressure control	166 NMHRC 2009	A
In people with type 2 diabetes and microalbuminuria or macroalbuminuria, angiotensin receptor blocker (ARB) or angiotensin converting enzyme inhibitor (ACEI) antihypertensive should be used to protect against progression of kidney disease	166 NHMRC, 2009	A
People with type 2 diabetes should be informed that smoking increases the risk of chronic kidney disease	166 NHMRC, 2009	B
People with diabetes and microalbuminuria are considered at high cardiovascular disease risk, and should be treated with multifactorial interventions (refer to Chapter 9. Managing cardiovascular risk)	149 NVDPA, 2012	D
*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence		

Clinical context

Diabetic nephropathy occurs in one in four women and one in five men with type 2 diabetes,[167] and is the single leading cause of end-stage renal disease.

Diabetic nephropathy is more common in Aboriginal and Torres Strait Islander peoples. Some non-European groups (eg South-East Asian, African American, Afro-Caribbean, Maori peoples) have high rates of end-stage diabetic nephropathy, possibly, but not entirely, due to later diagnosis and poorer care.[30]

There is strong evidence that treatment in the early stages of CKD reduces progression of kidney damage, morbidity and mortality. Therefore, people with type 2 diabetes should be screened and retested regularly to detect early indications of kidney damage and to monitor the effects of treatment.

SBP appears to be the best indicator of the risk of CKD in type 2 diabetes. However, the optimum and safest lower limit of SBP has not been clearly defined. Refer to general goals for diabetes (refer to Chapter 9. Managing cardiovascular risk) for appropriate individual targets for BP.

In practice

Assessment

Screening for microalbuminuria can be performed by measurement of the urine albumin-to-creatinine ratio (UACR) in a random spot collection (preferred method). Any positive UACR needs to be confirmed with a repeated collection and also a mid-stream urine to exclude urinary tract infection as a contributor to proteinuria.

The automatic calculation of eGFR on measurement of serum creatinine is now implemented within Australia.

Review of possible nephrotoxic medication, investigations to exclude treatable causes of kidney disease, and the assessment of a patient's CVD risk form a baseline approach to patients with confirmed kidney disease. Figure 5 provides an algorithm for the initial detection of CKD.[168]

Figure 5. Algorithm for initial detection of chronic kidney disease

Note: This algorithm does not allow for checks for people with glomerulonephritis, systemic lupus erythematosus or people on nephrotoxic drugs.

Offer Kidney Health check to people with any of the following indications:
- Diabetes
- Hypertension
- Established cardiovascular disease
- Family history of kidney failure
- Obesity
- Smoking
- Aboriginal or Torres Strait Islander origin aged ≥30 years

Indication not present → Kidney Health check not recommended

Estimated glomerular filtration rate (eGFR)

Urine albumin-to-creatinine ratio (UACR)

If UACR and eGFR are normal, repeat kidney health check in one to two years (annually if hypertension or diabetes present)

eGFR <60 mL/min/1.73 m²

Elevated UACR (males ≥2.5 mg/mmol, females ≥3.5 mg/mmol)

Repeat eGFR within seven days

≥20% reduction in eGFR → Possibly acute kidney injury – discuss with nephrologist

Repeat UACR twice within next three months (preferably first morning void)

Stable reduced eGFR

Repeat eGFR twice within three months

Minimum two out of three elevated UACR's present for ≥3 months

Minimum three reduced eGFRs present for ≥3 months

Kidney function stage	eGFR (mL/min/1.73 m²)	Albuminuria stage		
		Normal (UACR; mg/mmol) Male: <2.5 Female: <3.5	Microalbuminuria (UACR; mg/mmol) Male: 2.5–25 Female: 3.5–35	Macroalbuminuria (UACR; mg/mmol) Male: >25 Female: >35
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present	Yellow*	Red*
2	60–89		Yellow*	Red*
3a	45–59	Yellow*	Orange*	Red*
3b	30–44	Orange*	Orange*	Red*
4	15–29	Red*	Red*	Red*
5	<15 or on dialysis	Red*	Red*	Red*

Investigations to determine underlying diagnosis

Combine eGFR stage (1–5), albuminuria stage and underlying diagnosis to fully specify CKD (eg Stage 2 CKD with microalbuminuria because of diabetic kidney disease)

*Refer to colour–coded action plans (yellow, orange, red) in Kidney Health Australia's *Chronic kidney disease management in general practice*[168] for management strategies

Reproduced with permission from Kidney Health Australia. Chronic kidney disease management in general practice. 3rd edn. Melbourne: Kidney Health Australia, 2015.

Management

Due to potential reno-protective effects, the use of ACEIs or an ARB should be considered for the small subgroup of people with normal BP who have type 2 diabetes and microalbuminuria.[169] It is not recommended that ACEIs and ARB medication be used together.

ACEIs reduce the incidence of end-stage renal disease by 40%, and ARBs reduce this progression by 22%. However, the absolute benefit was small (1.5% versus 0.8% over approximately one year; number needed to treat = 160). Both drug classes reduced the risk of disease-oriented renal outcomes such as doubling of creatinine concentration and progression of micro-albuminuria to macro-albuminuria. Both classes of medication also increased rates of regression from micro albuminuria- to normoalbuminuria. There are no direct comparative trials of ACEIs and ARBs to determine which is more effective. However, there is clear evidence of both classes having benefits in comparison with placebo.[170,171]

A meta-analysis of RCTs demonstrated that ACEIs and ARBs differentially affect the risk of all-cause mortality, cardiovascular deaths and cardiovascular events in patients with diabetes.[172] ACEIs reduce the risk of mortality, MI and heart failure, while ARBs do not affect the risk of mortality and major cardiovascular events. No effect on stroke was seen with either treatment.

Medication considerations:

- **Metformin** – should be used with caution (as risks of lactic acidosis increases), and dose should be reduced when eGFR is 30–60 mL/min/1.73 m^2. It is not recommended and should be ceased when eGFR is <30 mL/min/1.73 m^2.
- **DPP-4i**[173] – reduction of dose of alogliptin, saxagliptin, sitagliptin and vildagliptin are required with eGFR <60 mL/min/1.73 m^2 due to pharmacologic accumulation without toxicity. All except saxagliptin can be used in end-stage renal failure. Linagliptin has no dose adjustment requirement in renal impairment due to hepatic metabolism.
- **Sulphonylureas**[173] – as renal function declines, the half-life of sulphonylureas increases, raising the risk of hypoglycaemia.
- **SGLT2i** – require renal function for glycaemic effect, dapagliflozin may be used if the eGFR is >60 mL/min/1.73m^2 and empagliflozin may be used if the eGFR is >45 mL/min/1.73 m^2.
- **Acarbose**[173] – avoid if eGFR < 25mL/min/1.73 m^2.
- **Glitazones** – dose adjustment in patients with renal dysfunction is not recommended. No information is available for patients on dialysis, therefore pioglitazone should not be used in such patients.
- **GLP-1 RA**[173] – exenatide and liraglutide use is not recommended below eGFR <30 mL/min/1.73 m^2.

- **Insulin** – dose review with increasing renal impairment as risks of hypoglycaemia increase
- any potentially nephrotoxic medications (eg non-steroidal anti-inflammatory drugs [NSAIDs]) should be avoided.

Box 8 shows the criteria for referral to a renal specialist.

Box 8. Referral criteria for specialist renal care[168]

Referral criteria for specialist renal care may include:

- estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m^2 (Stage 4 or 5 chronic kidney disease [CKD] of any cause)
- persistent significant albuminuria (urine albumin-to-creatinine ratio [UACR] ≥30 mg/mmol)
- a sustained decrease in eGFR of ≥25% OR a sustained decrease in eGFR of 15 mL/min/1.73 m^2 within 12 months
- CKD with hypertension that is hard to get to target despite at least three antihypertensive agents

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10.5 Foot complications

Recommendations	Reference	Grade*
Assess all people with diabetes and stratify their risk of developing foot complications	160 NHMRC, 2011	C
Assess risk stratification by inquiring about previous foot ulceration and amputation plus falls risk, visually inspecting the feet for structural abnormalities and ulceration, assessing for neuropathy using either the neuropathy disability score or a 10 g monofilament and palpating foot	160 NHMRC, 2011	C
People assessed as having intermediate-risk or high-risk feet should be offered a foot protection program. This includes foot care education, podiatry review and appropriate footwear	160 NHMRC, 2011	C
Pressure reduction, otherwise referred to as redistribution of pressure or offloading, is required to optimise the healing of plantar foot ulcers	160 NHMRC, 2011	B
Offloading of the wound can be achieved with the use of a total contact cast or other device rendered irremovable	160 NHMRC, 2011	B
People with diabetes-related foot ulceration are best managed by a multidisciplinary foot care team	160 NHMRC, 2011	C

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Foot ulceration and limb amputation are among the major drivers of disability and healthcare costs in people with diabetes. Foot ulceration accounts for one in five of all diabetes-related admissions to hospital.

Indications for immediate referral to a podiatrist include concerns about vascular and/or neuropathic complications of diabetes. Predisposing structural problems often exist, heightening complication risks. Improper footwear and tinea infection have been associated with increased podiatry problems.

A care plan and foot protection program that includes prevention, patient education, multidisciplinary care, and close monitoring and treatment of foot ulcers can substantially reduce amputation rates.

Patients should understand the importance of appropriate footwear and foot care, establish a regular self-monitoring schedule (including visual checks), and have an action plan to respond to early problems (eg skin breakdown). Regular podiatric review needs to be considered. Refer to Appendix I. Tools for assessing neuropathy circulation and foot deformity for practice-based tools for assessing circulation and foot deformity.

In practice

Foot care education should be provided to all people with diabetes to assist with prevention of foot complications. For people with intermediate and high risk, a podiatry assessment is an important component of a foot protection program. However, where this is not possible, a suitably trained healthcare worker may perform the foot assessment.

A careful foot assessment should be performed to stratify the risk of developing foot complications. Assessment is dependent on three risk factors:

- **Peripheral arterial disease (PAD)** – which can be assessed by dorsalis pedis and tibialis anterior pulses or hand-held Doppler. If problems are suspected, consider ankle-brachial index (ABI) testing, toe brachial index (TBI) testing or absolute toe pressure.
- **Neurological testing** – which can be undertaken using a neuropathy disability score or a 10 g monofilament assessment.
- **Deformities and ulceration** – these can be assessed by visual inspection.

Practitioners are advised to stratify foot risk according to the presence of risk factors and history of ulceration and/or amputation. The intensity of monitoring and review increases according to the level of risk. Table 10 shows risk categorisation for complications and elements to consider during foot assessment.

Table 10. Guidance on risk categorisation for complications and elements to consider during foot assessment[160]

Stratification of foot ulceration and amputation risk in diabetes		NHMRC grade*	Foot care and education tailored to foot risk status
Low risk	No risk factors for foot ulceration or ulceration/amputation	C	Offer basic foot care information and annual foot assessment
Intermediate risk	One risk factor only (ie neuropathy, peripheral arterial disease [PAD]) and no previous history of foot ulceration or amputation	C	Offer program that includes foot care education, podiatry review every six months and footwear assessment
High risk	Two or more risk factors (ie neuropathy, PAD or foot deformity) and/or previous foot ulceration or amputation	C	Offer program that includes foot care education, podiatry review and footwear assessment
High risk	Aboriginal or Torres Strait Islander peoples with diabetes	Practice Point	Offer program that includes foot care education, podiatry review and footwear assessment

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Foot ulceration

A foot ulcer is a serious condition and needs to be managed immediately.

The University of Texas wound classification system is the most useful tool for grading foot ulcers (refer to Table 11).[174,175]

If arterial insufficiency is suspected, assessment and management of the peripheral vasculature is mandatory before removal of non-viable or necrotic tissue is considered.

Referral to a vascular surgeon, high-risk foot clinic and/or multidisciplinary team is suggested in this situation.

The first priority of management of foot ulceration is to prepare the surface and edges of a wound to facilitate healing. Local sharp debridement of non-ischaemic wounds should be performed because it improves ulcer healing. Arterial supply needs to be determined before beginning any treatment.

Wound dressings need to be tailored to the specific characteristics of the wound.

In non-ischaemic ulcers, create a moist wound environment. Currently, there is insufficient evidence to demonstrate the superiority of any one type of wound dressing over another in the management of ulcers. Appropriate management of wound exudate levels should be a guiding principle in dressing selection and the frequency of dressing change. In ischaemic ulcers, maintain a dry wound environment using a dry, non-adherent dressing until someone with experience in PAD has reviewed the wound.

Table 11: University of Texas wound grading system[174,175]

		Grade/depth: 'How deep is the wound?'			
Stage/comorbidities: 'Is the wound infected, ischaemic or both?'	Depth	Grade			
		0	I	II	III
	A	Pre-ulcerative or post-ulcerative lesion completely epithelialised	Superficial wound not involving tendon, capsule or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
	B	Pre-ulcerative or post-ulcerative lesion completely epithelialised with infection	Superficial wound not involving tendon, capsule or bone with infection	Wound penetrating to tendon or capsule with infection	Wound penetrating to bone or joint with infection
	C	Pre-ulcerative or post-ulcerative lesion completely epithelialised with ischaemia	Superficial wound not involving tendon, capsule or bone with ischaemia	Wound penetrating to tendon or capsule with ischaemia	Wound penetrating to bone or joint with ischaemia
	D	Pre-ulcerative or post-ulcerative lesion completely epithelialised with infection and ischaemia	Superficial wound not involving tendon, capsule or bone with infection and ischaemia	Wound penetrating to tendon or capsule with infection and ischaemia	Wound penetrating to bone or joint with infection and ischaemia

Reproduced with permission from Lavery LA, Armstrong DG, Harkless LB. Classification of diabetic foot wounds. J Foot Ankle Surg 1996;35(6):528–31.

Factors that favour referral to a multidisciplinary foot care team include:

- deep ulcers (eg probe to tendon, joint or bone)
- high-risk foot with active ulcer
- ulcers not reducing in size after four weeks despite appropriate treatment – if in regional or remote areas, a telemedical review or telephone review would be recommended
- the absence of foot pulses/ low ABI or TBI treading
- ascending cellulitis
- suspected Charcot neuroarthropathy (eg unilateral, red, hot, swollen, possibly aching foot).

If access to a multidisciplinary foot care team is limited, foot ulceration or complications other than those listed above may be managed by a GP together with a podiatrist and/or wound care nurse.[160]

An important reason for failure of an ulcer to heal is continued trauma to the bed of the wound. This generally occurs because the foot is insensate and the patient continues to bear weight through the wound. A number of offloading devices are currently available. These include total-contact casts and removable prefabricated devices (eg controlled ankle-movement walkers, half-shoes, therapeutic shoes). Patient ability to understand and undertake management should always be a factor in choosing a treatment and in counselling the patient regarding the treatment plan.

Infection

The need for antibiotics should be determined on clinical grounds.

It is appropriate for cultures to be collected for identification of microbiological organisms and antibiotic sensitivities. The most appropriate tissue samples for microbiological evaluation are either deep tissue swabs after debridement or tissue/ bone biopsies.

Infected ulcers should be treated with antimicrobial therapy according to published antibiotic guidelines (eg https://tgldcdp.tg.org.au/ guideLine?guidelinePage=Antibiotic&frompage=etgcomplete).

The duration of therapy may need to be for extended periods.

There is no need to culture clinically uninfected ulcers as colonising organisms will always be detected.

11. Glycaemic emergencies

Refer to Appendix J. Detailed information on glycaemic emergencies for more detailed information.

Recommendations	Reference	Grade*
The potential harmful effects of optimising blood glucose control in people with diabetes should be considered when setting individual glycaemic targets	96 NHMRC, 2009	A
Improving blood glucose control increases the risk of hypoglycaemia	96 NHMRC, 2009	None provided (Level I evidence)

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

In patients with type 2 diabetes, very high and low glucose states can occur. Both have significant impacts and implications, and require patient knowledge and active management planning.

Hypoglycaemia occurs in people with diabetes when their blood glucose level falls below 4 mmol/L or is at a level that causes symptoms and signs. It is more common in people taking insulin. However, it can also occur with sulphonylurea therapy either alone or supplementing other oral therapies. Other causative factors are deficient carbohydrate intake, renal impairment and excessive alcohol ingestion.

The frequency of hypoglycaemia must not be underestimated, particularly in patients where the morbidity of hypoglycaemia poses particular problems and symptoms may be unrecognised.

Higher risk patients include the elderly, and those with renal impairment and recent hospitalisation or multiple medications.[176] Impaired hypoglycaemia unawareness is a clinical risk that increases with the duration of diabetes, and occurs where the pathophysiologic symptoms that arise in response to mild or severe hypoglycaemia (refer to below) are reduced or absent. In such cases, symptoms may be recognised by other family members and carers before the patient. Such patients need referral to an endocrinologist.

Emergency hyperglycaemic states include diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic states (HHS; formerly hyperosmolar known as non-ketotic coma [HONC]). These conditions occur due to very poor glycaemic control,

implying diabetes management issues or underlying and/or precipitating causes such as infection or MI and require emergency management. DKA is rare, but can occur in type 2 diabetes. It is no longer a complication unique to type 1 diabetes. Hyperglycaemic thresholds for notification and intervention may be considered in management planning (eg SMBG >15mmol/L on two subsequent occasions, two hours apart).

In practice

All patients with type 2 diabetes on insulin and/or sulphonylureas and their families need to be informed about the risks, signs and symptoms, and actions to be taken. If there has been severe hypoglycaemia, identification of a carer who may be trained in glucagon administration may assist earlier intervention if recurrence is to be avoided. The Australian Diabetes Educators Association (ADEA) sick day management guidelines may be used to assist practical patient management. Visit www.adea.com.au/about-us/our-publications

Recognising signs and symptoms

Symptoms of hypoglycaemia vary between persons. Common symptoms are:

- Adrenaline activation symptoms that include pale skin, sweating, shaking, palpitations and a feeling of anxiety or dizziness.
- Neuroglycopenic symptoms that may include hunger, change in intellectual processing, confusion and changes in behaviour (eg irritability), paraesthesiae, then coma and seizures.
- Signs of hyperglycaemic states include severe dehydration, altered consciousness, shock and ketotic breath in patients with DKA.

Intervention

Hypoglycaemia

Mild and moderate hypoglycaemia can be treated by following the 'Rule of 15' (refer to Appendix J. Detailed information on glycaemic emergencies). For severe hypoglycaemia, the patient requires treatment by a carer or health professional.

Severe hypoglycaemia resulting in an hypoglycaemic coma (a person with diabetes who presents unconscious, drowsy or unable to swallow) is an emergency.

Management is as follows:

- Commence appropriate resuscitation protocols.

- Give an injection of glucagon 1 mg intramuscular or subcutaneous if available.
- If intravenous access is obtained, glucose 50% – 20 mL intravenous via a securely positioned cannula (optimally the antecubital veins). Use 10% glucose in children, as hyperosmolality has caused harm.
- Phone for an ambulance (dial 000) stating a 'diabetic emergency'.
- Wait with the patient until the ambulance arrives.
- When the person regains full consciousness and can swallow, they can then be orally given a source of carbohydrate.
- Review of medications, dietary intake, driving or licensing requirements and hypoglycaemia management is mandatory.

Hyperglycaemic emergencies

A patient with a hyperglycaemic emergency requires the following:

- Correct extracellular fluid deficit and then slowly correct water depletion and hyperglycaemia, monitoring sodium and potassium closely.
- Give subcutaneous rapid acting insulin 0.1 units/kg while awaiting transfer.
- Look for an underlying cause – sepsis, MI.
- Monitor blood glucose every one to two hours for the first four hours (then revert to usual testing when BGL is <15 mmol/L).
- Transfer to a specialist unit as soon as possible.
- Review medications, dietary intake and hyperglycaemic and sick day management.

Refer to Appendix J. Detailed information on glycaemic emergencies for more information.

12. Diabetes, multimorbidity and medication complications

12.1 Multimorbidity

Clinical context

Healthcare systems around the world face a growing challenge of managing populations with multiple co-existent chronic conditions, including diabetes. On an individual level, multimorbidity can have a profound effect on a patient's ability to self care and balance different treatment needs across multiple conditions.

The challenge for general practice is to optimise the care for these patients. Guidelines are usually configured for individual diseases rather than multimorbidity. Guidelines can only provide structured information and evidence-based recommendations. They are a guide for decision making for individual patients and infrequently address the problems of implementation in different patients with varying personal and clinical priorities.

High-quality management of diabetes cannot occur in isolation from other co-existing physical or mental health disorders, nor can management ignore age and socioeconomic issues.[177]

Three out of four adults with diabetes have at least one comorbid chronic disease[177] and up to 40% have at least three (refer to Figure 6).[178,179] These comorbidities may or may not be diabetes related, and awareness and treatment of comorbidities is related to better glycaemic control.[180]

While many conditions have a concordant treatment focus (eg use of an ACEI to reduce the risk of cardiovascular events), others, such as depression, chronic obstructive pulmonary disease (COPD) and painful conditions may be discordant.[181,182] For example, patients may require medications such as NSAIDs that may adversely affect the management of diabetes and whose presence is a risk factor for poorer self care, more frequent diabetes complications and death.[183]
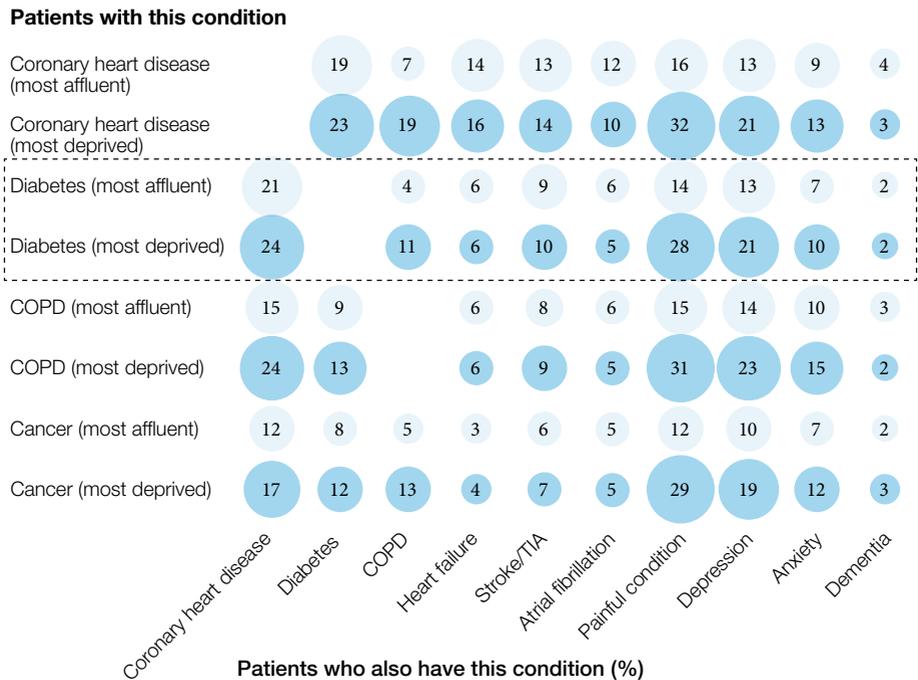
Age and multimorbidity

Symptomatic conditions may receive priority from patients. Studies of symptomatic burden have found that adults with type 2 diabetes aged ≥60 years report more physical symptoms such as acute pain and dyspnoea, and are more likely to have cognitive impairment and physical disability than those without diabetes.[184]

Combined with obesity, these risks are approximately doubled. People aged <60 years report more psychosocial symptoms, such as depressed mood and insomnia. Acute pain was prevalent (41.8%) and 39.7% reported chronic pain, 24.6% fatigue, 23.7% neuropathy, 23.5% depression, 24.2% insomnia and 15.6% physical/emotional disability.[184]

Socioeconomic status and multimorbidity

Being part of the most socially disadvantaged groups in Australia doubles the risk of developing diabetes. Within low socioeconomic groups, financial stressors may also play a role in treatment choices. Hence, the management of diabetes should always be considered as part of a comprehensive management plan, which addresses whole-patient priorities.

Figure 6. Many patients with diabetes have other medical conditions

Patients with this condition

	Coronary heart disease	Diabetes	COPD	Heart failure	Stroke/TIA	Atrial fibrillation	Painful condition	Depression	Anxiety	Dementia
Coronary heart disease (most affluent)		19	7	14	13	12	16	13	9	4
Coronary heart disease (most deprived)		23	19	16	14	10	32	21	13	3
Diabetes (most affluent)	21		4	6	9	6	14	13	7	2
Diabetes (most deprived)	24		11	6	10	5	28	21	10	2
COPD (most affluent)	15	9		6	8	6	15	14	10	3
COPD (most deprived)	24	13		6	9	5	31	23	15	2
Cancer (most affluent)	12	8	5	3	6	5	12	10	7	2
Cancer (most deprived)	17	12	13	4	7	5	29	19	12	3

Patients who also have this condition (%)

COPD; chronic obstructive pulmonary disease; TIA; transient ischaemic attack

Reproduced with permission from Elsevier from Barnett K, Mercer SW, Norbury M, et al. Epidemiology of multimorbidity and implications for healthcare, research, and medical education: A cross-sectional study. Lancet 2012;380(9836):37–43.

Approach to multimorbidity

A number of comorbidities are commonly associated with diabetes (refer to Table 12). The best approach for a patient with multimorbidity is the subject of international debate. Unfortunately, multimorbidity increases clinical complexity, which is unlikely to be effectively addressed by more sophisticated guidelines[181,185–187] or the Chronic Care Model.[188–191] Hence, a set of principles to guide an approach seems to offer a clinical solution.

Table 12. Comorbidities associated with diabetes or arising from complications of diabetes

Classification	Common comorbidities
Psychological	Chronic pain disorders Depression and anxiety Dementia
Macrovascular disease	Hypertension Coronary disease Cerebrovascular disease Peripheral vascular disease High-risk foot issues
Microvascular disease	Renal impairment and chronic kidney disease Neuropathy – peripheral, autonomic Retinopathy
Metabolic disorders	Dyslipidaemia Low testosterone in males Hepatic steatosis Joint issues (eg frozen shoulder) Pancreatitis
Overweight and obesity-related comorbidities	Obstructive sleep apnoea Osteoarthritis
Other	Bacterial, fungal and viral infections Periodontal disease

In practice

Consider the following key principles in the approach to management of patients with type 2 diabetes and co-existing morbidities.

Recognise clinical context and prognosis

Consider clinical management decisions within the context of risks, burdens, benefits, and prognosis of a patient's life (eg remaining life expectancy, functional status, quality

of life).[181,185,186] Treatment outcomes of glycaemia, hypertension, and hyperlipidaemia all have multi-year time horizons required to provide benefit and these may not be available in all clinical contexts.

Set treatment priorities with the patient

Focus on outcomes that matter most to the individual. Shared decision making with patients is vital to ensure care is aligned with their values and preferences.[185,192–195]

Even though diabetes may be a clinically dominant condition, patients may prioritise therapeutic interventions differently. For example, pain relief from low back pain or respiratory relief from COPD may be considered above their diabetes needs, many of which may not cause daily symptoms.

Recognise the limitations of the evidence base

Many of the patterns of multimorbidity have similar pathogenesis and therapeutic management strategies (eg diabetes, hypertension, CAD).

Clinical guidance regarding discordant conditions such as steroid-dependent conditions (which potentiate poor glycaemic control), mental health conditions, chronic pain, cancer or conditions that alter medication pharmacokinetics (eg renal disease, cardiac failure, liver disease, malabsorptive states) is often lacking or sparse. The absolute harms and benefits of diabetic medications and burdens are not readily known in these populations. Other unknowns are the realistic estimate of benefit to the patient and treatment horizon (ie the length of time taken for the patient to benefit).[185]

A degree of clinical judgement and a 'best care given the circumstances' is required in these situations.

Optimise therapies

Polypharmacy (taking >5 medications) is one consequence of following single-disease guidelines in people with multimorbidity.[185,196–198]

Polypharmacy can be appropriate and has been said to be the price of success in creating effective treatments. It is also associated with higher rates of adverse drug events and hospitalisation, and is often particularly problematic in people who are physically frail[189] or have cognitive impairment.

Adherence to therapy can be much more difficult for patients taking numerous medications for multiple conditions.[199] Out-of-pocket costs for medication can be significantly higher for patients with diabetes than for most other chronic conditions[200] and the financial burden can lead to underuse of preventive services.[201,202]

Consideration of a home medicine review may assist in some cases.

Use strategies for choosing therapies that optimise benefit, minimise harm and enhance quality of life, particularly in older adults with multimorbidity.

Be aware of common comorbidities with diabetes

Macrovascular disease – includes CAD, hypertension, chronic heart failure, hyperlipidaemia and cerebrovascular disease. CVD is the primary cause of death for many persons with diabetes and is often found in patients with type 2 diabetes (refer to Chapter 9. Managing cardiovascular risk).

Painful conditions (acute and chronic) – are common in patients with type 2 diabetes. Peripheral neuropathies and arthritis account for most causes of pain, as well as tendinopathies.

Arthritis – is particularly problematic as it can reduce self-management capability (eg hand arthritis causing medication administration issues). Arthritis and tendinopathy (and any other cause of pain) can also affect the patient's ability to engage in physical activity.

Fractures – research has shown that overall fracture risks are significantly higher for men and women with type 2 diabetes. The increased risk of hip fracture has been observed despite patients having higher bone mineral density.

Obstructive sleep apnoea (OSA) – or sleep deprivation from any cause can aggravate insulin resistance, hypertension and hyperglycaemia. OSA is especially common in adults with diabetes (up to 17% of men).[203] The usual approach to obstructive sleep apnoea is diagnosis via a sleep study and management with individualised interventions including continuous positive airway pressure. Driver's licence requirements, particularly in commercial drivers, are particularly relevant.

Cancer – is the second largest cause of death in type 2 diabetes. A growing body of evidence suggests that diabetes and some antidiabetic treatments may increase cancer risk. Patients with diabetes should undergo appropriate cancer screening as recommended for all people in their age and sex. Patients should also try to reduce modifiable cancer risk factors, including quitting smoking, losing weight and increasing physical activity levels.[204]

Renal impairment – CKD affects approximately 40% of patients with diabetes. It is both a complication of diabetes (http://outpatient.aace.com/type-2-diabetes/management-of-common-comorbidities-of-diabetes) and an independent comorbidity present before diabetes onset. The presence of kidney disease worsens CVD risk and limits the number of glucose-lowering medication options available. Further, the availability of over-the-counter nephrotoxic medications (eg NSAIDs) can easily

exacerbate disease, and the 'triple-whammy' effect (ACEI/diuretic/NSAID) may go unrecognised without specific questioning. The onset of renal disease can be insidious.

Cognitive impairment – has been associated with type 2 diabetes[205,206] as well as a higher rates of dementia.[207] Recurrent symptomatic and asymptomatic hypoglycaemia have been suggested as possible causal links to this association.

Mental health issues – such as diabetes-related distress, depression and anxiety are common. Rates of depression are increased by 15% in people with diabetes compared with people without diabetes. An Australian study[208] using the Patient health questionaire-9 (PHQ-9) scale revealed moderate to severe depressive symptoms in 23% of patients with non-insulin treated type 2 diabetes, rising to 35% in those using insulin, with a proportion of these being undiagnosed. The odds ratio (OR) for depression in patients with type 2 diabetes compared with people without diabetes is higher in males (OR: 1.9; 95% confidence interval [CI]: 1.7–2.1) than females (OR: 1.3; 95% CI: 1.2–1.4).[209] Anxiety issues also affect people living with diabetes, and, as with depression, higher rates were seen in women.[208]

Mental health issues can adversely affect practitioner–patient communication, and the patient's ability to live and apply the principles of a diabetes management plan and glycaemic control, as well as add to the burden of disease and reduce quality of life. Depression and diabetes are also associated with a significantly increased all-cause and CVD-related mortality.

Some antipsychotic medications can increase the risk of developing diabetes. Olanzapine and clozapine are associated with higher rates of diabetes compared with other antipsychotic agents.

Dental problems – such as periodontitis (ie localised inflammation of the supporting structures of the teeth due to a chronic bacterial infection) are more common in patients with diabetes. Periodontitis can result in tooth loss and other dental complications that can interfere with the diet. Additionally, there is a two-way relationship between diabetes and periodontitis – the management of periodontitis may lead to a modest reduction in HbA1c of approximately 0.4%.[210–213] Inversely, improving glycaemic control may also improve the severity and complications associated with periodontitis.

Oral and periodontal health reviews should be incorporated into the systematic individualised care of patients with diabetes. Early prevention and intervention may prevent permanent dental loss and help aid in glycaemic control.

12.2 Medication complications

Clinical context

People with type 2 diabetes often take many medications – a significant proportion of patients take more than eight. There will always be a tension between multiple drug therapy to achieve recommended goals on the one hand and health issues from polypharmacy, drug–drug interactions and confusion (especially in the elderly) on the other. Refer to the Australian Institute of Health and Welfare (AIHW) for more information (www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442453548).

In 2007–08, 63% of people with diabetes reported using medications to help manage their condition; 13% of people with type 2 diabetes reported using insulin; and approximately 6% of people with diabetes reported using vitamin or mineral supplements or herbal remedies, although this is likely to be underestimated.[214]

Additionally, approximately 50% of patients do not take their medication doses exactly as prescribed by their healthcare professional.[215] This may be a barrier to achieving treatment targets and result in adverse outcomes.

It is important to understand the overall medication burden as it can lead to many issues including non-adherence, increased risk of falls and hypoglycaemia.

Contributing factors to non-adherence may include cost, complex treatment schedules and side effects.

In practice

GPs should be aware and assess for non-adherence, possible drug interactions and side effects in every patient with type 2 diabetes. Patients on prescribed monotherapy may be using complementary therapy or misusing their prescribed medication.

Medication use, both conventional and complementary, should be reviewed at least once per year as part of the annual cycle of care.[216,217]

The RACGP's *Putting prevention into practice: Guidelines for the implementation of prevention in the general practice setting*[218] provides some strategies to address patient adherence on pages 24–26. The full reference for the guide is available at www.racgp.org.au/your-practice/guidelines/greenbook

For information regarding drug interactions, consult publications such as the *Australian medicines handbook*.

Pharmacists can be an invaluable resource as they have access to extensive medications databases, can detect potential drug interactions, and provide useful advice to the health professional and person with diabetes.[219,220]

Important drug interactions and side effects

People with diabetes may be taking multiple glucose-lowering medications in addition to other prescription and non-prescription agents. Some drug interactions are dangerous and special care is required in older patients and patients with comorbidities such as renal impairment and autonomic neuropathy. A full list of potential drug interactions is beyond the scope of these guidelines. Use a home medicines review and pharmacy support on an individualised basis in any patient with diabetes.

Reporting of adverse events

Many medications are becoming available for the management of diabetes for which long-term safety data is not yet available. Primary healthcare professionals have an important role to play in identifying and reporting adverse events and possible drug interactions. These should be reported to the TGA, either online or by completing the Blue card (go to www.tga.gov.au/reporting-problems for further information).

Home medicines review

Multiple systematic reviews have concluded that there is a lack of evidence for improved health outcomes for medication reviews. Benefit has been proposed where pharmacists work in close liaison with primary care doctors.[221]

The review should include consideration of:

- the need for each medication
- issues around patient compliance and understanding of the medication
- enquiry regarding medication side effects, particularly falls and cognitive impairment
- the use of aids such as dosette boxes and Webster packaging.[222]

Complementary medicines

The use of complementary medicines is increasing in Australia. A survey of pharmacy customers found that 72% of respondents had used complementary medicines within the previous 12 months; 61% used prescription medicines daily and 43% had used these concurrently. Multivitamins, fish oils, vitamin C, glucosamine and probiotics were the five most popular complementary medicines.[223]

A substudy of the Fremantle Diabetes Study found that 23% of participants had consumed at least one complementary medication in the last year. Of the medications used, approximately 42% potentially necessitated additional patient monitoring or could be considered inappropriate for patients with diabetes.[216]

Predictable positive and negative interactions between complementary medicines and prescribed diabetes medications may be variable as there is little formal assessment of many of these products.

NPS MedicineWise has compiled a guide to medicines information resources relevant to Australian health professionals. This list is available at www.nps.org.au/health-professionals/guide-to-medicines-information-resources

Interactions can be checked by searching these resources, although NPS does not guarantee their completeness or accuracy.

13. Diabetes and reproductive health

13.1 Polycystic ovary syndrome

Clinical context

PCOS is a metabolic and endocrine disorder affecting about one in 15 women worldwide.[224] The endocrine disruptions consist of excessive androgen secretion or activity, and a large proportion of women also have insulin resistance and metabolic syndrome. The cause of PCOS is unknown, but studies suggest a strong genetic component that is affected by gestational environment, lifestyle factors or both.

Clinical manifestations include menstrual dysfunction, infertility, hirsutism, acne, obesity and glucose intolerance. Women with PCOS have an established increased risk of developing type 2 diabetes and a 2.4-fold increased odds of GDM, independent of age, race/ethnicity and multiple gestation.[225] Refer to Section 3.1. Identifying risk of diabetes in asymptomatic potients for identifying risk of diabetes in women with PCOS.

The diagnostic criteria of PCOS are hyperandrogenism, chronic anovulation and polycystic ovaries, after exclusion of other conditions that cause these same features (refer to Table 13).[226,227] A consensus definition of the disorder based on the importance of the three diagnostic criteria relative to each other remain controversial.

Table 13. Criteria for diagnosis of PCOS

The Rotterdam criteria are inclusive of National Institutes of Health (NIH) criteria; however, the Rotterdam criteria may not meet NIH criteria
The Rotterdam diagnostic criteria requires two of: • Oligo-ovulation or anovulation   • Clinical and/or biochemical signs of hyperandrogenism   • Polycystic ovaries   and exclusion of other aetiologies such as hyperthyroidism, hyperprolactinaemia, congenital adrenal hyperplasia, androgen-secreting tumours and Cushing's syndrome
The NIH diagnostic criteria requires: • Oligo-ovulation or anovulation   • Clinical and/or biochemical signs of hyperandrogenism   and exclusion of other aetiologies such as congenital adrenal hyperplasia, androgen-secreting tumours and Cushing's syndrome
Reproduced with permission from Teede HJ, Misso ML, Deeks AA, et al. Assessment and management of polycystic ovary syndrome: Summary of an evidence-based guideline. Med J Aust 2011;195 (6):65–112.

In practice

Lifestyle modification is the foundation of management. The principles are similar to those for diabetes prevention (ie weight control and ideally weight loss, support for a balanced individual healthy eating plan, increased physical activity).

There are several targeted interventions for other manifestations of PCOS.

Oligomenorrhoea and amenorrhoea

Options include:

- an oral contraceptive pill (OCP; low oestrogen doses [eg 20 μg] may have less impact on insulin resistance but also less impact on clinical hyperandrogenism)
- cyclic progestins (eg 10 mg medroxyprogesterone acetate, 10–14 days every two to three months)
- metformin (improves ovulation and menstrual cycles – though it is not PBS reimbursed for this option).[228]

Hirsutism

Choice of options depends on patient preference, impact on wellbeing, and access to and affordability of professional cosmetic laser therapy. Eflornithine cream can be added and may induce a more rapid response.

Pharmacological therapy is as follows:

- Primary therapy is the OCP.
- Anti-androgen monotherapy (eg spironolactone or cyproterone acetate) should not be used without adequate contraception. Therapies should be trialled for ≥6 months before changing dose or medication.
- Combination therapy – if ≥6 months of OCP is ineffective, add anti-androgen to OCP (twice daily spironolactone >50 mg or cyproterone acetate 25 mg/day, days one to 10 of OCP).[229]

Infertility

Patients and their partner may need advice and appropriate referral for fertility management.

13.2 Pregnancy with pre-existing diabetes

Recommendations	Reference	Grade*
Pre-pregnancy glycaemic control should be maintained as close to the non-diabetic range as possible, taking into account risk of maternal hypoglycaemia	65 SIGN, 2014	C
All women with diabetes should be prescribed† high-dose pre-pregnancy folate supplementation, continuing up to 12 weeks' gestation	65 SIGN, 2014	B
All women with pre-gestational diabetes should be encouraged to achieve excellent glycaemic control‡	65 SIGN, 2014	D
Postprandial glucose monitoring should be carried out in pregnant women with type 1 or 2 diabetes. Postprandial glucose monitoring should be carried out in pregnant women with gestational diabetes and may be considered in pregnant women with type 1 or 2 diabetes	65 SIGN, 2014	C
Pre-pregnancy care provided by a multidisciplinary team is strongly recommended for women with diabetes	65 SIGN, 2014	C

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

†5 mg of folate

‡HbA1c <48 mmol/mol (<6.5%) and consider stabilisation using metformin and/or insulin to achieve glycaemic targets. However, metformin has a category C rating in pregnancy. Continuation or initiation of metformin therapy should be considered only following full disclosure to the patient and under specialist supervision. Sulphonylureas may be associated with adverse neonatal outcomes and are thus best avoided[65,230–233]

All pregnant women with diabetes should be encouraged to achieve optimal glycaemic control.

Clinical context

GPs have a significant role in advising women of reproductive age with pre-gestational diabetes to consider the benefits of contraception to prevent inadvertent pregnancy before glycaemia can be optimised. Women should be advised of the need for advice, education and support to achieve optimal glycaemic control before pregnancy.[234] Women with type 2 diabetes and PCOS or irregular periods must be advised that improved fertility may accompany use of metformin. Pre-pregnancy counselling should include assessment of diabetes complication status, review of all medications and commencement of folic acid (5 mg). Poor glycaemic control at conception and early in pregnancy is associated with increased risk of congenital malformations and first trimester miscarriages.

Women with pre-gestational diabetes (types 1 and 2) are more prone to the complications of pregnancy such as higher rates of pre-eclampsia prematurity and caesarean section.[235] In addition, pregnancy may accelerate maternal complications of diabetes.[236] Both maternal and fetal complications are increased by diabetes. Risk is progressive with increasing glycaemia.[234]

Good glycaemic control can mitigate the risk of maternal and fetal complications and the likelihood of birth trauma, and reduce the risk of early induction of labour and need for caesarean section.

Refer to www.pregnancyanddiabetes.com.au for advice on pre-pregnancy blood glucose targets

In practice

Pre-pregnancy

Where possible and practicable, formal pregnancy planning should occur prior to pregnancy and be patient focused, support self management and involve a multidisciplinary team on an individual basis. Deferring pregnancy should be a recommendation until glycaemic control is optimal. Women should be reassured that any reduction in HbA1c towards the individualised target is likely to reduce the risk of congenital malformations. Medications should be reviewed and ceased or replaced as appropriate.

It is important to do the following:

- Advise that optimisation with a balanced diet, physical activity and healthy weight management may positively affect pregnancy outcomes.
- Advise that nausea and vomiting in pregnancy may affect blood glucose control.
- Advise that use of some form of contraception is recommended until glucose control is optimised.
- Aim for glycaemic control to be as close to the normal (non-diabetic) range as possible, ensuring risks of maternal hypoglycaemia are minimised. The risk of fetal abnormalities increases with higher HbA1c levels at the time of conception and during the first trimester.
- Review SMBG to determine if medication adjustment and/or commencement of insulin is required and assess risk of hypoglycaemia.
- Recommend higher folate supplementation (5 mg) per day, starting one month before pregnancy[237] and continuing until 12 weeks of gestation to reduce the risk of neural tube defects.

- Be aware that women treated for hypothyroidism may require higher doses of thyroid hormone replacement therapy.[238] Based on re-assessment, a suggested dose change is a 30% increase in dose (eg if on one tablet per day, to increase by two tablets per week) may be needed.

- Advise examination of the retina prior to conception and during each trimester for women with types 1 and 2 diabetes. More frequent assessment may be required if retinopathy is present. Patients with active moderate to severe non-proliferative retinopathy or with proliferative retinopathy who have not had an ophthalmological assessment within the preceding six months should undergo testing prior to pregnancy to see if the retinopathy is stable enough for pregnancy. Similarly, renal function should be tested if this has not been done within the preceding three months. Elevated creatinine or eGFR <45 mL/min/1.73 m^2 or an ACR >30 mg/mmol is an indication for pre-pregnancy nephrology review.[239,240]

- Counsel the patient that the risks associated with diabetes in pregnancy can be reduced but not eliminated.

In pregnancy

Specialist endocrine and obstetric referral for multidisciplinary shared care is considered best practice.

Safety and risks of medications before and during pregnancy

Consideration of the safety of current therapies should be undertaken ideally before pregnancy is planned or urgently once pregnancy is confirmed. Consultation with local specialist services is advised. Agents such as sulphonylureas, glitazones, SGLT2i and incretin-based therapies will need to be reviewed or ceased, and insulin therapy instituted.

Metformin

Metformin is not associated with an increase in congenital malformation or early pregnancy loss, but remains a category C classified drug (refer to Table 14 for further information).[241,242] Some diabetes services believe that metformin may be used as an adjunct or alternative to insulin in women with type 2 diabetes in the pre-conception period and during pregnancy. Consult with your local specialist endocrine and obstetric services.

Insulin

Rapid-acting insulin analogues aspart and lispro are safe to use during pregnancy. There is insufficient evidence about the use of the long-acting insulin analogues (glargine

– category B3). Detemir insulin (a long-acting insulin analogue) is now classified as category A drug in pregnancy. Patients already stabilised on insulin glargine may have this therapy continued in preference to switching to human insulin, but the B3 category rating needs to be discussed with the woman. Isophane insulin (Neutral Protamine Hagedorn [NPH] insulin) remains the most common long-acting insulin choice during pregnancy for women with type 2 diabetes (refer to Table 14 for further information).

Antihypertensive medications

ACEIs and angiotensin-II receptor antagonists should be discontinued during the pregnancy planning period or as soon as pregnancy is confirmed. Table 14 provides advice on antihypertensive agents to be avoided before and during pregnancy.

Table 14. Antihypertensive agents to be reviewed pre-conception and during pregnancy[243]

Antihypertensive agent	Category in pregnancy*	Advice
Angiotensin-converting enzyme inhibitors	D	Contraindicated
Angiotensin receptor blocker	D	Contraindicated
Calcium channel blocker	C	Avoid (except nifedipine)
β blockers	C	Avoid (except labetalol and oxprenolol)
Thiazide and loop diuretics	C	Seek advice
Methyldopa	A	Safe
Spironolactone	B3	Seek advice
Moxonidine	B3	Seek advice

*For definitions of the Australian categories for prescribing medicines in pregnancy, visit the Therapeutic Goods Administration, Australian categorisation system for prescribing medicines in pregnancy at www.tga.gov.au/australian-categorisation-system-prescribing-medicines-pregnancy

Statins

Statins should be discontinued during the pregnancy planning period or as soon as pregnancy is confirmed.

Antenatal care

Intensive glycaemic control guided by SMBG results is required in the management of diabetes in pregnancy. Insulin therapy will need regular review and titration to achieve glycaemic goals.

Close surveillance for new diabetes complications and monitoring of existing complications should occur routinely.

Ultrasound screening at 10–13 weeks' gestation (with biochemistry) for trisomies, and at 18–20 weeks for congenital cardiac and other malformations, is advised. Pregnant women with diabetes should be offered ultrasound monitoring of fetal growth and amniotic fluid volume every four weeks from 28 to 36 weeks.[240] Fetal growth and wellbeing monitoring should occur under specialist supervision. Consult with your local specialist endocrine and obstetric services.

Postpartum

The GP should maintain or re-establish contact with mother and child as early as practicable to address any issues arising from the pregnancy, labour, surgery or breastfeeding as well as review medications. Metformin may be continued while breastfeeding with minimal effect on the baby.[244] Glycaemic monitoring and medications (especially insulin) need careful review during breastfeeding to minimise the risk of hypoglycaemia. Re-establishing glycaemic management goals, re-assessment of complications and timely contraceptive advice are also appropriate in the postnatal period.

13.3 Gestational diabetes mellitus

Clinical context

Gestational diabetes, or GDM, is defined as glucose intolerance that begins or is first diagnosed during pregnancy. It may appear earlier, particularly in women with a high level of risk for GDM.

GDM generally develops and is diagnosed in the late second or early third trimester of the pregnancy. GDM affects about 9.6–13.6% of pregnancies in Australia.[245,246]

The reported prevalence of GDM varies for a number of reasons. One reason is the use of different screening and diagnostic criteria. The prevalence is also affected by maternal factors such as history of previous gestational diabetes, ethnicity, advanced maternal age, family history of diabetes, pre-pregnancy weight and high gestational weight gain. Mothers of different ethnicity born in areas with high diabetes prevalence such as Polynesia, Asia and the Middle East, are three times as likely to have GDM as mothers born in Australia. Among Aboriginal and Torres Strait Islander mothers, GDM is twice as common, and pre-gestational diabetes affecting pregnancy is three to four times as common as in non-Indigenous mothers.[245]

In pregnancy, there is a natural increase in levels of hormones including cortisol, growth hormone, human placental lactogen, and progesterone and prolactin levels, causing two to three fold increases in insulin resistance. The action of these hormones is usually compensated by increased insulin release. In pregnant women with abnormal glucose tolerance or impaired β-cell reserve, the pancreas is unable to sufficiently increase insulin secretion in order to control BGLs.

Potential maternal complications during pregnancy and delivery include pre-eclampsia and higher rates of caesarean delivery, maternal birth injury, postpartum haemorrhage.

For the neonate, complications can include macrosomia (large for gestational age) growth restriction, birth injuries, respiratory distress, hypoglycaemia and jaundice.

The precise level of glucose intolerance characterising GDM has been controversial due to differences in screening, diagnostic criteria and outcomes to which criteria are applied. The NHMRC criteria[25] were determined from glucose levels on antenatal glucose tolerance tests that were associated with subsequent development of diabetes in the mother. A recent Cochrane review[247] determined that increased levels of screening and management have not been clearly linked to improved health outcomes.

The Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study was published in 2008. This study reported a correlation between increasing maternal glucose levels at 24–32 weeks' gestation and a range of adverse maternal and fetal outcomes. The study suggested that the relationship between increasing BGLs and adverse effects was continuous, with no threshold or inflection point at which lower BGLs confer protection.

In response to the HAPO study, the International Association of the Diabetes and Pregnancy Study Groups (IADPSG) developed new consensus guidelines for the testing and diagnosis of GDM. Although The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG) and the Australasian Diabetes in Pregnancy Society (ADIPS)[248] have recommended that these consensus guidelines should be implemented, there has been controversy nationally and internationally.[249] Independent analysis of the HAPO data by the National Institute for Health and Care Excellence (NICE) guideline groups has suggested alternative diagnostic and management criteria that are not as low as IADPSG criteria,[240] and the UK has now developed its own diagnostic criteria.[240]

Ultimately, there is at present little evidence that clinical intervention is beneficial for the additional women identified by the new screening criteria.[250–253] As with any screening intervention, the evidence must be clear that the benefits outweigh potential harms.

Until this evidence is forthcoming, the original NHMRC recommendations for screening of GDM remain the preferred diagnostic criteria preferred by the RACGP.

There is still a need for further studies to clearly outline the evidence of the benefits and risks of altering diagnostic criteria, including the health economic costs of any such consensus for change.

Acknowledging that in Australian general practice there are alternative diagnostic criteria for GDM, the RACGP (preferred) and ADIPS (alternative) diagnostic criteria are presented in Box 9 and in Chapter 16. Issues under debate. Furthermore, it is important that each GP be aware of their obstetric service diagnostic criteria, and support and manage patients in a manner confluent with their specialist team guidelines to avoid conflict and patient confusion.

In practice

Screening for gestational diabetes mellitus

GDM is diagnosed by screening during pregnancy:

- All pregnant women should be screened between 26 and 28 weeks' gestation with a non-fasting glucose challenge.
- Women at high risk (refer to Box 3) should be screened at the first opportunity early in pregnancy and repeat if negative screening at 24–28 weeks.
- Women whose levels are ≥7.8 mmol/L should have a formal (fasting) 75 g OGTT.
- The diagnosis of GDM is made on the basis of a 75 g OGTT (Box 9).

Box 9. Screening and diagnosis of gestational diabetes mellitus	
The Royal Australian College of General Practitioners (RACGP; preferred criteria)	Australian Diabetes in Pregnancy Society (ADIPS; alternative criteria)
Fasting plasma glucose ≥5.5 mmol/L, or	Fasting plasma glucose 5.1–6.9 mmol/L, or
two-hour plasma glucose or random glucose ≥8.0 mmol/L	one-hour post ≥10.0 mmol/L, or
	two-hour 8.5–11.0 mmol/L

ADIPS (http://adips.org/information-for-health-care-providers-approved.asp) suggests a universal single step 75 g OGTT at 24–28 weeks.

GPs should be aware of the differences in the diagnostic and management algorithms of the RACGP (preferred) versus ADIPS (alternative) that are relevant to their local referral pathways for patients with gestational diabetes services and pragmatically manage patients within the context of these services.

Management

Recommendations	Reference	Grade*
Pregnant women with gestational diabetes mellitus should be offered dietary advice and blood glucose monitoring, and be treated with glucose-lowering therapy depending on target values for fasting and postprandial targets	65 SIGN, 2014	A

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

The basis for management for women with GDM includes nutritional therapy, weight optimisation, physical activity, blood glucose monitoring and insulin therapy.

All women with GDM should be offered education, blood glucose monitoring and dietary advice. Most GDM responds positively to lifestyle management.[254] Referrals to an APD and AEP are advised unless already provided by the obstetric services.

Limiting weight gain in pregnancy for obese or overweight women with GDM is desirable. It is recognised that glycaemic targets in the treatment of GDM vary between centres and clinicians around Australia. Targets suggested by ADIPS criteria have been described within Australia as being aggressive. Care should be taken in approaching these targets as most blood glucose monitors have a 5–10% margin of error and risks of maternal hypoglycaemia need to be considered.

Given the lack of agreement for treatment targets and the accuracy of blood glucose monitors, the RACGP suggests that readings between 4–6 mmol/L preprandially are reasonable.

Metformin has been used internationally[65] as initial glucose-lowering treatment in women with GDM. However, it has not been approved for this use in Australia for this indication. Lifestyle and insulin therapy remain the mainstay of therapy.

Close cooperation with the obstetric team is advised to monitor maternal and fetal welfare.

Hypoglycaemia may have serious effects on placental function and the fetus in patients with GDM. Thorough investigation is required in such patients.

Aim to achieve blood glucose levels:

- between 4 and 6 mmol/L preprandially
- <7 mmol/L two hours postprandially.

Follow-up of patients with a history of gestational diabetes mellitus

Recommendations	Reference	Grade*
Women with a history of gestational diabetes mellitus should receive a postpartum oral glucose tolerance test at 6–12 weeks	19 American Diabetes Association, 2015	E

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Box 10 provides the RACGP (preferred criteria) and ADIPS (alternative criteria) for follow up of patients with GDM history.

Box 10. Follow up of patients with gestational diabetes mellitus history

The Royal Australian College of General Practitioners (RACGP; preferred criteria)

75 g two-hour oral glucose tolerance test at 6–12 weeks postpartum

Thereafter, a fasting blood glucose or glycated haemoglobin (HbA1c) test every three years

Australian Diabetes in Pregnancy Society (ADIPS; alternative criteria)

75 g two-hour oral glucose tolerance test, at 6–12 weeks postpartum

The frequency and nature of this surveillance will depend on future pregnancy plans and the perceived risk of converting to type 2 diabetes. Women contemplating another pregnancy should have an oral glucose tolerance test annually

Although GDM usually resolves following birth, it is associated with increased risk for developing maternal type 2 diabetes in later life. The lifetime risk of developing type 2 diabetes following a diagnosis of GDM is 60%.[255]

After delivery, it is recommended that advice be given on healthy diet and exercise, which may include referral to a dietitian and physical activity program. Encourage increasing physical activity (eg 30 minutes brisk walking five times a week) and/or weight loss, which reduces risk of developing diabetes by 40–60% in those at high risk.[256]

Breastfeeding is encouraged for its many health advantages.

Women with normal glucose tolerance should be counselled regarding their risk of developing GDM in subsequent pregnancies.

13.4 Contraception

Contraception advice should follow guidelines that apply to women without diabetes. However, the benefits of the use of an intrauterine contraceptive device (IUCD) versus the less preferred, combined OCP, and any risks or contraindications caused by the presence of diabetes complications, need to be discussed with each patient on an individual basis. Smoking combined with diabetes and the use of the combined OCP significantly elevates risks.

Progesterone-only oral contraceptives may then be used as an alternative. Other contraceptive implants and intrauterine devices may also be an option on an individual basis.

13.5 Sexual problems – Men

Clinical context

Erectile dysfunction is a common problem for men with diabetes.

Men with diabetes are four times more likely to develop erectile dysfunction than men without diabetes. The prevalence in men aged >40 years with diabetes may be as high as 50% and incidence increases by approximately 10% per annum. Men with diabetes are also affected by erectile dysfunction at an earlier age, with occurrence approximately a decade earlier.[257,258]

Erectile dysfunction may be acute or chronic during periods of high blood glucose. Failure to achieve erection may be due to psychological causes, macrovascular disease or pelvic autonomic neuropathy.

An organic cause is more likely when there are other macrovascular or microvascular complications.

In addition, as a population/group/cohort, men with diabetes (types 1 and 2), have been shown to have lower testosterone levels than men without diabetes. This may contribute to reduced libido and aggravate or exacerbate erectile dysfunction.

In practice

It is important to enquire about erectile dysfunction in the annual review.

Differentiate organic and psychological erectile dysfunction by taking a detailed history such as spontaneous early morning erections, anorgasmia and lack of libido.

Assessment of severity and management of psychological (anxiety and depression) and physical symptoms.

Psychological therapies such as supportive counselling for patients with organic erectile dysfunction and behavioural therapy for psychogenic erectile dysfunction are useful.[259]

Phosphodiesterase inhibitors are useful and side effects are generally mild. Concomitant use of vasodilating nitrates are contraindicated due to life-threatening hypotension.

The help of a urologist who specialises in erectile dysfunction should be sought for those considering penile injection with vasoactive agents such as prostaglandin E1 (alprostadil), surgical treatments, vacuum devices, penile prostheses or implants may help.

Potential CVD risk of engaging in and resuming sexual activity needs to be discussed.

13.6 Sexual problems – Women

Clinical context

The prevalence of sexual dysfunction among women appears to be lower than among men (although studies have typically focused more on men than women), some studies show that women may experience more psychological problems compared with men, who have more physical symptoms.

Women with diabetes may also experience higher rates of sexual dysfunction than women without diabetes.

Symptoms of sexual dysfunction in women include:

- decreased or total lack of interest in intimacy or sexual relations
- decreased or no sensation in the genital area
- a degree of anorgasmia
- dryness in the vaginal area (presumably due to pelvic autonomic neuropathy), leading to dyspareunia.

Genital infections such as monilial vaginitis occur more frequently in women with diabetes and may contribute to sexual dysfunction.

Rates of depression, anxiety and psychological distress are higher in people with diabetes and may contribute to sexual dysfunction in men and women.[95,260]

In practice

It is important to enquire about sexual problems in the annual review and manage physical and emotional aspects.

14. Management of other impacts of diabetes

14.1 Sick day management

Recommendations	Reference	Grade*
Patients should be educated to develop a sick day management plan after initial diagnosis. This plan should be reviewed at regular intervals	261 Australian Diabetes Educators Association, 2014	None provided
Assist in the development of a sick day care plan and preparation of a home sick day management kit for patients to use during episodes of sickness	261 Australian Diabetes Educators Association, 2014	None provided

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

Patients with diabetes require careful individualised management during times of illness (due to other causes) to prevent:

• hyperglycaemic and hypoglycaemic emergencies

• hyperosmolar hyperglycemic state

• DKA – uncommon (plus consider normoglycaemic DKA with SGLT2i medication).

A clear and specific action plan ensures that patients can either self-manage or have access to their healthcare team for advice and early intervention, supervision and support. General practices and GPs should consider routinely incorporating these plans as part of a patient's documented management plan.

The ADEA has developed clinical guiding principles for health professionals and a consumer resource on sick day management.[261,262] These are available at www.adea.com.au/about-us/our-publications

Patient information is also available from state and territory diabetes organisations.

In practice

Sick day management should be tailored to the individual patient and incorporate the following actions:

- Identify the underlying cause and treat as appropriate. Underlying causes include:
  - intercurrent illnesses, infections (eg skin, urinary tract and chest infections), trauma, acute MI and stroke
  - use of medications such as corticosteroids.
- An increase in SMBG may be required according to individual circumstances such as those patients at risk of hypoglycaemia or using insulins.
- Ensure continuity of advice and accessibility – provide telephone access or after-hours support.
- Review medications – Refer to Table 15.
- A written action plan (refer to Table 15), which should be regularly updated (at least once annually during the annual cycle of care) and provided to patient and carer.

Table 15. Action plan

Commence action plan	• When feeling unwell • Blood glucose >15 mmol/L on two consecutive readings
Frequent monitoring of blood glucose	Two to four hourly monitoring, or more frequently if blood glucose is low
Medication	Insulin or diabetes medications should be continued but with assessment on the use of metformin, sodium glucose co-transporter 2 (SGLT2) inhibitors (dapagliflozin, canagliflozin and empagliflozin) and glucagon-like peptide-1 receptor agonist (GLP-1 RA), which may require cessation if vomiting or dehydration is a concern. Increased risks of hypoglycaemia may occur if appropriate intake of meals are not able to be maintained.
Food and water intake	• Patients should try to maintain their normal meal plans if possible • Fluid intake (eg water) should be increased to prevent dehydration • Advise about alternative easy-to-digest foods like soups if the patient cannot tolerate a normal diet (some non-diet soft drinks may provide essential carbohydrate in this situation) • If blood glucose >15 mmol/L use non-glucose containing fluids • If blood glucose <15 mmol/L use oral rehydration solutions (may contain glucose) if needed • If unable to tolerate oral fluids and blood glucose continues to drop – inform patient to attend medical care

| Seek assistance | Individuals and support people need to assess whether they are well enough or able to follow the guidelines |
| | If not, they should call for help or attend hospital |

Practice points from ADEA – Clinical guiding principles for sick day management of adults with type 1 and type 2 diabetes – Technical document[261]

Special considerations

Different patient groups will need individualising of the sick day action plan

Managed with diet alone:

- Worsening control may require the introduction of medication and symptomatic management of hyperglycaemia.
- Patients with type 2 diabetes may have impaired body immune mechanisms that will make recovery slower.
- In addition, patients may become dehydrated because of the osmotic diuresis.

Managed with oral or non-insulin glucose lowering medication:

- Worsening control may require the urgent review by the GP or referral to a specialist diabetes service or endocrinologist.
- Consideration of the use of insulin may be temporarily required for persistent and resistant symptomatic hyperglycaemia (this may require hospital admission).
- In patients with nausea, vomiting and/or diarrhoea, consider stopping metformin and GLP-1 mimetics temporarily as metformin may aggravate these symptoms and GLP-1 mimetics may aggravate nausea/vomiting, and there may be a risk of acute renal impairment due to dehydration. Cessation of any SGLT2 inhibitor should be reviewed if gastrointestinal illnesses are present as they may further aggravate dehydration and hypovolaemia.

Type 2 diabetes managed on insulin:

- All patients should be advised to seek the urgent review by their GP or health professional when unwell or the blood glucose is consistently >15 mmol/L on two consecutive SMBG readings as per the action plan. Blood glucose monitoring should be increased to every two to four hours if unwell. Depending on these levels, patients may need to increase their morning intermediate or long-acting insulin dose by 10–20% if the glucose reading remains elevated and, depending on further blood glucose levels, modify subsequent doses of short-acting insulin during the day. Advice on the additional use of oral agents and GLP-1 RA is listed

above. Additional blood ketone testing may be incorporated if there are symptoms suggestive of ketosis (eg nausea, vomiting, shortness of breath or fruity odour, abdominal pains, altered consciousness) or there is a past history of DKA, or if the patient is using an SGLT2 inhibitor agent. This must be a documented strategy on their sick day management plan. Note: many patients are only on basal insulin or a premixed insulin. These patients require appropriate medical advice and access to additional rapid-acting insulin to use as a supplemental insulin dose.[262]

- Patients with gastrointestinal upset who are not eating, but who feel well and continue their usual activities, may need to reduce their insulin based upon their SMBG readings (especially rapid-acting insulin) to avoid hypoglycaemia.

- A warning for the use of SGLT2 inhibitors: there have been rare reports of euglycaemic DKA with all the SGLT2 inhibitors – symptoms of possible DKA need to be incorporated in clinical management when considering use of these agents. These agents are not indicated in type 1 diabetes.

14.2 Planned surgical procedures

People with diabetes should be seen several weeks before surgery for an assessment of glycaemic control and anaesthetic suitability, including their CVD risks and any treatment modifications instituted and stabilised before proceeding to surgery. Attaining glycaemic control (ie an HbA1c approaching 53 mmol/mol or 7%) in the preoperative period has been shown to result in fewer complications and shorter hospital stays after surgery.[169] A patient with a HbA1c of >75 mmol/mol (>9%) may need to have their surgery delayed until glycaemic management is optimised.

Preoperative care is the same for minor and major surgery, but blood glucose levels should be monitored intra-operatively (a prolonged procedure) and postoperatively for several days. Insulin may be required postoperatively for some people with type 2 diabetes.

In practice

Appropriate written instructions should be given to the patient beforehand.

Patients who are prescribed oral glucose-lowering medications (eg metformin, sulphonylureas, acarbose, glitazones, SGLT2i, DPP-4i) as well as injectable GLP-1 RA such as exenatide:

- can continue their diabetes medications on the day prior to surgery – be aware that gastric emptying is affected by GLP-1 agonists

- on the advice of their anaesthetic team, may omit their oral glycaemic medications on the morning of surgery, irrespective of whether they are on the morning or afternoon list. Insulin requires individualised advice and is usually not completely omitted. Proactively seek specialist endocrinology and anaesthetic advice before planned procedures

- can generally recommence medications when they are able to eat meals – specific advice is available in Australian Diabetes Society's *Peri-operative diabetes management guidelines*, available at https://diabetessociety.com.au/documents/ PerioperativeDiabetesManagementGuidelinesFINALCleanJuly2012.pdf

Metformin can generally be recommenced 24 hours after major surgery provided that there has been no deterioration in serum creatinine.[169] For patients pre-operatively and post-operatively using metformin and SGLT2 inhibitors, maintenance of hydration is important.

Patients with diabetes who are treated with insulin will usually require peri-operative insulin and glucose infusions, and close blood glucose monitoring. Many hospitals have a protocol or working plan that should be followed for the individual patient in that service.

For colonoscopy preparation,[263] colonlytely or glycoprep rather than fleet or phosphoprep should be used in patients with renal impairment who may become severely hyperphosphataemic with phosphate preparations. Modifications of diet advised for colonoscopy preparation may alter glucose management and hypoglycaemic risks, so instruction on appropriate SMBG testing may be required.

14.3 Driving

Diabetes is identified as one of the medical conditions that may impair driving ability. Diabetes and diabetes medications may alter the capacity to drive safely.

Impairment can occur due to unexpected hypoglycaemia (main hazard) for drivers with type 2 diabetes on glucose-lowering medications and/or sensory or end-organ complications, particularly reduced vision and sensation in the feet. Other comorbidities such as sleep apnoea and cardiovascular problems have substantial implications.

Drivers with diabetes must meet specific national standards. Certain criteria must be met to ensure that their health status does not increase the risk of a crash. Medical assessment should include:

- therapeutic regimens (eg diabetes treated by any glucose-lowering agents including insulin)
- commercial or private standards
- satisfactory control of diabetes
- hypoglycaemic unawareness
- recent severe hypoglycaemic event
- comorbidities and end-organ complications.

In practice

Evaluate patients with diabetes on their capacity to drive against national standards.

Medical standards for licensing and clinical management guidelines for commercial and private vehicle drivers are contained in the Austroads and National Transport Commission document *Assessing fitness to drive for commercial and private vehicle drivers*, available at www.austroads.com.au/drivers-vehicles/assessing-fitness-to-drive[264] A flowchart to assist with the management of diabetes and driving can be found on page 59 of the above document. The document is currently being updated and the new version will be published on 1 October 2016.

As each state and territory has differing medical assessment instructions, check with the relevant transport authority for requirements in respective states.

Specialist referral is usually required for commercial licences if the patient is on oral glucose-lowering agents or insulin therapy. Licensing review periods are also determined by therapeutic regimes. People with diabetes on commercial licences are subject to yearly specialist review if they are on any form of glucose-lowering therapy, unless they are on metformin alone, where ongoing fitness to drive may be assessed by the treating GP by mutual agreement with the treating specialist. The initial recommendation of a conditional licence must, however, be based on the opinion of a specialist in diabetes.

An HbA1c level of ≥75 mmol/mol (≥9.0%) should not be used administratively by licensing authorities to deny eligibility for a licence in the absence of a medical review. There is no strong evidence of high average blood glucose levels and driving risk. The new national standards, which is due for publication later in 2016, will likely not include an upper HbA1c threshold of concern regards driving requirements.

These are national standards, so it is important to contact the driving authority in individual states and territories as variations to the national standards do exist.

The *Diabetes and driving: Above 5 to drive!* consumer booklet (available at https://static.diabetesaustralia.com.au/s/fileassets/diabetes-australia/bee30f0d-9b45-49f0-9800-5d66ee1f49d9.pdf) provides a checklist and offers advice for people with diabetes to ensure that they have safe blood glucose levels before they drive. The importance of taking extra precautions to maximise road safety and reduce risks of road accidents caused by hypoglycaemic incidents is highlighted and should be actively promoted.

For example, drivers are required to perform a blood glucose check before they drive and again during the journey if driving for more than two hours.

14.4 Diving

People with type 2 diabetes, including those who use medication (eg oral glucose-lowering agents, insulin), can participate in recreational scuba diving. They must be otherwise qualified to dive and meet several criteria as outlined in consensus guidelines for recreational diving with diabetes that was developed in 2005. For more information, visit www.diversalertnetwork.org/medical/articles/DAN_and_UHMS_Publish_Guidelines_for_Recreational_Diving_with_Diabetes

When evaluating persons with diabetes for medical fitness to dive, first ensure that no other exclusionary conditions (eg epilepsy, pulmonary disease) exist.

The physiological demands of diving must then be considered. People with diabetes are at higher risk than the general diving population of medical complications such as MI, angina and hypoglycaemia.

14.5 Travel

People with diabetes can travel safely, provided a few extra precautions are taken and the travel is planned.

Those not using insulin generally have few problems during travel. The stress of travel may increase BGLs slightly. The decreased activity experienced in a long plane trip, together with the amount of food given en route often results in increased BGLs. These return to normal once a more usual lifestyle has been resumed at the destination.

Extra precautions before and during travel include:

- a medical consultation at least six weeks before the proposed travel to allow time to assess control and alter management as required

- checking of routine immunisation status and other medical conditions
- having a covering letter from their doctor and extra supplies of food, medication and monitoring equipment
- getting advice about special insurance
- finding out about Australian air security guidelines.

Australian air authorities stipulate the following security guidelines. If the patient is not using an Australian carrier, it is advisable for the patient to check with the chosen airline for applicable security guidelines.

- All diabetes supplies including testing equipment, insulin and glucagon delivery devices (eg syringes, pen needles, insulin pump consumables) carried on board must be in the hand luggage of the person who has diabetes and whose name appears on the airline ticket. It is not advisable to pack extra insulin in checked-in luggage as insulin exposed to extreme temperatures of the aircraft holds will lose efficacy.
- The traveller's name should appear on the insulin and/or glucagon prescription labels.
- It is advisable to carry legible prescriptions for all medications. The prescriptions must include the traveller's name, name and type of medication, and contact details of attending medical practitioner.
- The NDSS card is accepted as primary proof that a person with insulin-treated diabetes needs to carry with them their diabetes equipment such as insulin pen, pump, syringes, needles and glucagon kit. Supplementary photographic proof of identity such as a driver's licence may also be requested.
- It is advisable to carry a letter from the attending medical practitioner that outlines medical diagnoses, prescribed medications, if insulin is used and, if so, the delivery device(s). The letter must stress the importance of the patient having to carry medications with them and include the frequency of dosage. For those using an insulin pump, the letter must stress the need for the pump to be worn at all times.
- Some international regulations set limits on fluid containers that may be personally taken on board aircraft. People with diabetes who need to carry supplies of insulin are exempt. They will be required to present the insulin at the security point and carry proof of their condition and need for insulin.
- People wearing electronic devices to monitor blood glucose levels or to infuse insulin should check with the airline as to whether these devices can be operated during the flight.

Rights of people with diabetes during security check

People with diabetes who use an insulin pump are not required to remove their pump at the security point. If the security staff request this, the person with diabetes has the right to request access to a private consultation room, which security staff are required to provide. People with diabetes are also entitled to make this request if discussion about their condition is required.

For more information about travel and diabetes, visit www.diabetesaustralia.com.au/travel or http://travelsecure.infrastructure.gov.au and scroll down to 'special needs'.

15. Diabetes and end-of-life care

Recommendations	Reference	Grade*
To minimise the risks of hypoglycaemia and metabolic compensation, a blood glucose range of 6–15 mmol/L is appropriate for most palliative care patients	265 Diabetes UK, 2013	None provided
Maintain at glycated haemoglobin (HbA1c) at no lower than 58 mmol/mol (7.5%) if on hypoglycaemic medication depending on the individual's life expectancy, as HbA1c will be less relevant in patients with months or days left to live	265 Diabetes UK, 2013	None provided

*Refer to Summary, explanation and source of recommendations for an explanation of the level of evidence and grade of evidence

Clinical context

The aim of glycaemic control in patients at the end of life changes from preventing and managing long-term complications of diabetes to preserving quality of life.

Terminally ill patients often have multiple factors affecting their glycaemic control (refer to Box 11). Glucose-lowering therapy should be tailored to minimise the risks of hypoglycaemia and hyperglycaemic states and symptoms.

> ### Box 11. Factors affecting glycaemic control in patients with type 2 diabetes at end of life
>
> - Stress response to severe or sustained illness
> - Organ failure
> - Malignancy
> - Chemotherapy
> - Use of steroids
> - Frequent infections
> - Poor appetite/smaller meals
> - Poor nutrition
> - Cachexia
> - Dehydration
> - Difficulty taking medications (eg difficulty swallowing, nausea, stress)
> - Weight loss

Hyperglycaemia can worsen pain, confusion, thirst, cognition, confusion and incontinence. Blood glucose levels >15 mmol/L may cause polyuria and increase risks of infection.

Hypoglycaemia can also cause discomfort, confusion and impaired cognitive function. DKA can mimic terminal illness. If not recognised and treated, it can severely impair quality and even duration of life.

Although there is little evidence about optimal blood glucose range, it is generally agreed that a range of 6–15 mmol/L is appropriate for most palliative care patients to optimise patient wellbeing and cognitive function.[266,267]

In practice

Aim to provide an appropriate level of intervention according to stage of illness, symptom profile and respect for dignity. In most cases, tight glycaemic control to meet general targets is no longer appropriate in patients nearing the end of life.

Ideally, discuss dying with patients and their families prior to the need for end-of-life care so that the important considerations can be addressed in advance care planning.[268] Liaison with the palliative care team and community diabetes team is recommended as part of a multidisciplinary approach to end-of-life diabetes care.[269]

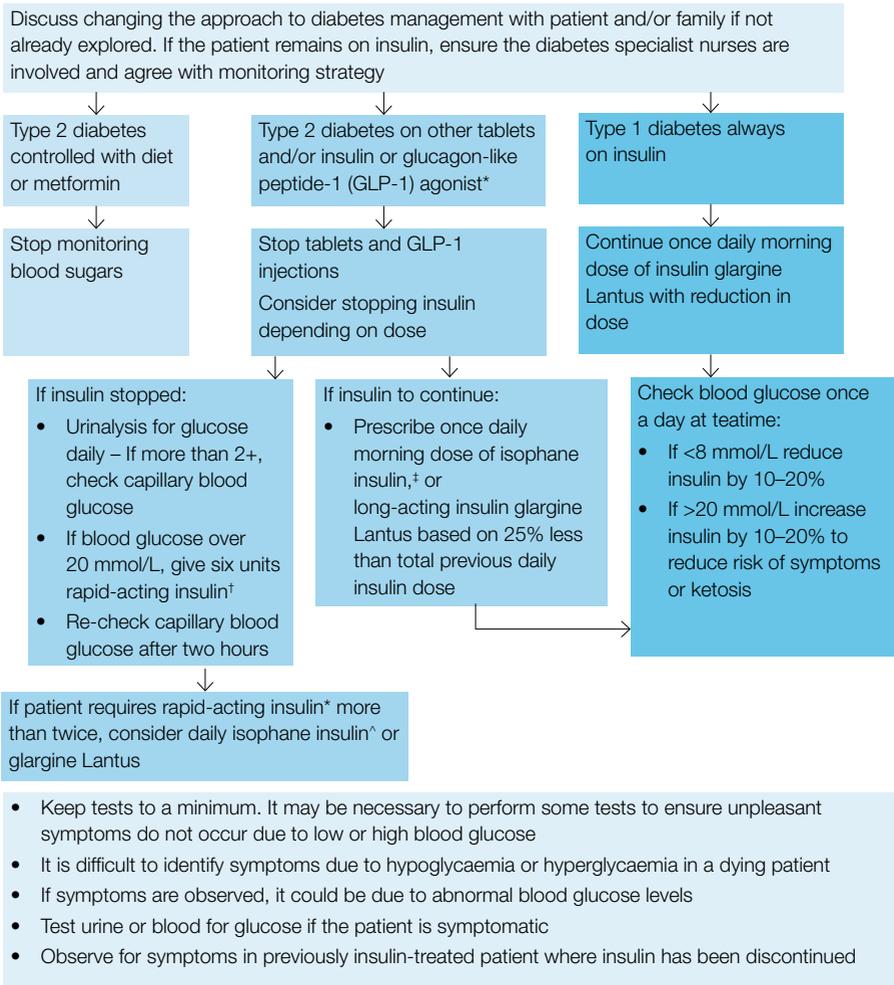## Diabetes medications at end of life

Insulin alone is a simpler option for patients and their carers than combinations of tablets and insulin. Consider switching patients from combinations to insulin alone, once or twice daily. Patients on insulin with poor intake will need lower doses.

Avoid long-acting sulphonylurea preparations (eg glibenclamide, glimepiride) if small meals are being taken.

Figure 7 shows an algorithm for an end-of-life diabetes care management strategy.[265]

Consider referral to specialist care for assistance with complex treatment decisions such as frequent hypoglycaemia, use of insulin or managing the effects of steroids on glycaemia.

Figure 7. Algorithm for an end-of-life diabetes care management strategy

Discuss changing the approach to diabetes management with patient and/or family if not already explored. If the patient remains on insulin, ensure the diabetes specialist nurses are involved and agree with monitoring strategy

Type 2 diabetes controlled with diet or metformin

Type 2 diabetes on other tablets and/or insulin or glucagon-like peptide-1 (GLP-1) agonist*

Type 1 diabetes always on insulin

Stop monitoring blood sugars

Stop tablets and GLP-1 injections

Consider stopping insulin depending on dose

Continue once daily morning dose of insulin glargine Lantus with reduction in dose

If insulin stopped:
- Urinalysis for glucose daily – If more than 2+, check capillary blood glucose
- If blood glucose over 20 mmol/L, give six units rapid-acting insulin†
- Re-check capillary blood glucose after two hours

If insulin to continue:
- Prescribe once daily morning dose of isophane insulin,‡ or long-acting insulin glargine Lantus based on 25% less than total previous daily insulin dose

Check blood glucose once a day at teatime:
- If <8 mmol/L reduce insulin by 10–20%
- If >20 mmol/L increase insulin by 10–20% to reduce risk of symptoms or ketosis

If patient requires rapid-acting insulin* more than twice, consider daily isophane insulin^ or glargine Lantus

- Keep tests to a minimum. It may be necessary to perform some tests to ensure unpleasant symptoms do not occur due to low or high blood glucose
- It is difficult to identify symptoms due to hypoglycaemia or hyperglycaemia in a dying patient
- If symptoms are observed, it could be due to abnormal blood glucose levels
- Test urine or blood for glucose if the patient is symptomatic
- Observe for symptoms in previously insulin-treated patient where insulin has been discontinued

*Byetta (Exenatide)/Victoza, (Liraglutide), Lyxumia (Lixisenatide)

†Humalog/Novorapid/Apidra

‡Humulin I/Insulatard/Insuman Basal

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16. Issues under debate

Issue – Diagnostic criteria for GDM
ADIPS guidelines significantly lower diagnostic threshold
ADIPS guidelines are not accepted by the RACGP due to the need for high-quality evidence and implications for resource utilisation[270]

Issue – Postnatal screening for type 2 diabetes mellitus
ADIPS guidelines have significantly increase screening frequency
ADIPS guidelines are not accepted by the RACGP due to the need for high-quality evidence and implications for resource utilisation
Screening remains as in the RACGP's *Guidelines for preventive activities in general practice*, 8th edition (Red Book) – fasting blood glucose every three years
Postpartum oral glucose tolerance test should be performed every three years

Comparison tables of RACGP (preferred) and ADIPS (alternative) criteria

Screening and diagnosis of gestational diabetes mellitus (on the basis of a 75 g oral glucose tolerance test)

The Royal Australian College of General Practitioners (RACGP; preferred criteria)	Australian Diabetes in Pregnancy Society (ADIPS; alternative criteria)
Fasting plasma glucose ≥5.5 mmol/L, or two-hour plasma glucose or random glucose ≥8.0 mmol/L	Fasting plasma glucose 5.1–6.9 mmol/L, or one-hour post ≥10.0 mmol/L, or two-hour 8.5–11.0 mmol/L

Follow-up of patients with a history of gestational diabetes mellitus

The Royal Australian College of General Practitioners (RACGP; preferred criteria)	Australian Diabetes in Pregnancy Society (ADIPS; alternative criteria)
75 g two-hour oral glucose tolerance test at 6–12 weeks postpartum	75 g two-hour oral glucose tolerance test, at 6–12 weeks postpartum
Thereafter, a fasting blood glucose or glycated haemoglobin (HbA1c) test every three years	
The frequency and nature of this surveillance will depend on future pregnancy plans and the perceived risk of converting to type 2 diabetes. Women contemplating another pregnancy should have an oral glucose tolerance test annually	

In patients with GDM, hypoglycaemia may have serious effects on placental function and the fetus. Thorough investigation is required in such patients.

Aim to achieve blood glucose levels:

- between 4 and 6 mmol/L preprandially
- <7 mmol/L two hours postprandially.

Issue – Blood pressure targets in diabetes management

Guidelines routinely advocate blood pressure (BP) target of systolic blood pressure (SBP) ≤130 mmHg

BP-lowering reduces cardiovascular events and mortality in people with type 2 diabetes. However, the target levels for BP therapy have been based on little direct evidence. Meta-analyses demonstrate that more intensive BP control (SBP ≤130 mmHg) was only associated with further reduction in stroke. A 40% increase in serious adverse events was observed

The target level for optimum BP remains controversial

A number of international guidelines have changed their blood pressure targets to <140/90 mmHg, while others remain at <130/80 mmHg. The target levels for BP therapy have been based on little direct evidence. A number of meta-analyses have demonstrated that the benefits of intensive BP control needs to be balanced with the risks. One meta-analysis demonstrated that more intensive BP control (SBP ≤130 mmHg) compared with usual (<140/90 mmHg) was associated with further reduction in stroke only, but there was a 40% increase in serious adverse events.[153] Two additional meta-analyses have recently been published. The analysis by Emdin at al[48] found that risk reduction was attenuated in SBP <140 mmHg. However, there did appear to be lower risk of stroke, retinopathy and albuminuria when blood pressure was reduced to <130 mmHg. A more recent meta-analysis, however, found that treatment of SBP <140 mmHg was associated with increased cardiovascular disease (CVD) death.[154] This may in part be related to the selection of trials in this analysis, which included patients with comorbidites such as chronic kidney disease (CKD), heart failure and CVD[155]

In line with these findings, it would be reasonable for GPs to shift the BP target to <140/90 mmHg for people with diabetes, with lower targets considered for younger people and those at high risk of stroke (secondary prevention) as long as treatment burden is not high. The target BP for people with diabetes and microalbuminuria or proteinuria remains <130/80 mmHg. As always, treatment targets should be individualised and people with diabetes monitored for side effects from use of medications to achieve lower targets

Issue – Screening for adults aged 40–70 years who are overweight or obese

The US Preventive Services Task Force (USPSTF) has recently recommended screening for abnormal blood glucose as part of cardiovascular disease (CVD) risk assessment in adults aged 40–70 years who are overweight or obese. Clinicians should offer or refer patients with abnormal blood glucose to intensive behavioural counselling interventions to promote a healthful diet and physical activity (USPSTF Grade B recommendation)[271]

This recommendation applies to such adults seen in primary care settings who do not have symptoms of diabetes. The target population includes persons who are most likely to have glucose abnormalities that are associated with increased CVD risk and can be expected to benefit from primary prevention of CVD through risk factor modification

Issue – Target HbA1c levels

The National Institute for Health and Clinical Excellence Quality and Outcomes Framework (NICEQOF) in the UK changed the glycated haemoglobin (HbA1c) target from 7.0% to 7.5% because of the several large trials showing harm with a target that is too low. Because of the measurement error, a range around that mean of, for example, 6.5–8% would be needed. This will allow for measurement variation as well as some individualisation and negotiation with the patient, in a more person-centred approach

Issue – Other

- Is an estimated glomerular filtration rate (eGFR) 45–60 mL/min/1.73 m² of any clinical consequence?
- Use of the Problem areas in diabetes (PAID)/Patient health questionnaire-2 (PHQ-2) tools to detect depression and distress in diabetes and linkage to long-term improved outcomes and complication reduction
- Are there possible benefits of low carbohydrate diets in diabetes management?[272]
- Reduction in carbohydrate intake has been shown to translate into lower glycaemic excursions and lower overall glycaemic load[273–275]
  - A recent study has highlighted the benefits of a very low carbohydrate, high unsaturated fat, low saturated fat diet versus a high carbohydrate, low fat diet. The investigators evaluated weight loss, glycaemic control and cardiovascular disease risk factors in patients with type 2 diabetes after 52 weeks. With the likely pathophysiological interaction between carbohydrate and hyperglycaemia, this approach may be considered when more evidence is available.[276] The definition of a low-carbohydrate diet varies across the spectrum of studies, causing difficulty in generalising results
  - Care may need to be exercised with patients on sodium glucose co-transporter 2 (SGLT2) inhibitors due to risks of ketoacidosis[277]

Appendix A. Accessing government support for diabetes care in general practice

Support for developing management plans and organising team care

The Australian government supports general practices that are taking a high-quality and proactive approach to diabetes care through Medicare Benefits Schedule (MBS) payments to general practitioners (GPs), nurses, allied health professionals and general practices. These include the chronic disease management (CDM) items (formerly enhanced primary care), Service Incentive Payments (SIPs) and the Practice Incentives Program (PIP).

The CDM items provide support for developing management plans and organising team care.

General practice management plan (GPMP, Medicare item number 721)

These are documented plans developed together by the GP and the patient. They incorporate the patient's needs and goals, how these are to be achieved, and reference to any resources used. Templates are available via medical software and various general practice networks and Primary Health Networks (PHNs).

Payments are made for development and for structured reviews of GPMPs.

Team care arrangement (TCA, Medicare item number 723)

These are expansions of the GPMP that detail allied healthcare professionals and other members of the team who implement any part of the GPMP. This includes active participation by at least two other providers who contribute to the GPMP or TCA and the goals of management for the patient.

Both GPMPs and TCAs can have practice nurses or similar practice team members involved in their development.

Payments are made for development and for structured reviews of the GPMP and TCA under MBS item number 732.

There is evidence that GPMPs that are reviewed on a regular basis can result in improvement in process and clinical outcomes.[17] Medicare payments also support the involvement of suitably qualified allied health members in providing care as documented in the TCA. Up to five treatments per year are subsidised at the time of publication.

Medicare has strict eligibility criteria for claims for MBS items 721 and 723, and the structured reviews under MBS item 732. Further information is available at www. health.gov.au/internet/mbsonline/publishing.nsf/Content/Medicare-Benefits-Schedule-MBS-1

Support for the annual cycle of care

The annual cycle of care is a method of incentivising quality diabetes care. However, the scope of the annual cycle of care recommendations is less than the guidelines' recommendations.

Completion of an annual cycle of care requires assessment of a number of parameters (refer to Table A.1), and the professional attendance and appropriate documentation by a GP, with any clinically relevant issues including:

• taking a patient history

• performing a clinical examination

• arranging any necessary investigation

• implementing a management plan

• providing appropriate preventive healthcare.

Patients and practitioners need to discuss desired outcomes and agree on goals to achieve these. An example of a structured patient-centred care plan is provided in Appendix B. Structured patient-centred care plan.

For practitioners – Support payments are provided for completing the annual cycle of care. When a patient with diabetes completes their annual cycle of care, their GP notifies Medicare and is paid a SIP.

For practices – When more than 50% of practice patients with diabetes have completed their annual cycle of care, practices are automatically paid a quality outcome payment. This is calculated by Medicare and is dependent on the number of SIP payments claimed by GPs.

Table A.1. Medicare Benefits Schedule item number 2517 – Minimum requirements of care to complete an annual diabetes cycle of care for patients with established diabetes mellitus

Minimum requirements	Frequency
Weight and height plus body mass index (BMI)	At least twice every cycle of care
Blood pressure index	At least twice every cycle of care
Feet examination	At least twice every cycle of care
Measure total cholesterol, triglycerides and high-density lipoprotein-cholesterol (HDL-C)	At least once every year
Glycated haemoglobin (HbA1c)	At least once every year
Microalbuminuria	At least once every year
Estimated glomerular filtration rate (eGFR)	At least once every year
Self-care education, diet, physical activity, smoking evaluation	At least once every year
Medication review	At least once every year
Ensure that a comprehensive eye examination is carried out at least once every two years	At least once a year if complications are detected

NB: A new item on the Medicare Benefits Schedule (MBS) for retinal photography with a non-mydriatic retinal camera will be available for general practice use from November 2016. The listing is expected to benefit Aboriginal and Torres Strait Islander peoples and communities in rural and remote locations, where there is limited access to optometric and ophthalmic services to diagnose diabetic retinopathy

Appendix B. Structured patient-centred care plan – Example of a General practice management plan and Patient care plan

[Insert practice letterhead]

General practice management plan (GPMP) (MBS item number 721 – Diabetes)	
Patient name:	Date of birth:
[Full name]	[Date of birth]
Contact:	Medicare or private health insurance:
[Address]	[Medicare number]
[Telephone]	[Private health insurance number]
Date of last GPMP (if done): [Date]	

This diabetes care plan was developed by staff at the [insert practice name]. While it specifically relates to the management of your diabetes, your other health problems will also be considered. This care plan uses the skills of many health professionals to help you to have the best of healthcare and for you to manage your diabetes.

This plan focuses on proven therapies that, with support and care, may help prevent complications. Diabetes is best treated early and some difficulties of management can occur when complications arise. The management goals in this plan are set by national diabetes expert bodies. Your diabetes will be monitored against these goals.

This plan encourages you to be actively involved in your care. It is important that you and your healthcare team monitor your diabetes and report anything that is untoward.

We particularly urge you to report any chest pains, unexplained weakness, foot problems, visual changes, or any symptom that concerns you. Report if you feel that you cannot cope or manage, and if you feel distressed or sad by living with your illness.

Emergency contact at [insert practice name] for diabetes – [name] [contact number]

This document should be brought along with you to each visit to the dietitian, diabetes educator, practice nurse, other health professional and to the doctor when your review is due.

Management plan outcomes

Patient needs	• To become educated regarding diabetes • To understand the role of diabetes 'goals' – glucose management, minimising heart risks and individually appropriate preventive activities • To appropriately manage medication for diabetes and other supportive therapies • [Insert individual patient needs]
Management goals	• To lead a happy, healthy lifestyle • To progress toward/achieve recognised goals for diabetes care • To prevent onset or progression of cadiovasular disease or its complications • To remain free of serious side effects from medication • To minimise the burden of diabetes management and care • To overcome barriers to self management including psychological and social factors
Treatment services	• To participate in structured care system at the [insert practice name] • To involve other health service providers • To provide holistic, person-centred care • [Insert individualised patient treatment services] to assist in provision of services
Patient actions	• To undertake appropriate lifestyle measures if needed (eg quit smoking, regular exercise, dietary changes) • To participate in this management plan and be aware of the impact of illness • To become educated regarding diabetes • [Insert individualised patient actions]
Monitoring and review	• The first review will usually be at one to four weeks – to monitor impact of any initial or ongoing therapy medication and other strategies • Every three to six months a major review of the management plan goals will occur • Thereafter, reviews will depend on response to therapy and complexity of all health issues • A recall will be instituted at least every three months to monitor progress
Review date	• [Insert review date]

Past medical history

Family history
[Clinical details of family history]
Medications
[Clinical details – medication list]
Allergies
[Allergy details]
Social history
Alcohol intake
Cigarette smoking history

Patient name: [Full name]

GPMP (MBS Item 721, Diabetes)			
Patient problems/ needs/relevant conditions	Goals – changes to be achieved	Required treatments and services including patient actions	Arrangements for treatments/services (when, who, contact details)
1. General			
Patient's understanding of diabetes and self-management	Patient to have a clear understanding of diabetes and their role in managing the condition	Patient education	GP/nurse/diabetes educator
	Patient to understand the psychological and social impact of living with type 2 diabetes	Patient education	GP/nurse/ diabetes educator/ psychologist
	Patient to understand the role of self-monitoring of glucose (SMBG) if this is required	Patient education	GP/nurse/diabetes educator

GPMP (MBS Item 721, Diabetes)

Patient problems/ needs/relevant conditions	Goals – changes to be achieved	Required treatments and services including patient actions	Arrangements for treatments/services (when, who, contact details)
2. Lifestyle			
Nutrition	Eating according to *Australian dietary guidelines* (www.nhmrc.gov.au/guidelines-publications/n55) with attention to quantity and type of food If concerns regarding cardiovascular disease risk, advise dietary review	Patient education OR As per Lifescripts action plan	GP to monitor Accredited Practising Dietitian (APD)
Weight/body mass index (BMI)	Your target: [insert patient target] kg/m^2 Therapeutic goal is 5–10% loss for people who are overweight and obese with type 2 diabetes With BMI >35 kg/m^2 and comorbidities or BMI >40 kg/m^2, greater weight loss measures should be considered	Monitor Review six monthly OR As per Lifescripts action plan	Patient to monitor GP/nurse to review APD when appropriate BMI >35 kg/m^2 – consider specialist referral and bariatric options
Physical activity	Your target: [insert patient target] minutes/week At least 30 minutes of moderate physical activity on most if not all days of the week (total ≥150 minutes/week)	Patient exercise routine OR As per Lifescripts action plan	GP/Accredited Exercise Physiologist (AEP) Patient to implement

Smoking	Complete cessation	Smoking cessation strategy:
		• Consider:
		  – quit
		  – medication
		OR
		• As per Lifescripts action plan
			Patient to manage
			GP to monitor

GPMP (MBS Item 721, Diabetes)

Patient problems/ needs/relevant conditions	Goals – changes to be achieved	Required treatments and services including patient actions	Arrangements for treatments/services (when, who, contact details)
Alcohol intake	Your target: <[insert patient target] standard drinks/day Healthy: ≤2 standard drinks/day (adults)	Reduce alcohol intake Patient education OR As per Lifescripts action plan	Patient to manage GP to monitor
3. Biomedical			
Cardiovascular disease risk calculation			
Cholesterol/lipids Blood pressure (BP)	Cholesterol level to accepted national target BP to accepted national target	Initiation of pharmacotherapy is dependent on the assessment of absolute cardiovascular disease risk (Australian absolute CVD risk calculator) This requires using multiple risk factors, which is considered to be more accurate than the use of individual parameters Once therapy is initiated, the specified targets apply; however, these targets are somewhat arbitrary and should be used as a guide to treatment, and not as a mandatory requirement Check every six months	GP GP/nurse

GPMP (MBS Item 721, Diabetes)

Patient problems/ needs/relevant conditions	Goals – changes to be achieved	Required treatments and services including patient actions	Arrangements for treatments/services (when, who, contact details)
Glycated haemoglobin (HbA1c)	Your target: <[insert patient target] mmol/mol Healthy: ≤53 mmol/mol (range 48–58 mmol/mol) ≤7% (range 6.5–7.5%)	Needs individualisation according to patient circumstances Check every there to six months or as advised by your GP	GP/nurse
Blood glucose level	Advise 6–8 mmol/L fasting and 8–10 mmol/L postprandial Ongoing self-monitoring of blood glucose is recommended for people with diabetes using insulin, people using sulphonylureas or other medicines that may cause hypoglycaemia, hyperglycaemia arising from illness, with haemoglobinopathies, pregnancy or other conditions where data on glycaemic patterns is required Routine self-monitoring of blood glucose in low-risk patients who are using oral glucose-lowering drugs (with the exception of sulphonylureas) is not recommended	Monitoring is dependent on individual circumstances	Patient/GP/nurse Diabetes educator when required
4. Medication			
Medication review	Targeted and careful use of medications to maximise benefit and minimise side effects	Patient education Review medications	GP/Credentialled Diabetes Educator (CDE) to review and provide education Pharmacist when required/home medicines review
Vaccinations	Influenza Pneumococcal and diphtheria-tetanus-acellular pertussis (dTpa) vaccine	Annually At appropriate intervals	GP/nurse
5. Complications of diabetes			

GPMP (MBS Item 721, Diabetes)

Patient problems/needs/relevant conditions	Goals – changes to be achieved	Required treatments and services including patient actions	Arrangements for treatments/services (when, who, contact details)
Eye complications	Early detection of any problems	Eye check every two years Retinal photography or referral by GP	GP/optometrist/ophthalmologist
Foot complications	Optimal foot care and avoidance of ulceration and amputation by: • patient education on foot care and self-check • professional check feet every six months • early detection and management of complications	Stratify the risk of developing foot complications: • low/intermediate/high risk • the intensity of monitoring and review increases according to level of risk	GP/podiatrist/nurse Patient GP referral to specialist foot clinic if high risks detected
Kidney damage	Avoid kidney complications urine albumin-to-creatinine ratio (UACR): <3.5 mg/mmol women <2.5 mg/mmol men	Test for microalbuminuria annually	GP
Sexual dysfunction	Maintain sexual function	To be discussed with patient where applicable	GP
6. Psychosocial/psychological			
Mood and distress from diabetes	Minimise distress and depression Minimise social isolation, support positive advocacy against social stigma		GP/nurse Psychologist when required
Licence assessment	Maintain safe driving to road authority standards		GP/nurse/endocrinologist/specialist
7. Register with National Diabetes Services Scheme (NDSS)			
	Provide access to best practice consumer resources to support self-management	Provision of self-management information and consumer support and advocacy	GP/nurse/ CDE Diabetes Australia

Patient monitoring

Measurements	Target		Progress				
Glycated haemoglobin (HbA1c): This is a measure of how well your blood glucose has been controlled over the last three months	48–58 mmol/mol or 6.5–7.5% Individualised, as low as reasonably possible without side effects						
Cardiovascular disease (CVD) risk assessment: This is your risk of having a heart attack or stroke in the next five years							
Systolic blood pressure (SBP): The highest reading in blood perssure (BP), is more closely related to poor outcomes.	<130–140	Initiation of drug therapy depends on the assessment of absolute CVD risk Targets should be used as a guide to treatment, and not as a mandatory requirement					
Low-density lipoprotein-cholesterol (LDL-C): This is the 'bad' cholesterol implicated in causing CVD	<2.0						
High-density lipoprotein-cholesterol (HDL-C): This is the 'good' cholesterol associated with protection against CVD	≥1.0						
Triglycerides	<2.0						
Renal function: Estimated glomerular filtration rate (eGFR) is an indicator of overall kidney function Microalbuminuria Microalbuminuria is a sign of kidney stress. Identification at an early stage can prevent kidney problems and/or progression to kidney failure	eGFR Reduce albuminuria by decreasing BP and blood glucose levels						
Foot examination: To identify potential and active foot problems (eg presence of ulcers, infection, corns, calluses, fissures)	Foot risk = low/intermediate/high Today's examination						

Vision:	Visual care					
This is to aid detection of early cataract formation	Full eye review every two years					
Ophthalmology review – to detect small vessel changes in your eyes						

Copy of GPMP offered to patient? [_____]

Copy/relevant parts of the GPMP supplied to other providers? [_____]

GPMP added to the patient's records? [_____]

Date service completed: [_____]

Proposed review date: [_____]

I have explained the steps and costs involved, and the patient has agreed to proceed with the service. [Steps and costs explained, patient agreed]

GP signature: _____   Date: _____

Appendix C. Problem areas in diabetes questionnaire

The Problem areas in diabetes (PAID) questionnaire is a psychometrically sound tool for detecting diabetes-related distress. The PAID questionnaire includes 20 items, each of which focuses on a different commonly experienced problem with diabetes.

Patients indicate how much each issue affects them personally, on a scale of 0 (not a problem) to 4 (serious problem). Individual items scored ≥3 (indicating a somewhat serious or serious problem area) should be discussed with the patient.

Item scores can also be added and standardised to a score out of 100 (by multiplying the total by 1.25). Higher scores indicate higher levels of diabetes-related distress.

Scores ≥40 indicate severe diabetes-related distress and warrant further exploration and discussion with the patient.

Please read each question carefully. Put an X in the box that best describes you for each question					
Which of the following diabetes issues are currently a problem for you?	Not a problem	Minor problem	Moderate problem	Somewhat serious problem	Serious problem
1. Not having clear and concrete goals for your diabetes care	0	1	2	3	4
2. Feeling discouraged with your diabetes treatment plan	0	1	2	3	4
3. Feeling scared when you think about living with diabetes	0	1	2	3	4
4. Uncomfortable social situations related to your diabetes care (eg people telling you what to eat)	0	1	2	3	4
5. Feelings of deprivation regarding food and meals	0	1	2	3	4
6. Feeling depressed when you think about living with diabetes	0	1	2	3	4

Please read each question carefully. Put an X in the box that best describes you for each question					
Which of the following diabetes issues are currently a problem for you?	Not a problem	Minor problem	Moderate problem	Somewhat serious problem	Serious problem
7. Not knowing if your mood or feelings are related to your diabetes	0	1	2	3	4
8. Feeling overwhelmed by your diabetes	0	1	2	3	4
9. Worrying about low blood sugar reactions	0	1	2	3	4
10. Feeling angry when you think about living with diabetes	0	1	2	3	4
11. Feeling constantly concerned about food and eating	0	1	2	3	4
12. Worrying about the future and the possibility of serious diabetes complications	0	1	2	3	4
13. Feelings of guilt or anxiety when you get off track with your diabetes management	0	1	2	3	4
14. Not 'accepting' your diabetes	0	1	2	3	4
15. Feeling unsatisfied with your diabetes physician	0	1	2	3	4
16. Feeling that diabetes is taking up too much of your mental and physical energy every day	0	1	2	3	4
17. Feeling alone with your diabetes	0	1	2	3	4

Please read each question carefully. Put an X in the box that best describes you for each question					
Which of the following diabetes issues are currently a problem for you?	**Not a problem**	**Minor problem**	**Moderate problem**	**Somewhat serious problem**	**Serious problem**
18. Feeling that your friends and family are not supportive of your diabetes management efforts	0	1	2	3	4
19. Coping with complications of diabetes	0	1	2	3	4
20. Feeling 'burned out' by the constant effort needed to manage diabetes	0	1	2	3	4

Investigation of specific concerns highlighted by the PAID questionnaire is useful for formulating and adjusting treatment options for your patients. Severe and persistent diabetes-related distress may warrant referral to a mental health specialist

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Appendix D. Patient health questionnaire-2 tool

The Patient health questionnaire-2 (PHQ-2) is a psychometrically sound tool for detecting depression and anhedonia. The PHQ-2 is composed of the first two items from the Patient health questionnaire-9 (PHQ-9; each describing a different problem/symptom of depression), making it ideal for use in busy clinical settings.

Patients indicate how frequently they have been bothered by each problem (item) over the past two weeks. The items are scored on a four-point Likert scale from 0 (not at all) to 3 (nearly every day). Individual item scores are added together, resulting in a total score from 0 to 6. Total scores ≥3 warrant further assessment for depression using a diagnostic instrument or interview. Patients who are subsequently diagnosed with depression should be provided with ongoing healthcare professional support for the management and treatment of their depression and their ongoing diabetes care.

Note that as this tool has only two items, it may seem unnecessary to administer this tool to patients using paper and pen. However, an advantage of doing so is that it allows the patients to 'grade' their symptoms and allows the healthcare professional to track their patient's scores over time.

Over the past two weeks, how often have you been bothered by any of the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed or hopeless	0	1	2	3

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Appendix E. Available glucose-lowering agents

When evaluating the clinical evidence of the following interventions, high-quality, long-term prospective trials on clinical outcomes specific to type 2 diabetes and its complications are useful. Of note, agents recently listed for glycaemic management have short-term trials that have reported cardiovascular safety but no cardiovascular benefits.

Metformin

Prospective trials have demonstrated reduced progression (31%) from impaired fasting glucose (IFG) or impaired glucose tolerance (IGT) to diabetes when metformin is used. However, metformin is not currently Therapeutic Goods Administration (TGA) indicated for this use.

In patients with type 2 diabetes, the UK Prospective Diabetes Study (UKPDS) has demonstrated cardiovascular benefits with metformin use in overweight patients.

Metformin:

- is the medication of first choice for people with type 2 diabetes
- reduces hepatic glucose output and improves muscle cell insulin receptor resistance
- does not stimulate insulin release
- significantly reduces the risk of diabetes-related morbidity and mortality in overweight patients
- should be used with caution in people with hepatic or cardiac disease, and those with a heavy alcohol intake or dehydration (eg acute gastroenteritis) and renal impairment due to risks of lactic acidosis.

Contraindication:

- Advanced renal impairment (estimated glomerular filtration rate [eGFR] <30 mL/min/1.73 m²) is the only absolute contraindication to metformin. It should be used with caution in people with an eGFR of 30–45 mL/min/1.73 m² with dose reduction is recommended.

Main side effects:[278]

- anorexia, nausea, vomiting
- diarrhoea, abdominal cramps, flatulence
- lactic acidosis (uncommon, but may occur with dehydration and co-existing renal, liver or cardiovascular disease [CVD])
- possible vitamin B12 deficiency.

Sulphonylureas

The UKPDS has demonstrated microvascular benefits with sulphonylurea use. Cardiovascular benefits only emerged with long-term, post-trial follow-up of newly diagnosed patients previously intensively treated with sulphonylureas and insulin. This has been called the 'legacy' effect. Clear microvascular benefits have also been shown in the Advance in Diabetes and Vascular Disease (ADVANCE) trial, which used the more contemporary sulphonylurea agent gliclazide.

Sulphonylureas:

- act to increase insulin secretion in a non-glucose dependent fashion and rely on some residual β-cell function
- can be considered after a trial of healthy lifestyle and used in combination with agents such as metformin.

Main side effects:

- weight gain
- symptomatic hypoglycaemia
- anorexia, nausea, diarrhoea, skin rashes
- occasionally blood dyscrasias
- glibenclamide and glimepiride may cause high rates of hypoglycaemia (in older patients and in patients with autonomic neuropathy or nephropathy).

Acarbose

One prospective trial Study to Prevent Non-Insulin-Dependant Diabetes Mellitus (STOP-NIDDM) has shown reduced progression to diabetes in patients with IGT, as well as macrovascular benefits.[279] As yet, no prospective cardiovascular trials have reported on acarbose use in type 2 diabetes.

Acarbose:

- can be used when blood glucose values remain high after meals despite dietary modification
- inhibits the digestion of carbohydrate and thus slows the rate of glucose delivery into the circulation
- needs to be taken at the time of starting the meal and introduced gradually to avoid flatulence and abdominal discomfort.

If hypoglycaemia occurs (because of concurrent sulphonylurea or insulin treatment), glucose rather than other carbohydrates is required. Care is necessary in those with renal impairment or gastrointestinal disease, and liver enzymes need to be monitored.

Main side effects:

- flatulence and abdominal bloating
- nonresponse to carbohydrates other than glucose if hypoglycaemic
- liver abnormalities (rare).

Glitazones (pioglitazone and rosiglitazone)

The Prospective pioglitazone clinical trial in macrovascular events (PROactive) trial did not demonstrate benefit for the primary outcome of major adverse cardiovascular events (MACE) but did report significantly increased risk of heart failure (11% versus 8% in placebo).[116]

Several meta-analyses have reported that glitazones (including rosiglitazone) increase risk of hospitalisation with heart failure or heart failure death.

Glitazones:

- sensitise the liver and peripheral tissues to insulin and are effective in lowering blood glucose by reducing insulin resistance
- can (pioglitazone and rosiglitazone) be used as combination therapy with metformin, sulphonylureas or insulin.

Contraindications:[280,281]

- moderate to severe cardiac failure (pioglitazone and rosiglitazone)
- increased risk of bladder cancer.

Rosiglitazone is not recommended in patients with known ischaemic heart disease, particularly in those taking nitrates.

Rosiglitazone is not listed on the Pharmaceutical Benefits Scheme (PBS) for triple therapy with metformin and a sulphonylurea, or in combination with insulin.

Main side effects:

- increased subcutaneous fat and/or fluid
- decreased haemoglobin levels
- increased risk of peripheral fractures in women
- possible increased risk of myocardial infarction (rosiglitazone)
- increased LDL-C (rosiglitazone).

Incretins

Two classes of incretin medications exist – dipeptidyl peptidase-4 inhibitor (DPP-4i) and glucagon-like peptide-1 receptor agonist (GLP-1 RA).[282]

DPP-4i

DPP-4i includes sitagliptin, linagliptin, saxagliptin, vildagliptin and alogliptin.

Saxagliptin (Saxagliptin Assessment of Vascular Outcomes Recorded in Patients with Diabetes Mellitus Thrombolysis in Myocardial Infarction [SAVOR-TIMI] trial) showed secondary endpoint data: statistically significant increase in hospital admissions for congestive heart failure. No demonstrated macrovascular benefits.

Alogliptin (Examination of Cardiovascular Outcomes with Alogliptin versus Standard of Care [EXAMINE] trial) and sitagliptin (Trial to Evaluate Cardiovascular Outcomes after Treatment with Sitagliptin [TECOS] trial) showed no increased cardiovascular risks, but also did not demonstrate macrovascular benefits.

Other DPP-4i have no reported prospective cardiovascular trials demonstrating benefits.

DPP-4i:

- are oral agents and act by increasing levels of circulating incretins – glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) – which are released by intestinal cells in response to food
- block the enzyme dipeptidyl peptidase-4 (DPP-4), which is responsible for rapid breakdown of GLP-1 and GIP
- cause elevated and prolonged action of physiologically released incretin hormones
- such as GLP-1 and GIP act on pancreatic cells to increase insulin levels and also suppress α-cell secretion of glucagon (elevated in type 2 diabetes)
- are weight neutral and improve postprandial control
- rarely cause hypoglycaemia except in combination with agents such as sulphonylureas and insulin
- dose reduction in renal impairment eGFR <60 mL/min/1.73 m^2 for alogliptin, saxagliptin, sitaglitpin, and vildagliptin; no dose adjustment required for hepatically metabolised linagliptin; saxagplitin may not be used in end-stage renal failure stage five of chronic kidney disease.[283]

Main side effects:

- nasopharyngitis
- headache
- upper respiratory tract symptoms.

GLP-1 RA

GLP-1 RA includes exenatide, liraglutide and lixisenitide.

Lixisenatide (Evaluation of Lixisenatide in Acute Coronary Syndrome [ELIXA] trial) showed no increased cardiovascular risks but also did not demonstrate macrovascular benefits. A prospective cardiovascular safety outcomes trial (Liraglutide Effect and Action in Diabetes: Evaluation of Cardiovascular Outcome Results [LEADER]) for liraglutide, another GLP-1 RA in high risk patients, had a 13% reduction in major adverse cardiac events and 22% reduction in cardiovascular death.[130]

GLP-1 RAs:

- are injectable medications that bind to the GLP-1 receptor (exenatide is currently PBS subsidised; once weekly exenatide, liraglutide and lixisenitide are TGA approved for use in Australia but are currently not PBS-listed)
- cause weight loss through actions on cerebral hormonal responses to insulin and appetite
- may affect gastric emptying
- only cause hypoglycaemia in combination with other medications such as sulphonylureas and insulin.

Main side effects:

- nausea, vomiting
- pancreatitis (rarely)
- weight loss.

Sodium glucose co-transporter 2 inhibitors

The EMPA-REG (Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes trial) OUTCOME trial (empagliflozin) showed reduced rates of death from cardiovascular causes (38% relative risk reduction) and any cause (32% relative reduction). In secondary endpoint analyses, a 35% reduction in hospitalisation for heart failure was observed. The mechanism through which empagliflozin may mediate these outcomes is still under investigation.

The other sodium glucose co-transporter 2 (SGLT2) inhibitors have no reported prospective cardiovascular trials.

SGLT2 inhibitors:

- are novel oral medications that selectively inhibit SGLT2, the main renal glucose reabsorptive mechanism
- result in glycosuria with resultant lowering of glucose in a non-insulin dependent manner and modest weight loss plus lowered BP
- rely on adequate renal function
- promote weight loss.

Side effects:

- weight loss
- increased urogenital mycotic and urinary tract infections
- aggravate dehydration
- euglycaemic diabetic ketoacidosis (DKA).

Appendix F. Table of evidence and properties of glucose-lowering agents

Refer to Figure 4 for more information.

Glucose-lowering class and drugs	Mechanism of action	Outcome data	Contraindications	
Biguanide • metformin • metformin XR	Reduces hepatic glucose output, lowers fasting glucose levels	UKPDS[1]	Renal impairment (estimated glomerular filtration rate [eGFR] <30 ml/min/1.73 m^2) Severe hepatic impairment	
Sulphonylureas • glibenclamide • gliclazide • gliclazide modified release (MR) • glimepiride • glipizide	Triggers insulin release in a glucose-independent manner	UKPDS[2,3]	Severe renal or hepatic impairment	
Dipeptidyl peptidase-4 inhibitors (DPP-4i) • alogliptin • linagliptin • saxagliptin • sitagliptin • vildagliptin	Decreases inactivation of glucagon-like peptide 1 (GLP-1) thereby increasing its availability GLP-1 stimulates beta cell insulin release and slows gastric emptying	EXAMINE[4,5] – Alogliptin SAVOR-TIMI 53[6,7] – Saxagliptin TECOS[8] – Sitaglipitn	Pancreatitis[9]	
Thiazolidinediones (TZD) • pioglitazone • rosiglitazon	Transcription factor peroxisome proliferator-activated receptor PPARγ agonists Lowers glucose levels through insulin sensitisation	PROACTIVE[11] – Pioglitazone		

	Precautions, side effects and administration	Cost and accessibility
	Precautions Suspend treatment during acute disease/conditions with the potential to cause tissue hypoxia or alter renal function. **Side effects** Gastrointestinal side effects, lactic acidosis, weight neutral **Administration** Oral administration Start at low dose and up-titrate Slow release preparations available	General schedule on Pharmaceutical Benefits Scheme (PBS)
	Precautions Hypoglycaemia **Side effects** Weight gain **Administration** Oral administration Start at low dose and up-titrate Slow release preparation available	General schedule on PBS
	Precautions Nasopharyngitis – often subsides in 10–14 days **Side effects** Rash, pancreatitis, gastrointestinal disturbances, weight neutral **Administration** Oral administration Dosage adjustment in renal impairment (except Linagliptin)[10]	Alogliptin, linagliptin, saxagliptin, sitagliptin, vildagliptin: • PBS-subsidised for use in combination with metformin and sulphonylureas or both* Sitagliptin: • Linagliptin and sitagliptin have been recommended by Pharmaceutical Benefits Advisory Committee (PBAC) for listing on PBS (date is not yet available)
	Precautions Symptomatic heart failure **Side effects** Fluid retention, heart failure, increased risk of non-axial fractures in women, increased risk of bladder cancer, weight gain **Administration** Oral administration	PBS-subsidised for use in combination with metformin, sulphonylurea or both Patient must have a contraindication or intolerance to metformin-sulphonylurea combination PBS-subsidised for use with insulin

Table of evidence and properties of glucose-lowering agents – Continued

Glucose-lowering class and drugs	Mechanism of action	Outcome data	Contraindications	
Alpha 1 glucosidase inhibitors • acarbose	Slows intestinal carbohydrate absorption and reduces postprandial glucose levels		Severe renal impairment (creatinine clearance <25 mL/min/m^2)	
Sodium-glucose co-transporter-2 (SGLT2) inhibitors • canagliflozin • dapagliflozin • empagliflozin	Inhibits a sodium-glucose co-transporter to produce urinary glucose loss and decrease glucose levels	EMPA-REG OUTCOME[12] – Empagliflozin	Diminished efficacy with renal impairment (eGFR <60 ml/min/1.73 m^2)	
Glucagon-like peptide-1 receptor agonist (GLP-1 RA) • exenatide • exenatide ER • liraglutide • lixisenatide	Stimulates beta-cell insulin release and slows gastric emptying	ELIXA[13,14] – Lixisenatide LEADER[15] – Liraglutide	Avoid with history of pancreatitis or pancreatic malignancy	
Insulin	Directly activates the insulin receptor	UKPDS[2]		

*Saxagliptin and sitagliptin are currently PBS listed for triple oral therapy, linagliptin and vildagliptin have been recommended by PBAC for triple oral therapy (date is not yet available)

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Precautions, side effects and administration	Cost and accessibility
Precautions GI disorders associated with malabsorption **Side effects** Bloating and flatulence, weight neutral **Administration** Oral administration Take with meals as tolerated	General schedule on PBS
Precautions Avoid use with loop diuretics **Side effects** Dehydration, dizziness, genitourinary infections (advise adequate fluid intake and meticulous toileting hygiene), ketoacidosis, weight loss **Administration** Oral administration	Dapagliflozin and empagliflozin: • PBS-subsidised for use in combination with metformin, sulphonylurea or both • PBS-subsidised for use with insulin Not PBS-subsidised for use as monotherapy or in combination with a thiazolidinedione (glitazone), a dipeptidyl peptidase 4 inhibitor (gliptin) or a glucagon-like peptide-1 Canagliflozin: PBS-subsidisation withdrawn
Precautions Dosage adjustment in moderate-severe renal impairment Increased risk of pancreatitis **Side effects** Nausea, vomiting, weight loss **Administration** Subcutaneous injection	Exenatide and exenatide ER: • PBS-subsidised for use in combination with metformin, sulphonylurea or both • Exenatide • PBS-subsidised for use with insulin Not PBS-subsidised for use as monotherapy or in combination with a dipeptidyl peptidase 4 inhibitor (gliptin), a thiazolidinedione (glitazone) or an SGLT2 inhibitor Liraglutide: not PBS-subsidised
Precautions Consider need for dosage adjustment in moderate-severe renal disease **Side effects** Hypoglycaemia, weight gain **Administration** Subcutaneous injection Considered early if blood glucose level (BGL) is very high	General schedule on PBS Levemir insulin: PBS-subsidisation restricted to type 1 Diabetes

References – Appendix F

1. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). Lancet 1998;352(9131):854–65.

2. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet 1998;352(9131):837–53.

3. The ADVANCE Collaborative Group. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. N Engl J Med 2008;358:2560–72.

4. White WB, Cannon CP, Heller SR, et al. Alogliptin after acute coronary syndrome in patients with type 2 diabetes. N Engl J Med 2013;369:1327–35.

5. Zannad F, Cannon CP, Cushman WC, et al. Heart failure and mortality outcomes in patients with type 2 diabetes taking alogliptin versus placebo in EXAMINE: A multicentre, randomised, double-blind trial. Lancet 2015;385(9982):2067–76.

6. Scirica BM, Bhatt DL, Braunwald E, et al. Saxagliptin and cardiovascular outcomes in patients with type 2 diabetes mellitus. N Engl J Med 2013;369:1317–26.

7. Scirica BM, Braunwald E, Raz I, et al. Heart failure, saxagliptin, and diabetes mellitus: Observations from the SAVOR-TIMI 53 randomized trial. Circulation 2014;130(18):1579–88.

8. Green JB, Bethel MA, Armstrong PW, et al. Effect of sitagliptin on cardiovascular outcomes in type 2 diabetes. N Engl J Med 2015;373(3):232–42.

9. Meier JJ, Nauck MA. Risk of pancreatitis in patients treated with incretin-based therapies. Diabetologia 2014;57(7):1320–24.

10. McGill JB, Sloan L, Newman J, et al. Long-term efficacy and safety of linagliptin in patients with type 2 diabetes and severe renal impairment: A 1-year, randomized, double-blind, placebo-controlled study. Diabetes Care 2013;36(2):237–44.

11. Dormandy JA, Charbonnel B, Eckland DJ, et al. Secondary prevention of macrovascular events in patients with type 2 diabetes in the PROactive Study (PROspective pioglitAzone Clinical Trial In macroVascular Events): A randomised controlled trial. Lancet 2005;366(9493):1279–89.

12. Zinman B, Wanner C, Lachin JM, et al. Empagliflozin, cardiovascular outcomes, and mortality in type 2 diabetes. N Engl J Med 2015;373(22):2117–28.

13. Diaz R, McMurrayJJV, Lewis EF, et al. The evaluation of lixisenatide in acute coronary syndrome – The results of ELIXA symposium. Boston, MA: 75th Scientific Sessions of the American Diabetes Association, 2015.

14. Bentley-Lewis R, Aguilar D, Riddle MC, et al. Rationale, design, and baseline characteristics in evaluation of lixisenatide in acute coronary syndrome, a long-term cardiovascular end point trial of lixisenatide versus placebo. Am Heart J 2015;169(5):631–38.e7.

15. Marso SP, Daniels GH, Brown-Frandsen K, et al. Liraglutide and cardiovascular outcomes in type 2 diabetes. N Engl J Med 2016;375(4):311–22.

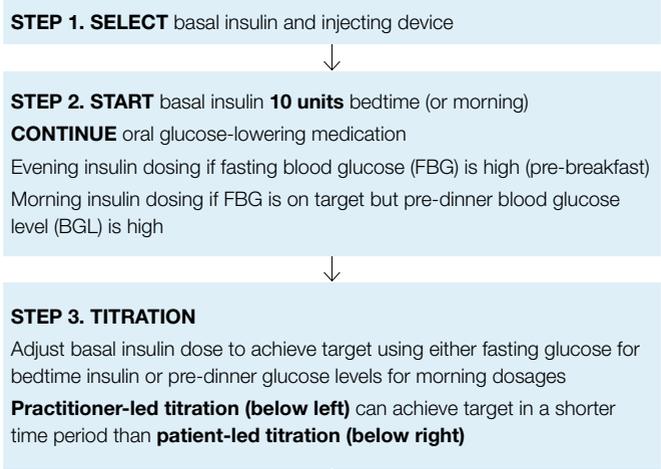# *Appendix G. Types of insulin available*

The pharmacokinetics of the different insulins is patient-dependent. Review product information for each product before prescribing. An empirical approach to dosage together with a 'go slow' policy will result in the smoothest fine tuning of management. Some of these insulins are available as injection devices, pen injectors, disposable insulin pens, cartridges and vials.

Type	Brand name	Manufacturer	Nature
Rapid-acting (peak at one hour, last 3.5–4.5 hours)			
Insulin lispro	Humalog*	Lilly	Analogue
Insulin aspart	NovoRapid*	Novo Nordisk	Analogue
Insulin glulisine	Apidra*	Sanofi	Analogue
Short-acting (peak at two to five hours, last six to eight hours)			
Neutral	Actrapid	Novo Nordisk	Human
	Humulin R	Lilly	Human
	Hypurin Neutral	Aspen	Bovine
Intermediate acting (12–24 hours)			
Isophane	Humulin NPH	Lilly	Human
	Protaphane	Novo Nordisk	Human
	Hypurin Isophane	Aspen	Bovine
Long-acting (BASAL)			
Insulin detemir diabeyes	Levemir	Novo Nordisk	Analogue
Insulin glargine	Lantus	Sanofi	Analogue
Insulin glargine (U300)	Toujeo	Sanofi	Analogue
Premixed insulins			
Lispro 25%/lispro protamine 75%	Humalog Mix25*	Lilly	Analogue
Lispro 50%/lispro protamine 50%	Humalog Mix 50*	Lilly	Analogue
Insulin aspart 30%/insulin aspart protamine 70%	NovoMix 30*	Novo Nordisk	Analogue
Neutral 30%/isophane 70%	Humulin 30/70	Lilly	Human
	Mixtard 30/70	Novo Nordisk	Human
Neutral 50%/isophane 50%	Mixtard 50/50	Novo Nordisk	Human

*Rapid-acting – should be given immediately before eating

Appendix H. Examples for insulin initiation and titration

H.1. Guide to starting and adjusting basal insulin[141,284]

STEP 1. SELECT basal insulin and injecting device

↓

STEP 2. START basal insulin **10 units** bedtime (or morning)

CONTINUE oral glucose-lowering medication

Evening insulin dosing if fasting blood glucose (FBG) is high (pre-breakfast)

Morning insulin dosing if FBG is on target but pre-dinner blood glucose level (BGL) is high

↓

STEP 3. TITRATION

Adjust basal insulin dose to achieve target using either fasting glucose for bedtime insulin or pre-dinner glucose levels for morning dosages

Practitioner-led titration (below left) can achieve target in a shorter time period than **patient-led titration (below right)**

Practitioner-led titration   OR   Patient-led titration

Mean FBG over previous two days (mmol/L)*	Adjust insulin dose twice weekly until FBG target is achieved
10	↑ by 4 units
8–9.9	↑ by 2–4 units
7–7.9	No change or ↑ by 2 units
6–6.9	No change
4–5.9	↓ by 2 units
<4, or if severe hypoglycaemic episode	↓ by 2–4 units

↑ by 2 units every three days, until FBG target is achieved

A. If FBG ≥6 mmol/L but ≤8 mmol/L for three consecutive days, no change

B. If FBG is 4–6 mmol/L on any day, ↓ insulin dose by 2 units

C. If FBG <4 mmol/L on any day, ↓ insulin dose by 4 units

*Do not increase the insulin dose if FBG is <4 mmol/L at any time in the preceding week

H.2. Guide to starting and adjusting premixed insulin

STEP 1. SELECT premixed insulin and injecting device

↓

INSULIN-NAÏVE patients

STEP 2. START premixed insulin **10 units** immediately before or soon after the largest meal (usually evening meal)

CONTINUE metformin if indicated, consider tapering sulphonylureas as glycaemic control improves

↓

STEP 3. TITRATION

Adjust the evening premixed insulin dose once or twice a week according to the schedule below to a fasting blood glucose (FBG)[285]

Lowest blood glucose level (BGL) reading (mmol/L) of the previous three days – fasting or preprandial	Insulin dosage adjustment
>10	↑ by 4 units
8–10	↑ by 2 units
7–7.9	No change or ↑ by 2 units
6–6.9	No change
4–5.9	↓ by 2 units
<4.0 or severe hypoglycaemic event*	↓ by 4 units

If a morning insulin dose is given, adjust the insulin dose according to evening preprandial BGL according to the same titration recommendations

*Hypoglycaemia should prompt a review of other oral therapy. Which insulin is adjusted depends on regimen and target glucose

↓

STEP 4. INTENSIFICATION: Once daily insulin to twice daily premixed insulin

When?

- If the FBG is at target, if evening preprandial BGL > FBG or if evening preprandial BGL is high, or
- after three months if glycated haemoglobin (HbA1c) > target despite FBG and evening preprandial BGL at target

How?

1. Halve the current once daily insulin dose and give the total dose as a twice daily injection (pre-breakfast and pre-dinner)
2. Monitor pre-dinner BGL and FBG versus targets
3. Once a week, adjust both insulin doses independently (according to protocol above in step 3); pre-breakfast insulin is adjusted according to pre-dinner BGL and pre-dinner insulin is adjusted according to FBG

H.3. Guide to basal plus insulin intensification schedules

STEP 1. SELECT rapid-acting (prandial) insulin and injecting device to be added in addition to basal insulin

↓

STEP 2. START rapid-acting insulin (4 units) to be given before the meal with the largest carbohydrate content

CONTINUE basal insulin at the current dose

CONTINUE metformin, consider tapering sulphonylureas as glycaemic control improves

MONITOR two-hour postprandial blood glucose level (BGL)

↓

STEP 3. TITRATION

Increase rapid acting (prandial) insulin dose by 2 units every three days to achieve target

Two-hour postprandial BGL (mmol/L)	Rapid-acting (prandial) insulin dosage adjustment
≥8 (for three consecutive days)	No change or ↑ by 2 units
6.0–7.9	No change
4.0–5.9	↓ by 2 units
<4.0 on any day	↓ by 2–4 units

↓

4. Basal plus titration to basal bolus - intensification

When?

If HbA1c is not at target after three months, add a further prandial insulin dose to another meal (eg basal plus 2 to basal bolus)

How?

1. Keep the current prandial and basal insulin doses unchanged
2. Add a new rapid-acting (prandial) insulin to the next largest meal of the day (starting at 10% of the basal insulin dose or 4 units)
3. ↑ new prandial insulin dose by 2 units every three days until postprandial target is achieved as per step 3 above

Appendix I. Tools for assessing neuropathy, circulation and foot deformity[160]

Neuropathy
10 g monofilament sensitivity
Vibration perception (tuning fork or biothesiometer)
Neuropathy disability score – ankle (Achilles) reflexes and the sensory modalities of pinprick, vibration and temperature perception

Circulation
Palpation of peripheral pulses
Ankle-brachial pressure index
Toe-brachial pressure index

Foot deformity score
Six point scale (one point for each characteristic)
☐ small muscle wasting
☐ Charcot foot deformity
☐ bony prominence
☐ prominent metatarsal heads
☐ hammer or claw toes
☐ limited joint mobility
A score of ≥3 indicates foot deformity

Appendix J. Detailed information on glycaemic emergencies

J.1 Hypoglycaemia

Hypoglycaemia is a common complication of the management of type 1 diabetes. But the frequency of hypoglycaemia in type 2 diabetes is underestimated. Its clinical significance, especially in the elderly patient, is great. Hypoglycaemia can lead to falls, fractures, injuries, arrhythmias and, in severe cases, death. Symptoms may go unrecognised or may be mistaken for other conditions (eg transient ischaemic attack [TIA], vasovagal episodes).

Patients at risk of hypoglycaemia (apart from the elderly) include people who have:

- longstanding type 2 diabetes with cardiovascular disease (CVD)
- renal impairment and chronic kidney disease (CKD)
- monotherapy or combination therapy with insulin or long-acting sulphonylureas
- excessive alcohol intake
- beta-blocker therapy (rare), in particular vasodilatory agents (eg propranolol, atenolol)
- participated in unaccustomed or vigorous exercise.

The risk of hypoglycaemia with each sulphonylurea relates to the drugs' pharmacokinetic properties. Long-acting preparations are associated with higher risks of hypoglycaemia (eg glibenclamide [Daonil, Glimel]). Studies have shown significantly lower rates of hypoglycaemia associated with the use of gliclazide (Diamicron) compared with other sulphonylureas.

Although many newer therapies for type 2 diabetes do not cause hypoglycaemia when used as monotherapy, their use in combination with insulin or sulphonylureas increases the risk of hypoglycaemia. The use of insulin analogues may limit, but not eradicate, the risk of hypoglycaemia.

Symptoms of hypoglycaemia vary between persons. Patients often learn to recognise their unique symptoms. The onset of symptoms usually occurs with a blood glucose level (BGL) of <4.0 mmol/L. Common symptoms fall into two categories:

- Adrenergic symptoms – trembling or shaking, sweating, hunger, lightheadedness and numbness around the lips and fingers
- Neuroglycopaenic symptoms – lack of concentration, weakness, behavioural change, tearfulness/crying, irritability, headache and dizziness.

Severe hypoglycaemia occurs clinically when a patient develops a reduced conscious state and requires assistance from another person to manage an episode of hypoglycaemia. A BGL of <3.0 mmol/L puts the person at increased risk of severe hypogylcaemia.

Asymptomatic hypoglycaemia (or biochemical hypoglycaemia) occurs when a BGL is low (<3.9 mmol/L).

Impaired hypoglycaemia awareness is of particular concern and refers to the clinical situation where a patient loses the ability to detect the early symptoms of hypoglycaemia. This results from repeated episodes of mild hypoglycaemia or can arise with long duration of diabetes due to loss of adrenergic and glucagon response, with eventual loss of adrenergic and neuroglycopaenic symptoms. It can lead to confusion and marked behavioural change, which is not recognised by the patient and may progress to loss of consciousness.

The cause needs to be identified and the episode dealt with by reinforcing education, counselling the patient and perhaps changing treatment.

Management of an episode of hypoglycaemia

If a patient with diabetes is showing signs of potential hypoglycaemia, first make sure that the patient is safe (eg seated securely and not at risk of falling).

If possible, confirm that the symptoms are due to hypoglycaemia by performing a finger prick BGL.

If the person is awake, alert and can swallow, hypoglycaemia may be managed according to the **'Rule of 15'**.

If the patient is symptomatic, or if BGL is <4.0 mmol/L:

- provide **15 g** of quick-acting carbohydrate that is easy to consume (eg half a can of regular – non-diet – soft drink, half a glass of fruit juice, three teaspoons of sugar or honey, six or seven jellybeans, three glucose tablets)

- wait **15 minutes** and repeat blood glucose check – if the level is not rising, suggest eating another quick-acting carbohydrate from the above list
- provide some longer acting carbohydrate if the patient's next meal is more than **15 minutes** away (eg a sandwich; one glass of milk or soy milk; one piece of fruit; two or three pieces of dried apricots, figs or other dried fruit; one tub of natural low-fat yoghurt; six small dry biscuits and cheese)
- test glucose every one to two hours for the next four hours.

Patients and carers should be made aware of the use of an alternative 'Rule of 15'.

If the patient is symptomatic, but the blood glucose or capillary glucose cannot be performed to confirm that the episode is due to hypoglycaemia, treat the patient as if they have hypoglycaemia by administering 15 g of quick-acting carbohydrate. If there is no improvement after 15 minutes, the patient could have another cause for the episode and further medical assistance may be necessary.

If the patient cannot safely swallow 15 g of carbohydrate due to their reduced conscious state, consider the administration of 1 mg of glucagon intramuscularly or subcutaneously into the thigh, buttock or upper arm, if available. If not, further emergency medical assistance will be required.

If glucagon is administered, always review the monitored capillary glucose after 15 minutes to ensure effective management of the hypoglycaemia has occurred and the blood glucose remains ≥4 mmol/L. Test again one hour after severe hypoglycaemia to ensure stable glucose levels.

Post-hypoglycaemia – Re-assess the patient's circumstances, medication dosages, and dietary intake, as well as overall need for glucose monitoring after any severe hypoglycaemic episode with the patient and/or with their immediate family or support persons. Also ensure implications for driving competence (refer to Section 14.3 Driving and www.austroads.com.au/drivers-vehicles/assessing-fitness-to-drive), operation of machinery and other similar areas are discussed with the patient.

J.2 Hyperglycaemic emergencies

Severe hyperglycaemia has significant morbidity and mortality.

Hyperglycaemic emergencies should be preventable in people known to have diabetes, and their occurrence in this group signifies a major breakdown in medical management. Adequate early management of sick patients with diabetes will prevent the development of hyperglycaemic emergencies.

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) is a medical emergency requiring specialist care and should generally be managed in hospital. Whatever the setting, it is important that treatment commences as early as possible.

DKA, once thought to typify type 1 diabetes mellitus, can occur in patients with type 2 diabetes mellitus under stress (eg during surgery, trauma, infections, high dose steroids). The very young, older people and pregnant patients are also at greater risk of DKA. A type of ketoacidosis has now been identified (although rarely) with the use of sodium glucose co-transporter 2 (SGLT2) inhibitors alone or in combination with other glucose lowering medications. This type of diabetic ketosis is characterised by the absence of extreme hyperglycaemia and may lead to overlooked diagnoses. Refer to the Therapeutic Goods Administration (TGA) warning at www.tga.gov.au/alert/sodium-glucose-co-transporter-2-inhibitors-used-treat-type-2-diabetes

Pathophysiology

DKA occurs when there is an absolute deficiency of insulin. For DKA to occur in type 2 diabetes, there needs to be significantly impaired insulin secretion as a result of 'glucotoxicity', or a long duration of diabetes together with severe insulin resistance, typically as the result of severe infection or other stresses.

This results in:

- increasing hepatic glucose production causing hyperglycaemia
- increasing peripheral lipolysis releasing free fatty acids – these are converted to ketoacids by the liver resulting in a metabolic acidosis
- hyperglycaemia-induced osmotic diuresis leading to sodium, potassium and phosphate depletion
- dehydration causing pre-renal failure.

Assessment

The biochemical criteria for DKA are:

- hyperglycaemia, defined by a BGL >11 mmol/L*
- venous pH <7.3 or bicarbonate <15 mmol/L
- presence of blood ketones or urinary ketones (abnormal ketones is ≥0.5 mmol/L, severe ketosis is >3.0 mmol/L)

*Note the absence of hyperglycaemia may occur with ketosis with SGLT2 inhibitors

Blood ketone testing is preferred but urinalysis may be used for initial assessment if blood ketone testing is not available. Blood ketone testing equipment should be made available for medical practices and 'at risk' patient use.

When treating adult patients with DKA, the following must be considered and closely monitored:

- correction of fluid loss with intravenous fluids
- correction of hyperglycaemia and suppression of ketone production with insulin
- correction of electrolyte disturbances, particularly potassium loss
- resolution of acid–base balance
- treatment of concurrent infection/conditions (eg stroke, myocardial infarction, deep vein thrombosis), if present.

The main aim in treating DKA is to progressively normalise the blood pH and clear the body of excessive ketones – achieved by aggressive fluid replacement and insulin therapy. This also improves the blood glucose concentration. The hyperglycaemia corrects before the acidosis. Therefore, intravenous glucose is required to allow the insulin infusion to continue to suppress ketone production while acidosis resolves.[176]

Wherever possible the patient should be managed in a specialist medical unit. In rural and remote practice, this may not be possible. In this situation, it is advisable to contact the most appropriate diabetes resource person for advice while commencing treatment promptly.

Hyperosmolar hyperglycaemic state

Hyperosmolar hyperglycaemia usually occurs in type 2 diabetes most often in the elderly, or in those with newly diagnosed type 2 diabetes. It is characterised by severe hyperglycaemia (usually >25 mmol/L) , hyperosmolality, dehydration and a change in mental state with little or no ketoacidosis. It may present as hypovolaemic shock and coma in severe cases.[176] This is usually a result of illness or infection; however, it can also be due to poor patient compliance. Older patients are at higher risk of hyperosmolar hyperglycaemic state (HHS).

Pathophysiology

HHS develops because of relative rather than absolute insulin deficiency. Significant insulin deficiency causes hyperglycaemia due to increased hepatic gluconeogenesis. However, as absolute insulin deficiency is not present, peripheral lipolysis remains suppressed and the release of free fatty acids is low. Little substrate is available for the generation of ketoacids and a metabolic acidosis does not occur.

The hyperglycaemia results in an osmotic diuresis leading to pre-renal failure. Eventually, severe intravascular volume depletion occurs resulting in a further deterioration of renal function. Consequently, glomerular filtration diminishes, preventing the further excretion of glucose. With ongoing increasing hepatic glucose production, decreased peripheral glucose utilisation and reduced urinary glucose losses, severe hyperglycaemia results.

The depletion of the total body water leads to the hyperosmolality of body fluids reflected by the extreme hyperglycaemia and increased plasma sodium. This hyperosmolar state affects consciousness and may cause coma.

General outline for the management of HHS

Wherever possible, the patient with HHS should be managed in a specialist medical unit. It may present as hypovolaemic shock and coma in severe cases.[176] It is important to note that blood glucose meters do not register very high glucose levels, so access to a laboratory is necessary to monitor the correction of hyperglycaemia as well as to monitor sodium and potassium levels. Rapid correction of the hyperosmolar state is dangerous.

In remote rural practice, this may not be possible. In this situation it is advisable to contact the most appropriate diabetes resource person (specialist endocrinologist) for advice while promptly commencing treatment.

References

1. Shaw J, Tanamas S, editors. Diabetes: the silent pandemic and its impact on Australia. Melbourne: Baker IDI Heart and Diabetes Institute, 2012.

2. Johnson B, Abraham M, Conway J, et al. Partnering with patients and families to design a patient- and family-centered health care system: Recommendations and promising practices. Bethesda, MD: Institute for Patient- and Family-Centered Care, 2008.

3. Committee on Quality of Health Care in America: Institute of Medicine. Crossing the quality chasm: A new health system for the 21st century. Washington, DC: The National Academies Press, 2001.

4. The Royal Australian College of General Practitioners. Standards for general practices. 4th edn. Melbourne: RACGP, 2010.

5. Australian Commission on Safety and Quality in Health Care. Safety and quality improvement guide Standard 1: Governance for safety and quality in health service organisations (October 2012). Sydney: ACSQHC, 2012.

6. American Academy of Family Physicians, American Academy of Pediatrics, American College of Physicians, American Osteopathic Association. Joint principles of the patient-centered medical home. Leawood, KS: AAFP, 2007.

7. The Royal Australian College of General Practitioners. What is general practice? East Melbourne, Vic: RACGP, 2012. Available at www.racgp.org.au/becomingagp/what-is-a-gp/what-is-general-practice [Accessed 1 September 2013].

8. Saultz JW, Albedaiwi W. Interpersonal continuity of care and patient satisfaction: A critical review. Ann Fam Med 2004;2(5):445–51.

9. Grumbach K, Grundy P. Outcomes of implementing patient centred medical home interventions: A review of the evidence from prospective studies in the United States. Washington, DC: Patient-Centred Primary Care Collaborative, 2010.

10. Geisinger Health System. Presentation at White House Roundtable on advanced models of primary care. 10 August, 2009. Washington, DC: Geisinger Health System, 2009.

11. Steiner BD, Denham AC, Ashkin E, Newton WP, Wroth T, Dobson LA Jr. Community care of North Carolina: Improving care through community health networks. Ann Fam Med 2008;6(4):361–67.

12. Scholle SH. Developing and testing measures of patient centred care. Washington, DC: The Commonwealth Fund Health Care Quality Survey, 2006.

13. Beal A. Closing the divide: How medical homes promote equity in health care. New York: Commonwealth Fund, 2007.

14. MacKee N. Medical home potential. MJA Insight;Issue 21, 8 June 2015 [Accessed 15 February 2016].

15. Friedberg MW, Rosenthal MB, Werner RM, Volpp KG, Schneider EC. Effects of a Medical Home and Shared Savings Intervention on Quality and Utilization of Care. JAMA Intern Med 2015;175(8):1362–68.

16. Stellefson M, Dipnarine K, Stopka C. The chronic care model and diabetes management in US primary care settings: A systematic review. Prev Chronic Dis 2013;10:E26.

17. Wickramasinghe LK, Schattner P, Hibbert ME, Enticott JC, Georgeff MP, Russell GM. Impact on diabetes management of general practice management plans, team care arrangements and reviews. Med J Aust 2013;199(4):261–65.

18. The Royal Australian College of General Practitioners. A systems approach to the management of diabetes: A guide for general practice networks. South Melbourne, Vic: RACGP, 2010.

19. American Diabetes Association. Standards of medical care in diabetes – 2015. Diabetes Care 2015;38 Suppl 1:S1–94.

20. Harris MF, Jayasinghe UW, Taggart JR, et al. Multidisciplinary team care arrangements in the management of patients with chronic disease in Australian general practice. Med J Aust 2011;194(5):236–39.

21. Bellazzi R, Arcelloni M, Bensa G, et al. Design, methods, and evaluation directions of a multi-access service for the management of diabetes mellitus patients. Diabetes Technol Ther 2003;5(4):621–29.

22. Goldfracht M, Levin D, Peled O, et al. Twelve-year follow-up of a population-based primary care diabetes program in Israel. Int J Qual Health Care 2011;23(6):674–81.

23. Renders CM, Valk GD, Griffin S, Wagner EH, Eijk JT, Assendelft WJ. Interventions to improve the management of diabetes mellitus in primary care, outpatient and community settings. Cochrane Database Syst Rev 2001(1):CD001481.

24. Stroebel RJ, Scheitel SM, Fitz JS, et al. A randomized trial of three diabetes registry implementation strategies in a community internal medicine practice. Jt Comm J Qual Improv 2002;28(8):441–50.

25. Colagiuri S, Davies D, Girgis S, Colagiuri R. National evidence based guideline for case detection and diagnosis of type 2 diabetes. Canberra: Diabetes Australia, NHMRC, 2009.

26. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne, Vic: RACGP, 2012; p 55–57.

27. Australian Institute of Health and Welfare. Diabetes. AIHW; 2016. Available at www.aihw.gov.au/diabetes [Accessed 16 February 2016].

28. Whiting D, Unwin N, Roglic G. Diabetes: equity and social determinants. In: Blas E, Kurup AS, editors. Equity, social determinants and public health programmes. Geneva; WHO, 2010.

29. Chen L, Magliano DJ, Balkau B, et al. AUSDRISK: An Australian Type 2 Diabetes Risk Assessment Tool based on demographic, lifestyle and simple anthropometric measures. Med J Aust 2010;192(4):197–202.

30. Australian Institute of Health and Welfare. The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples 2015. Canberra: AIHW, 2015.

31. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2004;27 Suppl 1:S5–10.

32. Hermanns-Le T, Scheen A, Pierard GE. Acanthosis nigricans associated with insulin resistance: Pathophysiology and management. Am J Clin Dermatol 2004;5(3):199–203.

33. Twigg SM, Kamp MC, Davis TM, Neylon EK, Flack JR. Prediabetes: Position statement from the Australian Diabetes Society and Australian Diabetes Educators Association. Med J Aust 2007;186(9):461–65.

34. Selvin E, Steffes MW, Zhu H, et al. Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults. N Engl J Med 2010;362(9):800–11.

35. Sarwar N, Aspelund T, Eiriksdottir G, et al. Markers of dysglycaemia and risk of coronary heart disease in people without diabetes: Reykjavik prospective study and systematic review. PLoS Med 2010;7(5):e1000278.

36. World Health Organization. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: Report of a WHO/IDF consultation. Geneva: WHO, 2006.

37. d'Emden MC, Shaw JE, Colman PG, et al. The role of HbA1c in the diagnosis of diabetes mellitus in Australia. Med J Aust 2012;197(4):220–21.

38. d'Emden MC, Shaw JE, Jones GR, Cheung NW. Guidance concerning the use of glycated haemoglobin (HbA1c) for the diagnosis of diabetes mellitus. Med J Aust 2015;203(2):89–90.

39. Jones AG, Hattersley AT. The clinical utility of C-peptide measurement in the care of patients with diabetes. Diabet Med 2013;30(7):803–17.

40. Naik RG, Brooks-Worrell BM, Palmer JP. Latent autoimmune diabetes in adults. J Clin Endocrinol Metab 2009;94(12):4635–44.

41. Kavvoura FK, Owen KR. Maturity onset diabetes of the young: clinical characteristics, diagnosis and management. Pediatr Endocrinol Rev 2012;10(2):234–42.

42. Colagiuri R, Girgis S, Gomez M, Walker K, Colagiuri S, O'Dea K. National evidence based guideline for the primary prevention of type 2 diabetes. Canberra: Diabetes Australia and the NHMRC, 2009.

43. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002;346(6):393–403.

44. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. Diabetes Care 1997;20(4):537–44.

45. Ramachandran A, Snehalatha C, Mary S, Mukesh B, Bhaskar AD, Vijay V. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). Diabetologia 2006;49(2):289–97.

46. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001;344(18):1343–50.

47. Khavandi K, Amer H, Ibrahim B, Brownrigg J. Strategies for preventing type 2 diabetes: an update for clinicians. Ther Adv Chronic Dis 2013;4(5):242–61.

48. Emdin CA, Rahimi K, Neal B, Callender T, Perkovic V, Patel A. Blood pressure lowering in type 2 diabetes: A systematic review and meta-analysis. JAMA 2015;313(6):603–15.

49. Sumamo E, Ha C, Korownyk C, Vandermeer B, Dryden D. Lifestyle interventions for four conditions. Type 2 diabetes, metabolic syndrome, breast cancer, and prostate cancer. Technology Assessment Report. Rockville, MD: Agency for Healthcare Research and Quality, 2011.

50. Cavanaugh K, Wallston KA, Gebretsadik T, et al. Addressing literacy and numeracy to improve diabetes care: Two randomized controlled trials. Diabetes Care 2009;32(12):2149–55.

51. United States Department of Health and Human Services. Healthy People 2010. Washington, DC: US Department of Health and Human Services, 2000. Available at www.health.gov/healthypeople [Accessed 21 October 2013].

52. Australian Bureau of Statistics. Health literacy, Australia. Report no. 4233.0. Canberra: ABS, 2006. Available at www.abs.gov.au/ausstats/abs@.nsf/mf/4233.0 [Accessed 10 October 2013].

53. Edwards SG, Thompson AJ, Playford ED. Integrated care pathways: Disease-specific or process-specific? Clin Med 2004;4(2):132–35.

54. Silverman J, Kurtz SM, Drapper J. Skills for Communicating with Patients. 2nd edn. Oxford, UK: Radcliffe, 2005.

55. White RO, Wolff K, Cavanaugh KL, Rothman R. Addressing health literacy and numeracy to improve diabetes education and care. Diabetes Spectr 2010;23(4):238–43.

56. American Diabetes Association. Strategies for improving care. Sec. 1. In: Standards of Medical Care in Diabetes. Diabetes Care 2016;36 Suppl 1:S6–12.

57. National Institute for Health and Clinical Excellence. Type 2 diabetes in adults: management. [NG28]. London: NICE, 2015.

58. Wens J, Vermeire E, Hearnshaw H, Lindenmeyer A, Biot Y, Van Royen P. Educational interventions aiming at improving adherence to treatment recommendations in type 2 diabetes: A sub-analysis of a systematic review of randomised controlled trials. Diabetes Res Clin Pract 2008;79(3):377–88.

59. Steinsbekk A, Rygg LO, Lisulo M, Rise MB, Fretheim A. Group based diabetes self-management education compared to routine treatment for people with type 2 diabetes mellitus. A systematic review with meta-analysis. BMC Health Serv Res 2012;12:213.

60. Loveman E, Frampton GK, Clegg AJ. The clinical effectiveness of diabetes education models for Type 2 diabetes: A systematic review. Health Technol Assess 2008;12(9):1–116, iii.

61. Lau AN, Tang T, Halapy H, Thorpe K, Yu CH. Initiating insulin in patients with type 2 diabetes. CMAJ 2012;184(7):767–76.

62. National Institute for Clinical Excellence. Guidance for the use of patient-education models for diabetes. Technology appraisal 60. London: NICE, 2003.

63. Nordmann AJ, Suter-Zimmermann K, Bucher HC, et al. Meta-analysis comparing Mediterranean to low-fat diets for modification of cardiovascular risk factors. Am J Med 2011;124(9):841–51. e2.

64. Briffa T, Maiorana A, Allan R, et al. On behalf of the Executive Working Group and National Forum Participants. National Heart Foundation of Australia physical activity recommendations for people with cardiovascular disease. Sydney, National Heart Foundation of Australia, 2006.

65. Scottish Intercollegiate Guidelines Network. Management of diabetes. A national clinical guideline. (SIGN publication no. 116.) [Updated May 2014]. Edinburgh: SIGN, 2014. Available at www.sign.ac.uk [Accessed 17 February 2016].

66. Orozco LJ, Buchleitner AM, Gimenez-Perez G, Roque IFM, Richter B, Mauricio D. Exercise or exercise and diet for preventing type 2 diabetes mellitus. Cochrane Database Syst Rev 2008(3):CD003054.

67. Ryden L, Grant PJ, Anker SD, et al. ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD: The Task Force on diabetes, pre-diabetes, and cardiovascular diseases of the European Society of Cardiology (ESC) and developed in collaboration with the European Association for the Study of Diabetes (EASD). Eur Heart J 2013;34(39):3035–87.

68. Department of Health. Australia's physical activity & sedentary behaviour guidelines for adults (18–64 years). Canberra: DOH, 2014.

69. Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. J Am Coll Cardiol 2001;37(1):153–56.

70. Gellish RL, Goslin BR, Olson RE, McDonald A, Russi GD, Moudgil VK. Longitudinal modeling of the relationship between age and maximal heart rate. Med Sci Sports Exerc 2007;39(5):822–29.

71. National Health and Medical Research Council. Australian Dietary Guidelines. Canberra: NHMRC, 2013.

72. Evert AB, Boucher JL, Cypress M, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care 2013;36(11):3821–42.

73. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. Circulation 1999;99(6):779–85.

74. Estruch R, Ros E, Salas-Salvado J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med 2013;368(14):1279–90.

75. Evert AB, Boucher JL, Cypress M, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care 2014;37(Suppl 1):S120–43.

76. Shukla AP, Iliescu RG, Thomas CE, Aronne LJ. Food order has a significant impact on postprandial glucose and insulin levels. Diabetes Care 2015;38(7):e98–99.

77. Cheng AY. Canadian Diabetes Association 2013 clinical practice guidelines for the prevention and management of diabetes in Canada. Introduction. Can J Diabetes 2013;37 Suppl 1:S1–212.

78. National Health and Medical Research Council. Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia. Melbourne: NHMRC, 2013.

79. Leslie WS, Hankey CR, Lean ME. Weight gain as an adverse effect of some commonly prescribed drugs: A systematic review. QJM 2007;100(7):395–404.

80. Anderson JW, Kendall CW, Jenkins DJ. Importance of weight management in type 2 diabetes: review with meta-analysis of clinical studies. J Am Coll Nutr 2003;22(5):331–39.

81. Bazzano LA, Serdula M, Liu S. Prevention of type 2 diabetes by diet and lifestyle modification. J Am Coll Nutr 2005;24(5):310–19.

82. Wing RR, Bolin P, Brancati FL, et al. Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. N Engl J Med 2013;369(2):145–54.

83. Rush E, Plank L, Chandu V, et al. Body size, body composition, and fat distribution: a comparison of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities. N Z Med J 2004;117(1207):U1203.

84. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne, Vic: RACGP, 2012; p 44.

85. National Heart Lung and Blood Institute. The practical guide: identification, evaluation and treatment of overweight and obesity in adults. Bethesda, MD: National Institutes of Health, 2000.

86. The Royal Australian College of General Practitioners. Supporting smoking cessation: A guide for health professionals. East Melbourne, Vic: RACGP, 2011.

87. US Department of Health and Human Services. How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable disease: A report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2010.

88. Targher G, Alberiche M, Zenere MB, Bonadonna RC, Muggeo M, Bonora E. Cigarette smoking and insulin resistance in patients with noninsulin-dependent diabetes mellitus. J Clin Endocrinol Metab 1997;82(11):3619–24.

89. National Health and Medical Research Council. Australian Guidelines to reduce health risks from drinking alcohol. Canberra: NHMRC, 2009.

90. Cheyne EH, Sherwin RS, Lunt MJ, Cavan DA, Thomas PW, Kerr D. Influence of alcohol on cognitive performance during mild hypoglycaemia; implications for Type 1 diabetes. Diabet Med 2004;21(3):230–37.

91. Delahanty LM, Grant RW, Wittenberg E, et al. Association of diabetes-related emotional distress with diabetes treatment in primary care patients with Type 2 diabetes. Diabet Med 2007;24(1):48–54.

92. McBain-Rigg KE, Veitch C. Cultural barriers to health care for Aboriginal and Torres Strait Islanders in Mount Isa. Aust J Rural Health 2011;19(2):70–74.

93. National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee). Guide to management of hypertension (Updated Dec 2010). Melbourne: National Heart Foundation of Australia, 2008.

94. Polonsky WH, Anderson BJ, Lohrer PA, et al. Assessment of diabetes-related distress. Diabetes Care 1995;18(6):754–60.

95. Kroenke K, Spitzer RL, Williams JB. The Patient Health Questionnaire-2: Validity of a two-item depression screener. Med Care 2003;41(11):1284–92.

96. Colagiuri S, Dickinson S, Girgis S, Colagiuri R. National evidence based guideline for blood glucose control in type 2 diabetes. Canberra: NHMRC, 2009.

97. Koenig RJ, Peterson CM, Kilo C, Cerami A, Williamson JR. Hemoglobin Alc as an indicator of the degree of glucose intolerance in diabetes. Diabetes 1976;25(3):230–32.

98. Sundaram RC, Selvaraj N, Vijayan G, Bobby Z, Hamide A, Rattina Dasse N. Increased plasma malondialdehyde and fructosamine in iron deficiency anemia: Effect of treatment. Biomed Pharmacother 2007;61(10):682–85.

99. Schnedl WJ, Trinker M, Lipp RW. HbA1c determination in patients with hemoglobinopathies. Diabetes Care 1999;22(2):368–69.

100. Bosi E, Scavini M, Ceriello A, et al. Intensive structured self-monitoring of blood glucose and glycemic control in noninsulin-treated type 2 diabetes: the PRISMA randomized trial. Diabetes Care 2013;36(10):2887–94.

101. Farmer AJ, Perera R, Ward A, et al. Meta-analysis of individual patient data in randomised trials of self monitoring of blood glucose in people with non-insulin treated type 2 diabetes. BMJ 2012;344:e486.

102. Malanda UL, Welschen LM, Riphagen II, Dekker JM, Nijpels G, Bot SD. Self-monitoring of blood glucose in patients with type 2 diabetes mellitus who are not using insulin. Cochrane Database Syst Rev 2012;1:CD005060.

103. Nauck MA, Haastert B, Trautner C, Muller UA, Nauck MA, Heinemann L. A randomised, controlled trial of self-monitoring of blood glucose in patients with type 2 diabetes receiving conventional insulin treatment. Diabetologia 2014;57(5):868–77.

104. Schnell O, Barnard K, Bergenstal R, et al. Clinical utility of SMBG: Recommendations on the use and reporting of SMBG in clinical research. Diabetes Care 2015;38(9):1627–33.

105. Inzucchi SE, Bergenstal RM, Buse JB, et al. Management of hyperglycemia in type 2 diabetes, 2015: A patient-centered approach: Update to a position statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care 2015;38(1):140–49.

106. Toth PP. Overview of saxagliptin efficacy and safety in patients with type 2 diabetes and cardiovascular disease or risk factors for cardiovascular disease. Vasc Health Risk Manag 2015;11:9–23.

107. Scirica BM, Braunwald E, Raz I, et al. Heart failure, saxagliptin, and diabetes mellitus: Observations from the SAVOR-TIMI 53 randomized trial. Circulation 2014;130(18):1579–88.

108. Paneni F. DPP-4 inhibitors, heart failure and type 2 diabetes: All eyes on safety. Cardiovasc Diagn Ther 2015;5(6):471–78.

109. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). UK Prospective Diabetes Study (UKPDS) Group. Lancet 1998;352(9131):854–65.

110. Bolen S, Tseng E, Hutfless S, et al. AHRQ comparative effectiveness reviews: Diabetes medications for adults with type 2 diabetes: an update. Rockville, MD: Agency for Healthcare Research and Quality, 2016.

111. Green JB, Bethel MA, Armstrong PW, et al. Effect of sitagliptin on cardiovascular outcomes in type 2 diabetes. N Engl J Med 2015;373(3):232–42.

112. White WB, Cannon CP, Heller SR, et al. Alogliptin after acute coronary syndrome in patients with type 2 diabetes. N Engl J Med 2013;369(14):1327–35.

113. Zannad F, Cannon CP, Cushman WC, et al. Heart failure and mortality outcomes in patients with type 2 diabetes taking alogliptin versus placebo in EXAMINE: A multicentre, randomised, double-blind trial. Lancet 2015;385(9982):2067–76.

114. Scirica BM, Bhatt DL, Braunwald E, et al. Saxagliptin and cardiovascular outcomes in patients with type 2 diabetes mellitus. N Engl J Med 2013;369(14):1317–26.

115. Chiasson JL, Josse RG, Gomis R, Hanefeld M, Karasik A, Laakso M. Acarbose treatment and the risk of cardiovascular disease and hypertension in patients with impaired glucose tolerance: The STOP-NIDDM trial. JAMA 2003;290(4):486–94.

116. Dormandy JA, Charbonnel B, Eckland DJ, et al. Secondary prevention of macrovascular events in patients with type 2 diabetes in the PROactive Study (PROspective pioglitAzone Clinical Trial In macroVascular Events): A randomised controlled trial. Lancet 2005;366(9493):1279–89.

117. Zinman B, Wanner C, Lachin JM, et al. Empagliflozin, cardiovascular outcomes, and mortality in type 2 diabetes. N Engl J Med 2015;373(22):2117–28.

118. Bentley-Lewis R, Aguilar D, Riddle MC, et al. Rationale, design, and baseline characteristics in evaluation of LIXisenatide in acute coronary syndrome, a long-term cardiovascular end point trial of lixisenatide versus placebo. Am Heart J 2015;169(5):631–38.e7.

119. Pfeffer M, et al. The evaluation of lixisenatide in acute coronary syndrome—The results of the ELIXA trial. Symposium at the 75th Scientific Sessions of the American Diabetes Association; Boston, MA; June 8, 2015.

120. UK Prospective Diabetes Study Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. Lancet 1998;352(9131):837–53.

121. Gerstein HC, Bosch J, Dagenais GR, et al. Basal insulin and cardiovascular and other outcomes in dysglycemia. N Engl J Med 2012;367(4):319–28.

122. Schernthaner G, Grimaldi A, Di Mario U, et al. GUIDE study: Double-blind comparison of once-daily gliclazide MR and glimepiride in type 2 diabetic patients. Eur J Clin Invest 2004;34(8):535–42.

123. Mosenzon O, Raz I. Intensification of insulin therapy for type 2 diabetic patients in primary care: basal-bolus regimen versus premix insulin analogs: when and for whom? Diabetes Care 2013;36 Suppl 2:S212–18.

124. Davoren P. Safe prescribing of metformin in diabetes. Aust Prescr 2014;37:2–5.

125. Zoungas S, Chalmers J, Kengne AP, et al. The efficacy of lowering glycated haemoglobin with a gliclazide modified release-based intensive glucose lowering regimen in the ADVANCE trial. Diabetes Res Clin Pract 201;89(2):126–33.

126. Trujillo JM, Nuffer W, Ellis SL. GLP-1 receptor agonists: A review of head-to-head clinical studies. Ther Adv Endocrinol Metab 2015;6(1):19–28.

127. Bennett WL, Wilson LM, Bolen S, et al. AHRQ comparative effectiveness reviews. Oral diabetes medications for adults with type 2 diabetes: an update. Rockville MD: Agency for Healthcare Research and Quality, 2011.

128. Tuccori M, Filion KB, Yin H, Yu OH, Platt AW, Azoulay L. Pioglitazone use and risk of bladder cancer: population based cohort study. BMJ 2016;352:i1541.

129. Qaseem A, Humphrey LL, Sweet DE, Starkey M, Shekelle P. Oral pharmacologic treatment of type 2 diabetes mellitus: A clinical practice guideline from the American College of Physicians. Ann Intern Med 2012;156(3):218–31.

130. Marso SP, Daniels GH, Brown-Frandsen K, et al. Liraglutide and cardiovascular outcomes in type 2 diabetes. N Engl J Med 201; 375(4):311–22.

131. Gunton JE, Cheung NW, Davis TM, Zoungas S, Colagiuri S. A new blood glucose management algorithm for type 2 diabetes: a position statement of the Australian Diabetes Society. Med J Aust 2014;201(11):650–53.

132. Cheung NW, Conn JJ, d'Emden MC, et al. Position statement of the Australian Diabetes Society: Individualisation of glycated haemoglobin targets for adults with diabetes mellitus. Med J Aust 2009;191(6):339–44.

133. Ahren B. Avoiding hypoglycemia: A key to success for glucose-lowering therapy in type 2 diabetes. Vasc Health Risk Manag 2013;9:155–63.

134. Stahl M, Berger W. Higher incidence of severe hypoglycaemia leading to hospital admission in Type 2 diabetic patients treated with long–acting versus short-acting sulphonylureas. Diabet Med 1999;16(7):586–90.

135. Singh SR, Ahmad F, Lal A, Yu C, Bai Z, Bennett H. Efficacy and safety of insulin analogues for the management of diabetes mellitus: a meta-analysis. CMAJ 2009;180(4):385–97.

136. Rosenstock J, Dailey G, Massi-Benedetti M, Fritsche A, Lin Z, Salzman A. Reduced hypoglycemia risk with insulin glargine: a meta-analysis comparing insulin glargine with human NPH insulin in type 2 diabetes. Diabetes Care 2005;28(4):950–55.

137. Umpierrez GE, Hellman R, Korytkowski MT, et al. Management of hyperglycemia in hospitalized patients in non-critical care setting: an endocrine society clinical practice guideline. J Clin Endocrinol Metab 2012;97(1):16–38.

138. Craig ME, Twigg SM, Donaghue KC, et al. National evidence-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: DOHA, 2011.

139. Australian Diabetes Educators Association. Clinical guiding principles for sick day management of adults with type 1 and type 2 diabetes. Technical document. Woden, ACT: ADEA, 2014.

140. Holman RR, Farmer AJ, Davies MJ, et al. Three-year efficacy of complex insulin regimens in type 2 diabetes. N Engl J Med 2009;361(18):1736–47.

141. National Prescribing Service. Early use of insulin and oral antidiabetic drugs. Prescribing Practice Review 40. Surry Hills, NSW: NPS, 2008.

142. Phillips P. KISS: 'keep insulin safe and simple'. Part 1: initiating insulin in type 2 diabetes. Medicine Today 2007;8(3):23–34.

143. Handelsman Y, Mechanick JI, Blonde L, et al. American Association of Clinical Endocrinologists medical guidelines for clinical practice for developing a diabetes mellitus comprehensive care plan. Endocr Pract 2011;17 Suppl 2:1–53.

144. Goudswaard AN, Furlong NJ, Rutten GE, Stolk RP, Valk GD. Insulin monotherapy versus combinations of insulin with oral hypoglycaemic agents in patients with type 2 diabetes mellitus. Cochrane Database Syst Rev 2004(4):CD003418.

145. Phillips PJ. Insulin and type 2 diabetes – A simple guide to prevent 'stuff ups'. Aust Fam Physician 2006;35(12):975–78.

146. Barnett A, Begg A, Dyson P, Feher M, Hamilton S, Munro N. Insulin for type 2 diabetes: choosing a second-line insulin regimen. Int J Clin Pract 2008;62(11):1647–53.

147. Fulcher G, Colagiuri S, Phillips P, et al. Insulin intensification for people with type 2 diabetes: A practical approach. Australasian Medical Journal 2010;3(12):808–13.

148. Perfetti R. Combining basal insulin analogs with glucagon-like peptide-1 mimetics. Diabetes Technol Ther 2011;13(9):873–81.

149. National Vascular Disease Prevention Alliance. Guidelines for the management of absolute cardiovascular disease risk. Melbourne: Stroke Foundation, 2012.

150. Baker IDI Heart and Diabetes Institute. National evidence-based guideline on secondary prevention of cardiovascular disease in type 2 diabetes (Part of the guidelines on management of type 2 diabetes). Melbourne: Baker IDI Heart and Diabetes Institute, 2015.

151. Saha S, Carlsson KS, Gerdtham UG, et al. Are lifestyle interventions in primary care cost-effective? An analysis based on a Markov model, differences-in-differences approach and the Swedish Bjorknas study. PLoS One 2013;8(11):e80672.

152. Saha S, Gerdtham UG, Johansson P. Economic evaluation of lifestyle interventions for preventing diabetes and cardiovascular diseases. Int J Environ Res Public Health 2010;7(8):3150–95.

153. Bangalore S, Kumar S, Lobach I, Messerli FH. Blood pressure targets in subjects with type 2 diabetes mellitus/impaired fasting glucose: Observations from traditional and bayesian random-effects meta-analyses of randomized trials. Circulation 2011;123(24):2799–810, 9 p following 810.

154. Brunstrom M, Carlberg B. Effect of antihypertensive treatment at different blood pressure levels in patients with diabetes mellitus: Systematic review and meta-analyses. BMJ 2016;352:i717.

155. Ettehad D, Emdin CA, Kiran A, et al. Blood pressure lowering for prevention of cardiovascular disease and death: A systematic review and meta-analysis. Lancet 2016;387(10022):957–67.

156. Cannon CP, Blazing MA, Giugliano RP, et al. Ezetimibe added to statin therapy after acute coronary syndromes. N Engl J Med 2015;372(25):2387–97.

157. Canner PL, Berge KG, Wenger NK, et al. Fifteen year mortality in Coronary Drug Project patients: Long-term benefit with niacin. J Am Coll Cardiol 1986;8(6):1245–55.

158. National Health and Medical Research Council. Guidelines for the management of diabetic retinopathy. Canberra: NHMRC, 2008.

159. Simo R, Hernandez C. Prevention and treatment of diabetic retinopathy: evidence from large, randomized trials. The emerging role of fenofibrate. Rev Recent Clin Trials 2012;7(1):71–80.

160. National Health and Medical Research Council. National evidence-based guideline: Prevention, identification and management of foot complications in diabetes. Canberra: NHMRC, 2011.

161. Yang Z, Chen R, Zhang Y, et al. Scoring systems to screen for diabetic peripheral neuropathy (Protocol). Cochrane Database Syst Rev 2014(4):CD010974.

162. Meijer JW, Smit AJ, Sonderen EV, Groothoff JW, Eisma WH, Links TP. Symptom scoring systems to diagnose distal polyneuropathy in diabetes: The Diabetic Neuropathy Symptom score. Diabet Med 2002;19(11):962–65.

163. Moore RA, Wiffen PJ, Derry S, McQuay HJ. Gabapentin for chronic neuropathic pain and fibromyalgia in adults. Cochrane Database Syst Rev 2011;CD007938.

164. Moore RA, Straube S, Wiffen PJ, Derry S, McQuay HJ. Pregabalin for acute and chronic pain in adults. Cochrane Database Syst Rev 2009;(3):CD007076.

165. Boulton AJ, Vinik AI, Arezzo JC, et al. Diabetic neuropathies: A statement by the American Diabetes Association. Diabetes Care 2005;28(4):956–62.

166. Chadban S, Howell M, Twigg S, et al. National evidence based guideline for diagnosis, prevention and management of chronic kidney disease in type 2 diabetes. Canberra: NHMRC, 2009.

167. Thomas MC, Weekes AJ, Broadley OJ, Cooper ME, Mathew TH. The burden of chronic kidney disease in Australian patients with type 2 diabetes (the NEFRON study). Med J Aust 2006;185(3):140–44.

168. Kidney Health Australia. Chronic kidney disease (CKD) management in general practice, 3rd edn. Melbourne: Kidney Health Australia, 2015.

169. Australian Diabetes Society. Peri-operative diabetes management guidelines. Sydney: ADS, 2012. Available at https://diabetessociety.com.au/documents/ PerioperativeDiabetesManagementGuidelinesFINALCleanJuly2012.pdf [Accessed 12 February 2016].

170. Strippoli GF, Bonifati C, Craig M, Navaneethan SD, Craig JC. Angiotensin converting enzyme inhibitors and angiotensin II receptor antagonists for preventing the progression of diabetic kidney disease. Cochrane Database Syst Rev 2006(4):CD006257.

171. Palmer SC, Mavridis D, Navarese E, et al. Comparative efficacy and safety of blood pressure-lowering agents in adults with diabetes and kidney disease: A network meta-analysis. Lancet 2015;385(9982):2047–56.

172. Cheng J, Zhang W, Zhang X, et al. Effect of angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers on all-cause mortality, cardiovascular deaths, and cardiovascular events in patients with diabetes mellitus: A meta-analysis. JAMA Intern Med 2014;174(5):773–85.

173. Arnouts P, Bolignano D, Nistor I, et al. Glucose-lowering drugs in patients with chronic kidney disease: A narrative review on pharmacokinetic properties. Nephrol Dial Transplant 2014;29(7):1284–300.

174. Armstrong DG, Lavery LA, Harkless LB. Validation of a diabetic wound classification system. The contribution of depth, infection, and ischemia to risk of amputation. Diabetes Care 1998;21(5):855–59.

175. Lavery LA, Armstrong DG, Harkless LB. Classification of diabetic foot wounds. J Foot Ankle Surg 1996;35(6):528–31.

176. Expert Group for Endocrinology. Endocrinology guidelines, version 5. Melbourne: Therapeutic Guidelines Limited, 2014.

177. Druss BG, Marcus SC, Olfson M, Tanielian T, Elinson L, Pincus HA. Comparing the national economic burden of five chronic conditions. Health Aff (Millwood) 2001;20(6):233–41.

178. Maddigan SL, Feeny DH, Johnson JA. Health-related quality of life deficits associated with diabetes and comorbidities in a Canadian National Population Health Survey. Qual Life Res 2005;14(5):1311–20.

179. Wolff JL, Starfield B, Anderson G. Prevalence, expenditures, and complications of multiple chronic conditions in the elderly. Arch Intern Med 2002;162(20):2269–76.

180. Teljeur C, Smith SM, Paul G, Kelly A, O'Dowd T. Multimorbidity in a cohort of patients with type 2 diabetes. Eur J Gen Pract 2013;19(1):17–22.

181. Boyd CM, Fortin M. Future of multimorbidity research: How should understanding of multimorbidity Inform health system design? Public Health Reviews 2010;32(2):451–74.

182. Freund T, Kunz CU, Ose D, Szecsenyi J, Peters-Klimm F. Patterns of multimorbidity in primary care patients at high risk of future hospitalization. Popul Health Manag 2012;15(2):119–24.

183. Katon WJ, Lin EH, Von Korff M, et al. Collaborative care for patients with depression and chronic illnesses. N Engl J Med 2010;363(27):2611–20.

184. Sudore RL, Karter AJ, Huang ES, et al. Symptom burden of adults with type 2 diabetes across the disease course: Diabetes & aging study. J Gen Intern Med 2012;27(12):1674–81.

185. American Geriatrics Society Expert Panel on the Care of Older Adults with Multimorbidity. Guiding principles for the care of older adults with multimorbidity: An approach for clinicians. J Am Geriatr Soc 2012;60(10):E1–25.

186. Guthrie B, Payne K, Alderson P, McMurdo ME, Mercer SW. Adapting clinical guidelines to take account of multimorbidity. BMJ 2012;345:e6341.

187. Salisbury C. Multimorbidity: redesigning health care for people who use it. Lancet 2012;380(9836):7–9.

188. Gress S, Baan CA, Calnan M, et al. Co-ordination and management of chronic conditions in Europe: The role of primary care – position paper of the European Forum for Primary Care. Qual Prim Care 2009;17(1):75–86.

189. Rijken M, Bekkema N, Boeckxstaens P, Schellevis FG, De Maeseneer JM, Groenewegen PP. Chronic disease management programmes: An adequate response to patients' needs? Health Expect 2014;17(5):608–21.

190. Taylor D, Bury M. Chronic illness, expert patients and care transition. Sociol Health Illn 2007;29(1):27–45.

191. Thiem U, Theile G, Junius–Walker U, et al. Prerequisites for a new health care model for elderly people with multimorbidity: The PRISCUS research consortium. Z Gerontol Geriatr 2011;44(2):115–20.

192. Boult C, Wieland GD. Comprehensive primary care for older patients with multiple chronic conditions: 'Nobody rushes you through'. JAMA 2010;304(17):1936–43.

193. Healthcare Improvement Scotland. Living with multiple conditions: Issues, challenges and solutions. Edinburgh: Healthcare Improvement Scotland, 2012.

194. Noel PH, Parchman ML, Williams JW Jr, et al. The challenges of multimorbidity from the patient perspective. J Gen Intern Med 2007;22 Suppl 3:419–24.

195. Piette JD, Kerr EA. The impact of comorbid chronic conditions on diabetes care. Diabetes Care 2006;29(3):725–31.

196. Calderon-Larranaga A, Poblador-Plou B, Gonzalez-Rubio F, Gimeno-Feliu LA, Abad-Diez JM, Prados-Torres A. Multimorbidity, polypharmacy, referrals, and adverse drug events: Are we doing things well? Br J Gen Pract 2012;62(605):e821–26.

197. Schiff GD, Galanter WL, Duhig J, Lodolce AE, Koronkowski MJ, Lambert BL. Principles of conservative prescribing. Arch Intern Med 2011;171(16):1433–40.

198. Vyas A, Pan X, Sambamoorthi U. Chronic condition clusters and polypharmacy among adults. Int J Family Med 2012;2012:193168.

199. Bayliss EA, Steiner JF, Fernald DH, Crane LA, Main DS. Descriptions of barriers to self-care by persons with comorbid chronic diseases. Ann Fam Med 2003;1(1):15–21.

200. Rogowski J, Lillard LA, Kington R. The financial burden of prescription drug use among elderly persons. Gerontologist 1997;37(4):475–82.

201. Karter AJ, Stevens MR, Herman WH, et al. Out-of-pocket costs and diabetes preventive services: the Translating Research Into Action for Diabetes (TRIAD) study. Diabetes Care 2003;26(8):2294–99.

202. Piette JD, Heisler M, Wagner TH. Problems paying out-of-pocket medication costs among older adults with diabetes. Diabetes Care 2004;27(2):384–91.

203. West SD, Nicoll DJ, Stradling JR. Prevalence of obstructive sleep apnoea in men with type 2 diabetes. Thorax 2006;61(11):945–50.

204. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne, Vic: RACGP, 2012; p 60–72.

205. Palta P, Schneider AL, Biessels GJ, Touradji P, Hill-Briggs F. Magnitude of cognitive dysfunction in adults with type 2 diabetes: a meta-analysis of six cognitive domains and the most frequently reported neuropsychological tests within domains. J Int Neuropsychol Soc 2014;20(3):278–91.

206. Monette MC, Baird A, Jackson DL. A meta-analysis of cognitive functioning in nondemented adults with type 2 diabetes mellitus. Can J Diabetes 2014;38(6):401–08.

207. Barbagallo M, Dominguez LJ. Type 2 diabetes mellitus and Alzheimer's disease. World J Diabetes 2014;5(6):889–93.

208. Speight J, Browne JL, Holmes-Truscott E, Hendrieckx C, Pouwer F, on behalf of the Diabetes MILES–Australia reference group. Diabetes MILES–Australia 2011 Survey Report. Melbourne: Diabetes Australia, 2011.

209. Mezuk B, Eaton WW, Albrecht S, Golden SH. Depression and type 2 diabetes over the lifespan: A meta-analysis. Diabetes Care 2008;31(12):2383–90.

210. Morita I, Inagaki K, Nakamura F, et al. Relationship between periodontal status and levels of glycated hemoglobin. J Dent Res 2012;91(2):161–66.

211. Preshaw PM, Alba AL, Herrera D, et al. Periodontitis and diabetes: A two-way relationship. Diabetologia 2012;55(1):21–31.

212. Simpson TC, Needleman I, Wild SH, Moles DR, Mills EJ. Treatment of periodontal disease for glycaemic control in people with diabetes. Cochrane Database Syst Rev 2010(5):CD004714.

213. Teeuw WJ, Gerdes VE, Loos BG. Effect of periodontal treatment on glycemic control of diabetic patients: A systematic review and meta-analysis. Diabetes Care 2010;33(2):421–27.

214. Australian Bureau of Statistics. Diabetes in Australia: A snapshot, 2007–08. Report no. 4820.0.55.001. Canberra: ABS, 2011. Available at www.abs.gov.au/ausstats/abs@.nsf/mf/4820.0.55.001 [Accessed 7 March 2016].

215. PricewaterhouseCoopers. Evaluation of the DAA/PMP programs, June 2010. Canberra: DOHA, 2010.

216. Clifford R, Davis T, Batty K, Davis W. Prevalence and predictors of complementary medicine usage in diabetes: Fremantle Diabetes Study. J Pharm Pract Res 2003;33(4):260–64.

217. Medicare. Practice Incentives Program. Diabetes Incentive guidelines – July 2012. Canberra: DOHS, 2012.

218. The Royal Australian College of General Practitioners. Putting prevention into practice. Guidelines for the implementation of prevention in the general practice setting. South Melbourne, Vic: RACGP, 2006.

219. Alderman CP, Kong L, Kildea L. Medication-related problems identified in home medicines reviews conducted in an Australian rural setting. Consult Pharm 2013;28(7):432–42.

220. Wermeille J, Bennie M, Brown I, McKnight J. Pharmaceutical care model for patients with type 2 diabetes: Integration of the community pharmacist into the diabetes team – A pilot study. Pharm World Sci 2004;26(1):18–25.

221. Holland R, Smith R, Harvey I. Where now for pharmacist led medication review? J Epidemiol Community Health 2006;60(2):92–93.

222. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne, Vic: RACGP, 2012; p 28.

223. Braun L, Cohen M. Herbs and natural supplements: An evidence-based guide. 3rd edn. Sydney: Churchill Livingstone Elsevier, 2010.

224. Norman RJ, Dewailly D, Legro RS, Hickey TE. Polycystic ovary syndrome. Lancet 2007;370(9588):685–97.

225. Lo JC, Feigenbaum SL, Escobar GJ, Yang J, Crites YM, Ferrara A. Increased prevalence of gestational diabetes mellitus among women with diagnosed polycystic ovary syndrome: a population-based study. Diabetes Care 2006;29(8):1915–17.

226. Legro RS, Arslanian SA, Ehrmann DA, et al. Diagnosis and treatment of polycystic ovary syndrome: An endocrine society clinical practice guideline. J Clin Endocrinol Metab 2013;98(12):4565–92.

227. Teede HJ, Misso ML, Deeks AA, et al. Assessment and management of polycystic ovary syndrome: Summary of an evidence-based guideline. Med J Aust 2011;195(6):S65–112.

228. Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. Clin Epidemiol 2013;6:1–13.

229. Boyle J, Teede HJ. Polycystic ovary syndrome – An update. Aust Fam Physician 2012;41(10):752–56.

230. Gilbert C, Valois M, Koren G. Pregnancy outcome after first-trimester exposure to metformin: A meta-analysis. Fertil Steril 2006;86(3):658–63.

231. Rowan JA, Rush EC, Obolonkin V, Battin M, Wouldes T, Hague WM. Metformin in gestational diabetes: The offspring follow-up (MiG TOFU): body composition at 2 years of age. Diabetes Care 2011;34(10):2279–84.

232. Lautatzis ME, Goulis DG, Vrontakis M. Efficacy and safety of metformin during pregnancy in women with gestational diabetes mellitus or polycystic ovary syndrome: a systematic review. Metabolism 2013;62(11):1522–34.

233. Camelo Castillo W, Boggess K, Sturmer T, Brookhart MA, Benjamin DK Jr, Jonsson Funk M. Association of adverse pregnancy outcomes with glyburide vs insulin in women with gestational diabetes. JAMA Pediatr 2015;169(5):452–58.

234. Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008;358(19):1991–2002.

235. Evers IM, de Valk HW, Visser GH. Risk of complications of pregnancy in women with type 1 diabetes: Nationwide prospective study in the Netherlands. BMJ 2004;328(7445):915.

236. Sheth BP. Does pregnancy accelerate the rate of progression of diabetic retinopathy? Curr Diab Rep 2002;2(4):327–30.

237. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne, Vic: RACGP, 2012; p 12.

238. Forehan S. Thyroid disease in the perinatal period. Aust Fam Physician 2012;41(8):578–81.

239. Edipidis K. Pregnancy in women with renal disease. Yes or no? Hippokratia 2011;15(Suppl 1):8–12.

240. National Institute for Health and Care Excellence. Diabetes in pregnancy: management of diabetes and its complications from preconception to the postnatal period (NG3). London: NICE, 2015.

241. Moretti ME, Rezvani M, Koren G. Safety of glyburide for gestational diabetes: A meta-analysis of pregnancy outcomes. Ann Pharmacother 2008;42(4):483–90.

242. National Collaborating Centre for Women's and Children's Healthcare Improvement Scotland. Diabetes in pregnancy: Management of diabetes and its complications from preconception to the postnatal period. Evidence tables. London: RCOG, 2008.

243. Donovan P. Hypertensive disorders of pregnancy. Aust Prescr 2012;35:47–50.

244. Hague W. Metformin in pregnancy and lactation. Australian Prescriber 2007;30:68–69.

245. Moses RG, Morris GJ, Petocz P, San Gil F, Garg D. The impact of potential new diagnostic criteria on the prevalence of gestational diabetes mellitus in Australia. Med J Aust 2011;194(7):338–40.

246. Sacks DA, Hadden DR, Maresh M, et al. Frequency of gestational diabetes mellitus at collaborating centers based on IADPSG consensus panel-recommended criteria: The Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study. Diabetes Care 2012;35(3):526–28.

247. Tieu J, McPhee AJ, Crowther CA, Middleton P. Screening and subsequent management for gestational diabetes for improving maternal and infant health. Cochrane Database Syst Rev 2014;2:CD007222.

248. Nankervis A, McIntyre HD, Moses R, et al. ADIPS consensus guidelines for the testing and diagnosis of gestational diabetes mellitus in Australia. Australasian Diabetes in Pregnancy Society, 2012. Available at http://adips.org/downloads/2014ADIPSGDMGuidelinesV18.11.2014_000.pdf [Accessed 11 March 2016].

249. National Institutes of Health. Consensus development conference statement. Diagnosing gestational diabetes mellitus conference March 4–6. Bethesda, MD: NIH, 2013. Available at http://prevention.nih.gov/cdp/conferences/2013/gdm/final-statement.aspx [Accessed 5 July 2016].

250. Duran A, Saenz S, Torrejon MJ, et al. Introduction of IADPSG criteria for the screening and diagnosis of gestational diabetes mellitus results in improved pregnancy outcomes at a lower cost in a large cohort of pregnant women: The St. Carlos Gestational Diabetes Study. Diabetes Care 2014;37(9):2442–50.

251. O'Sullivan EP, Avalos G, O'Reilly M, Dennedy MC, Gaffney G, Dunne F. Atlantic diabetes in pregnancy (DIP): The prevalence and outcomes of gestational diabetes mellitus using new diagnostic criteria. Diabetologia 2011;54(7):1670–75.

252. Ethridge JK Jr, Catalano PM, Waters TP. Perinatal outcomes associated with the diagnosis of gestational diabetes made by the international association of the diabetes and pregnancy study groups criteria. Obstet Gynecol 2014;124(3):571–78.

253. Lapolla A, Dalfra MG, Ragazzi E, De Cata AP, Fedele D. New International Association of the Diabetes and Pregnancy Study Groups (IADPSG) recommendations for diagnosing gestational diabetes compared with former criteria: A retrospective study on pregnancy outcome. Diabet Med 2011;28(9):1074–77.

254. Koivusalo SB, Rono K, Klemetti MM, et al. Gestational diabetes mellitus can be prevented by lifestyle intervention: The Finnish Gestational Diabetes Prevention Study (RADIEL): A randomized controlled trial. Diabetes Care 2016;39(1):24–30.

255. Noctor E, Dunne FP. Type 2 diabetes after gestational diabetes: the influence of changing diagnostic criteria. World J Diabetes 2015;6(2):234–44.

256. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne, Vic: RACGP, 2012; p 57.

257. Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB. Impotence and its medical and psychosocial correlates: Results of the Massachusetts Male Aging Study. J Urol 1994;151(1):54–61.

258. Gandaglia G, Salonia A, Passoni N, Montorsi P, Briganti A, Montorsi F. Erectile dysfunction as a cardiovascular risk factor in patients with diabetes. Endocrine 2013;43(2):285–92.

259. Smith IA, McLeod N, Rashid P. Erectile dysfunction – When tablets don't work. Aust Fam Physician 2010;39(5):301–05.

260. Welch GW, Jacobson AM, Polonsky WH. The Problem Areas in Diabetes Scale. An evaluation of its clinical utility. Diabetes Care 1997;20(5):760–66.

261. Robins M, Coles M, Smith D, Armstrong M, Bryant W, Homeming L, on behalf of the Australian Diabetes Educators Association. Clinical guiding principles for sick day management of adults with type 1 and type 2 diabetes. Technical document. Canberra: ADEA, 2014.

262. Australian Diabetes Educators Association. Sick day management of adults with type 2 diabetes. Canberra: ADEA, 2014.

263. Sarre R. Bowel preparation. Aust Prescr 2005;28(1):16–17.

264. Austroads and National Transport Commission. Assessing fitness to drive. 4th edn (amended up to 30 June 2014). Sydney: Austroads and NTC, 2012.

265. Diabetes UK. End of life diabetes care: Full strategy document. 2nd edn. London: Diabetes UK, 2013.

266. Cox DJ, Kovatchev BP, Gonder-Frederick LA, et al. Relationships between hyperglycemia and cognitive performance among adults with type 1 and type 2 diabetes. Diabetes Care 2005;28(1):71–77.

267. Sommerfield AJ, Deary IJ, Frier BM. Acute hyperglycemia alters mood state and impairs cognitive performance in people with type 2 diabetes. Diabetes Care 2004;27(10):2335–40.

268. Dunning T, Martin P, Savage S, Duggan N. Guidelines for managing diabetes at the end of life. Geelong, Vic: Nurses Board of Victoria, 2010.

269. Deakin University and Barwon Health, Diabetes Australia, Palliative Care Australia. Caring for people with diabetes at the end of life: A position statement. Geelong: Centre for Nursing and Allied Health Research, 2014.

270. Kevat DA, Sinha AK, McLean AG. Lower treatment targets for gestational diabetes: Is lower really better? Med J Aust 2014;201(4):204–07.

271. Siu AL. Screening for abnormal blood glucose and type 2 diabetes mellitus: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med 2015;163(11):861–68.

272. Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr 2013;97(3):505–16.

273. Volek JS, Fernandez ML, Feinman RD, Phinney SD. Dietary carbohydrate restriction induces a unique metabolic state positively affecting atherogenic dyslipidemia, fatty acid partitioning, and metabolic syndrome. Prog Lipid Res 2008;47(5):307–18.

274. Feinman RD. Fad diets in the treatment of diabetes. Curr Diab Rep 2011;11(2):128–35.

275. Accurso A, Bernstein RK, Dahlqvist A, et al. Dietary carbohydrate restriction in type 2 diabetes mellitus and metabolic syndrome: time for a critical appraisal. Nutr Metab (Lond) 2008;5:9.

276. Tay J, Luscombe-Marsh ND, Thompson CH, et al. Comparison of low- and high-carbohydrate diets for type 2 diabetes management: A randomized trial. Am J Clin Nutr 2015;102(4):780–90.

277. Kalra S, Sahay R, Gupta Y. Sodium glucose transporter 2 (SGLT2) inhibition and ketogenesis. Indian J Endocrinol Metab 2015;19(4):524–28.

278. Bell DS. Metformin-induced vitamin B12 deficiency presenting as a peripheral neuropathy. South Med J 2010;103(3):265–67.

279. Chiasson JL. Acarbose for the prevention of diabetes, hypertension, and cardiovascular disease in subjects with impaired glucose tolerance: The Study to Prevent Non-Insulin-Dependent Diabetes Mellitus (STOP-NIDDM) trial. Endocr Pract 2006; 12 Suppl 1:25–30.

280. Azoulay L, Yin H, Filion KB, et al. The use of pioglitazone and the risk of bladder cancer in people with type 2 diabetes: Nested case-control study. BMJ 2012;344:e3645.

281. Hsiao FY, Hsieh PH, Huang WF, Tsai YW, Gau CS. Risk of bladder cancer in diabetic patients treated with rosiglitazone or pioglitazone: A nested case-control study. Drug Saf 2013;36(8):643–49.

282. Freeman JS. A physiologic and pharmacological basis for implementation of incretin hormones in the treatment of type 2 diabetes mellitus. Mayo Clin Proc 2010;85(12 Suppl):S5–14.

283. Gunton JE, Cheung NW, Davis TM, Zoungas S, Colagiuri S. A new blood glucose management algorithm for type 2 diabetes: A position statement of the Australian Diabetes Society. Appendix 1. Med J Aust 2014;201(11):650–53.

284. Nathan DM, Buse JB, Davidson MB, et al. Medical management of hyperglycemia in type 2 diabetes: A consensus algorithm for the initiation and adjustment of therapy: A consensus statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care 2009;32(1):193–203.

285. Wong J, Tabet E. The introduction of insulin in type 2 diabetes mellitus. Aust Fam Physician 2015;44(5):278–83.

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National Diabetes Services Scheme

The NDSS supports your patients with diabetes

If you have a patient with diabetes, make sure you register them with the NDSS to provide them with life-long access to diabetes education and support services. Where they meet the eligibility criteria, they will also have access to subsidised products to help them manage their diabetes.

Registration is free and only done once.

The reverse side of this page has information on the NDSS and how to register your patients. You can pull this card out of the booklet and place it on your desk or nearby for easy reference.

The National Diabetes Services Scheme is an initiative of the Australian Government administered with the assistance of Diabetes Australia.

The National Diabetes Services Scheme (NDSS) commenced in 1987, as an initiative of the Australian Government administered by Diabetes Australia.

Why register?

When you register your patients with diabetes on the NDSS, they will have life-long access to diabetes education and support. Subject to meeting NDSS eligibility criteria, they will also have access to subsidised products to help them manage their diabetes.

On registration, your patient will receive a 'starter' information pack, and a few months later they will receive a follow up phone call about their self-management of their diabetes.

Your patients will also be able to access a range of free diabetes information and support services. Encourage your patients to contact their local NDSS Agent on the NDSS Helpline 1300 136 588 to find out about the services they can take advantage of to improve their knowledge and management of diabetes.

Subject to eligibility, they will also be able to access:

» subsidised blood glucose and urine testing strips

» free needles and syringes for people with diabetes who require insulin or an approved non-insulin injectable medication

» subsidised insulin pump consumables for people with type 1 diabetes and gestational diabetes.

For a full list of products and prices, download the order form at www.ndss.com.au

Who can register?

All Australians who hold a Medicare card and have been diagnosed with diabetes can receive the benefits of NDSS registration.

How to register

Registration is free and is as simple as completing the NDSS registration form:

» Fill in the NDSS registration form.

» Ensure the form is signed by a medical practitioner or credentialled diabetes educator.

» Submit the completed registration form to the NDSS via:

– email ndss@diabetesaustralia.com.au

– fax 1300 536 953 or

– an NDSS Access Point.

There is a national network of NDSS community pharmacy Access Points. To find the nearest Access Point phone 1300 136 588 or visit osd.ndss.com.au

You can order multiple copies of the registration form by phoning your local NDSS Agent on 1300 136 588 or you can download it from the NDSS website www.ndss.com.au

National Gestational Diabetes Register

All women diagnosed with gestational diabetes and registered with the NDSS are also registered on the NDSS National Gestational Diabetes Register.

How registration will help your patients with gestational diabetes

Your patients will receive:

» regular reminders to have follow up diabetes screening after the birth

» valuable information on how to maintain a healthy lifestyle and minimise the risk of developing type 2 diabetes in the future.

Your patients with gestational diabetes will receive these benefits when you register them with the NDSS.

For more information

Refer your patients to their local NDSS Agent
P: 1300 136 588   W: www.ndss.com.au

Type 2 diabetes: Goals for optimum management

Encourage all people with type 2 diabetes to approach/reach these goals	
Diet	Advise eating according to *Australian dietary guidelines*, with attention to quantity and type of food If concerns are held regarding cardiovascular disease risk, advise individual dietary review
Body mass index (BMI)	Therapeutic goal is 5–10% weight loss for people who are overweight or obese with type 2 diabetes Those with BMI >35 kg/m^2 and comorbidities, or BMI >40 kg/m^2, greater weight loss measures should be considered Note that BMI is a difficult parameter to standardise between different population groups
Physical activity	At least 30 minutes of moderate physical activity on most if not all days of the week (total ≥150 minutes/week)
Cigarette consumption	0 per day
Alcohol consumption	Advise ≤2 standard drinks (20 g) per day for men and women
Blood glucose level (BGL)	Advise 6–8 mmol/L fasting and 8–10 mmol/L postprandial Ongoing self-monitoring of blood glucose is recommended for people with diabetes using insulin, people using sulphonylureas or other medicines that may cause hypoglycaemia, hyperglycaemia arising from illness, with haemoglobinopathies, pregnancy or other conditions where data on glycaemic patterns is required Routine self-monitoring of blood glucose in low-risk patients who are using oral glucose-lowering drugs (with the exception of sulphonylureas) is not recommended
Glycated haemoglobin (HbA1c)	Needs individualisation according to patient circumstances. Generally: • ≤53 mmol/mol (48–58 mmol/mol) • ≤7% (6.5–7.5%) Allowing for normal variation in test accuracy, HbA1c results that range between 6.5% and 7.5% (48 and 58 mmol/mol) would reflect this goal.
Total cholesterol <4.0 mmol/L	Initiation of pharmacotherapy is dependent on the assessment of absolute cardiovascular disease (CVD) risk (refer to the Australian absolute CVD risk calculator at www.cvdcheck.org.au). This requires using multiple risk factors, which is considered more accurate than the use of individual parameters Once therapy is initiated, the specified targets apply; however, these targets should be used as a guide to treatment and not as a mandatory target
High-density lipoprotein-cholesterol (HDL-C) ≥1.0 mmol/L	
Low-density lipoprotein-cholesterol (LDL-C) <2.0 mmol/L	
Non–HDL-C <2.5 mmol/L	
Triglycerides <2.0 mmol/L	
Blood pressure (BP) ≤140/90 mmHg	Lower BP targets may be considered for younger people and for secondary prevention in those at high risk of stroke, as long as treatment burden does not increase risk The target BP for people with diabetes and albuminuria/proteinuria remains <130/80 mmHg. As always, treatment targets should be individualised and monitored for side effects from medications used to lower BP
Urine albumin excretion	Urine albumin-to-creatinine ratio (UACR): • women: <3.5 mg/mmol • men: <2.5 mg/mmol. Timed overnight collection: <20 mcg/min Spot collection: <20 mg/L
Vaccination	Consider immunisation against influenza and pneumococcal disease, and the diphtheria-tetanus-acellular pertussis (dTpa) vaccine