

# Complications of diabetes

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## Introduction

Diabetes can result in a range of long-term complications in addition to causing acute metabolic reactions (e.g. ketoacidosis and diabetic coma). These complications are responsible for loss of working ability, invalidism, shortened life expectancy and reduced quality of life among people with diabetes.

People with diabetes are more prone to diseases of the large blood vessels (macrovascular disease) such as coronary heart disease, stroke and peripheral vascular disease as well as diseases of the small blood vessels (microvascular disease) such as retinopathy, kidney diseases and neuropathy (peripheral nerve disease). Other complications of or conditions associated with diabetes include digestive diseases (ulcers, coeliac disease, cancer of the pancreas, constipation, diarrhoea, liver disease and gallstones), infections, oral diseases, mental problems (depression and anxiety) and problems in pregnancy.

Complications arising from treatment can also occur in diabetes. These include hypoglycaemia from insulin or oral hypoglycaemic agents, side-effects of hypoglycaemic agents (liver toxicity, lactic acidosis, death from heart problems, allergic skin reactions), allergic reactions to insulin, and insulin resistance due to antibodies in the bloodstream binding the given insulin.

The underlying causes of diabetes complications remain controversial, although persistent high blood glucose and other consequences of insulin deficiency have been implicated. The Diabetes Control and Complications Trial, involving participants with Type 1 diabetes, showed that keeping blood glucose levels as close to normal as possible slows the onset and progression of eye, kidney and nerve diseases (NIDDK 2002). The United Kingdom Prospective Diabetes Study of people with Type 2 diabetes found that tight blood glucose control reduces the risk of major diabetic eye disorders by one-quarter and early kidney damage by one-third. Moreover, tight blood pressure control in people with high blood pressure reduces the risk of:

- death from long-term complications of diabetes by one-third;
- strokes by more than one-third; and
- serious deterioration of vision by more than a third (UKPDS 2002).

Improving the management and care of diabetes, particularly the early identification and reduction of risk factors, can delay the onset or slow the progression of complications.

Australian data on the complications of diabetes are limited. Those conditions for which data are currently available are discussed in more detail in this chapter.

## References and further reading

NIDDK (National Institute of Diabetes and Digestive and Kidney Diseases) 2002. Diabetes Control and Complications Trial. Viewed 2 May 2002, <<http://www.niddk.nih.gov/health/diabetes/pubs/dcct1/dcct.htm>>.

UKPDS (UK Prospective Diabetes Study) 2002. United Kingdom Prospective Diabetes Study. Viewed 2 May 2002, <<http://www.dtu.ox.ac.uk/index.html?maindoc=/ukpds/>>.

## Cardiovascular disease

Cardiovascular disease is a major complication of diabetes. People with diabetes are two to four times more likely to develop cardiovascular disease, their prognosis following a cardiovascular event (such as a heart attack) is not as good, nor do they fare as well after cardiac revascularisation procedures compared with those without diabetes (Wu, Brooks & Yue 1999). This section focuses on coronary heart disease, stroke and peripheral vascular disease, as they are the most common cardiovascular complications associated with diabetes.

### Coronary heart disease

Coronary heart disease is the most common cause of sudden death in Australia. It consists mainly of acute myocardial infarction (heart attack) and angina. A heart attack occurs when a vessel supplying blood to the heart muscle suddenly becomes blocked by a blood clot whereas angina is a temporary chest pain or discomfort caused by a reduced blood supply to the heart muscle.

### Stroke

Stroke (also referred to as cerebrovascular disease) includes ischaemic stroke, haemorrhagic stroke and transient ischaemic attack. The two main types of stroke are ischaemic, which occurs when an artery supplying blood to a part of the brain suddenly becomes blocked, and haemorrhagic, which is when an artery supplying blood to a part of the brain suddenly bleeds. These can damage part of the brain, which in turn can impair a range of functions including movement and speech. People who have had a transient ischaemic attack, which is a temporary cerebrovascular event that leaves no permanent damage, are at high risk for an acute ischaemic stroke attack.

### Peripheral vascular disease

Peripheral vascular disease occurs due to a reduced arterial blood supply to the legs. It ranges from asymptomatic disease, through pain on walking, to pain at rest. It can lead to amputation if blood supply

is significantly reduced. Although this is a significant cause of disability among people with peripheral vascular disease, the major cause of death in people with peripheral vascular disease is coronary heart disease.

### How does diabetes increase the risk of developing cardiovascular disease?

The reasons why diabetes increases the risk of cardiovascular disease are only partially understood. It is not clear, for example, whether the development of cardiovascular disease is the same in Type 1 and Type 2 diabetes, although both types are associated with an increased risk. It is also not clear why the increased risk of cardiovascular disease associated with diabetes is greater among women than men.

The prevailing explanation is that diabetes increases atherosclerosis (thickening of the walls of a blood vessel with deposits of plaque). However, improved blood glucose control by itself may not be sufficient to eliminate the excess risk of diabetic cardiovascular complications (Wu, Brooks & Yue 1999), despite its effectiveness in reducing the progression of diabetic microvascular complications such as eye and kidney disease (Diabetes Control and Complications Trial Research Group 1993).

Other factors possibly contributing to the excess risk of cardiovascular disease in diabetes include high blood pressure and dyslipidaemia (low levels of HDL cholesterol and high levels of LDL cholesterol and triglycerides). Both are risk factors for cardiovascular disease and their prevalence is higher among people with diabetes (Wu, Brooks & Yue 1999).

### Risk factors

The risk of developing cardiovascular disease increases when diabetes is present with other risk factors such as tobacco smoking, physical inactivity, high blood pressure, high blood cholesterol, and overweight and obesity.



## How many Australians with diabetes also have cardiovascular disease?

According to the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study, 12% of Australians aged 25 or over with diabetes had had a heart attack (approximately 113,000 people). Nine per cent had had a stroke (approximately 85,000 people). These proportions were much greater than among people without diabetes (3% and 2%, respectively).

There are limited national data on the number of Australians who have diabetes and peripheral vascular disease. Data collected through the Australian National Diabetes Information Audit and Benchmarking survey (refer to Appendixes for more information on data sources) in 2000 revealed that 14.5% of adults with diabetes also had peripheral vascular disease.

## General practice consultations

Data from the 1998–99 study of general practice activity in Australia show that the rate of diabetes in patients managed for coronary heart disease, stroke or peripheral vascular disease is far higher than the average, indicating a clear association between diabetes and cardiovascular disease.

## Hospitalisations

Diabetes increases the likelihood of being hospitalised for cardiovascular disease and of this occurring at a younger age.

In 1999–00, there were nearly 30,000 hospitalisations for coronary heart disease where diabetes was also present (19% of all hospitalisations for coronary heart disease). There were nearly 9,000 hospitalisations for stroke where diabetes was also present (17% of all hospitalisations for stroke) and approximately 1,700 hospitalisations for peripheral vascular disease where diabetes was also present (13% of all hospitalisations for peripheral vascular disease).

Males are much more likely to be hospitalised for cardiovascular disease than females irrespective of whether diabetes is present, although the presence of diabetes increases the likelihood of hospitalisation among females. The rate of hospitalisation for

coronary heart disease or stroke increased over the period 1993–94 to 1999–00 among both males and females with diabetes. The disparity between the sexes increased over this period also (refer to NHPA indicator 4.2 in Appendixes).

## Mortality

In 2000, diseases of the circulatory system were listed as the underlying cause of death in 55.7% of deaths where diabetes was an associated cause of death. Coronary heart disease accounted for almost two-thirds of these deaths while stroke accounted for one in five of these deaths. An examination of cardiovascular mortality associated with diabetes, such as this, is dependent on diabetes being recorded as a contributing cause of death on death certificates. This may not occur when death is sudden, as might occur with a heart attack.

Coronary heart disease (mainly heart attacks) was the leading cardiovascular cause of death in 2000, accounting for 26,521 deaths (21% of all deaths). Of these, 2,508 (9.5% of coronary heart disease deaths) recorded diabetes as a contributing cause of death. Stroke was the second most common cause of death, accounting for 12,354 deaths. Of these, 807 (6.5% of stroke deaths) recorded diabetes as a contributing cause of death. Peripheral vascular disease accounted for 2,046 deaths and of these, 32 (1.6% of deaths due to peripheral vascular disease) recorded diabetes as a contributing cause of death.

Males are more likely than females to die from cardiovascular disease. Death rates for coronary heart disease and peripheral vascular disease are almost twice as high among males but the difference between the sexes is not as great for stroke (Table 4.1). This pattern emerges irrespective of whether diabetes is recorded as a contributing cause.

Death rates for coronary heart disease or stroke where diabetes was recorded as a contributing cause of death fell slightly between 1997 and 2000 among both males and females (2.4% and 2.6%, respectively) (refer to NHPA indicator 5.2 in Appendixes). By comparison, over the same period, falls in the death rate for all cardiovascular disease were 4.4% and 3.8% for males and females, respectively.

**Table 4.1:** Death rate for coronary heart disease, stroke and peripheral vascular disease where diabetes was recorded as a contributing cause, by sex, 2000

	Coronary heart disease	Stroke	Peripheral vascular disease
	Rate (per 100,000 persons)		
Males	14.7	3.8	0.2
Females	8.3	3.1	0.1

#### Notes

1. The rates are age-standardised using the Australian population as at 30 June 1991.
2. The disease groupings are classified according to the ICD-10 codes: I20–I25 for coronary heart disease, G45, G46 and I60–I69 for stroke, I71–I74 for peripheral vascular disease and E10–E14 for diabetes as a contributing cause of death.

Source: AIHW National Mortality Database.

The higher prevalence of diabetes among people from different culturally and linguistically diverse backgrounds is also reflected in higher death rates for cardiovascular disease among this population group. Among males the death rate is nearly 25% higher and among females it is 35% higher compared with the death rates for coronary heart disease or stroke in the general population where diabetes was recorded as a contributing cause of death.

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1998–2000 Bettering the Evaluation and Care of Health Study (University of Sydney & Australian Institute of Health and Welfare).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

### References and further reading

AIHW (Australian Institute of Health and Welfare) 2001. Heart, stroke and vascular diseases—Australian facts 2001. Cardiovascular Disease Series No. 14. AIHW Cat. No. CVD 13. Canberra: AIHW, National Heart Foundation of Australia & National Stroke Foundation of Australia.

AIHW 2002. Australia's health 2002: the eighth biennial health report of the Australian Institute of Health and Welfare. AIHW Cat. No. AUS 25. Canberra: AIHW.

Diabetes Control and Complications Trial Research Group 1993. The effect of intensive therapy of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New England Journal of Medicine* 329:977–86.

Wingard DL & Barrett-Connor E 1995. Heart disease and diabetes. In: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. *Diabetes in America*. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, 429–48.

Wu T, Brooks B & Yue D 1999. Macrovascular disease: the sword of Damocles in diabetes. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney.

## Eye disease

People with diabetes are at an increased risk of developing eye disease, particularly diabetic retinopathy (retinal disease), cataract and glaucoma. Diabetic retinopathy is the most common cause of blindness in people aged 30–69 years (Constable 1999; Donnelly et al. 2000). Cataracts and glaucoma are also major causes of vision impairment and blindness among adults.

### Diabetic retinopathy

Diabetic retinopathy is a microvascular complication of diabetes caused by damage to the capillaries of the retina (the light-sensitive tissues at the back of the eye). It is a progressive disorder classified as non-proliferative or proliferative according to the presence of various clinical abnormalities (NHMRC 1997).

In the early stages, known as non-proliferative diabetic retinopathy (NPDR), retinal capillaries swell and leak fluid. NPDR is not usually associated with visual impairment. As the disease progresses (known as proliferative diabetic retinopathy or PDR) abnormal new capillaries grow on the surface of the retina. Without treatment, these capillaries can bleed causing cloudy vision or blindness. Abnormal fibrous tissue can also develop, leading to retinal detachment with severe vision loss. Blurred central vision may occur when the macular (the central part of the retina that gives the sharpest vision) swells from leaking fluid (called macular oedema).

Diabetic retinopathy is symptomless in its early phases. However, it can be treated successfully by laser surgery if identified early. It is estimated that early detection and timely treatment can prevent nearly all of severe vision loss and blindness due to diabetic retinopathy (Lee et al. 2001).

### Cataracts and glaucoma

A cataract is a clouding of the normally clear lens of the eye, leading to vision loss. A cloudy lens prevents light from entering the eye. Cataracts are more common and progress more rapidly in people with diabetes (Klein & Klein 1995).

Glaucoma is a condition where pressure builds up in the eye, pinching the capillaries that carry blood to the retina and optic nerve. Over time, the retina and optic nerve become damaged and vision is lost. People with diabetes are significantly more likely to develop glaucoma than people without diabetes (Klein & Klein 1995).

### Risk factors

Age at onset and duration of diabetes are key factors influencing the prevalence of diabetic retinopathy. In young people with diabetes (aged less than 30 years at diagnosis), the prevalence is as high as 25% during the first 5 years after diagnosis, increasing to 50% after 15 years since diagnosis. In older people (aged 30 years or more at diabetes diagnosis), up to 20% may have signs of retinopathy, rising to 60% after 15 years with diabetes (Mensah & Kohner 2002).

In addition to duration of diabetes, the risk of developing eye complications and visual impairment increases with coexisting medical problems or complications (such as high blood pressure and nephropathy), poor blood glucose control, pregnancy, elevated blood lipids and smoking (Cohen et al. 1998; NHMRC 1997).

### How many Australians with diabetes also have eye disease?

#### Diabetic retinopathy

The Australian Diabetes, Obesity and Lifestyle Study, carried out in 1999–2000, found that 15.4% of people with diabetes (known and newly diagnosed) had retinopathy. The prevalence among men was 14.0% and among women was 16.6%. The prevalence of retinopathy increased dramatically with duration of diabetes (duration 0–4 years 7.4%, 5–9 years 25.6%, 10–19 years 33.8%, and  $\geq 20$  years 60.5%). A similar rate of retinopathy was reported in a South Australian survey—19% of Type 2 diabetes sufferers had retinopathy (Phillips et al. 1998).

The National Divisions Diabetes Program Data Collation Project found that 11.5% of patients examined had retinopathy detected in at least one eye during 1999–00. Data on the prevalence of diabetic retinopathy among patients attending diabetes clinics are also available from the Australian National Diabetes Information Audit and Benchmarking (ANDIAB) survey. According to ANDIAB, of those patients who had a retinal assessment in 2000, 27.3% had retinopathy in the right eye and 27.1% had retinopathy in the left eye (NADC 2000). These latter estimates are not derived from population-based surveys. ANDIAB data reports on persons with diabetes requiring specialist clinical management, in particular those who have had poor control of their diabetes. Thus, ANDIAB figures may overstate the true rate of this condition among all people with diabetes. For further information on data sources refer to the 'Methods and data sources' section in the Appendixes.

### Cataracts and glaucoma

During the 1995 National Health Survey, 9.9% of respondents who reported ever having had diabetes also reported cataracts, and 3.2% reported glaucoma. These proportions were considerably higher than in people without diabetes (more than six times the rate of cataracts and more than four times the rate of glaucoma reported among persons without diabetes). A slightly larger proportion of females with diabetes reported cataracts than males (12.4% compared with 7.3% respectively). In contrast, more males than females reported glaucoma (4.6% compared with 1.8% respectively).

### Blindness

During the 1995 National Health Survey (NHS), 4.9% of respondents who reported ever having had diabetes also reported blindness. This was five times the rate reported among persons without diabetes. Similar proportions of males and females with diabetes reported blindness—5.6% for females and 4.2% for males.

Based on ANDIAB data for 2000, the incidence rate for blindness is estimated to be around 7 per 1,000 among persons with clinically diagnosed diabetes (refer to National Health Priority Areas indicator 3.2 in Appendixes). The ANDIAB figures are not directly comparable with the NHS figures because the NHS data on blindness was self-reported whereas the ANDIAB data was based on visual acuity measures.

### Hospitalisations

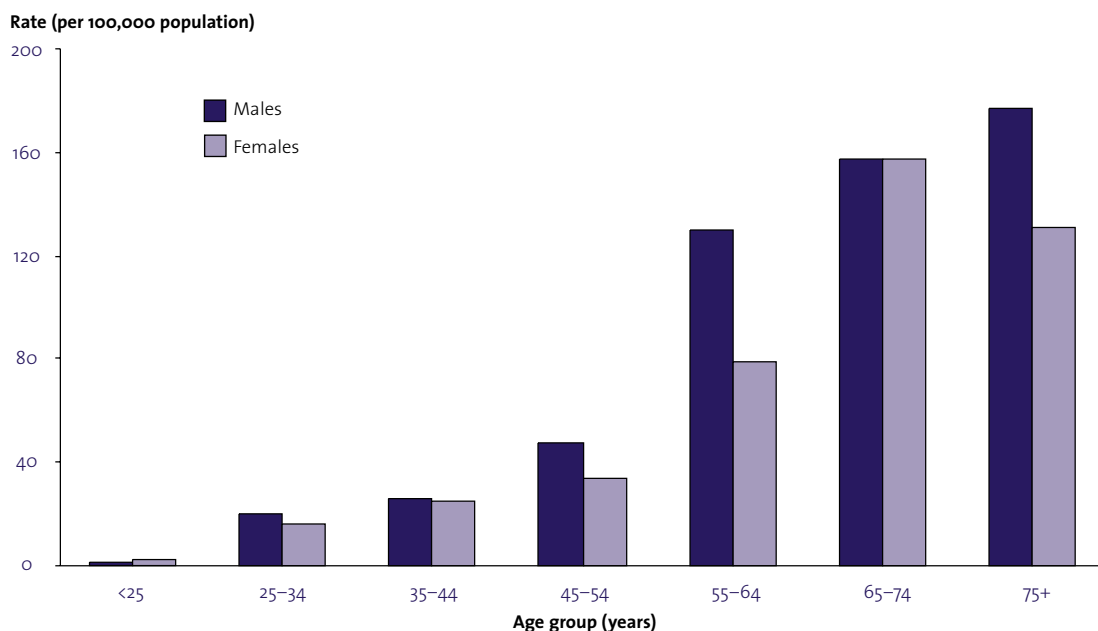
In 1999–00, there were 7,733 hospitalisations for diabetes-related eye complications (including retinopathy, glaucoma and cataract). These diabetes-specific disorders are discretely coded and accounted for 2.3% of all diabetes hospitalisations. Males were more likely to be hospitalised for diabetes-related eye complications than females. Hospital use for diabetes with eye complications tends to increase with age, with those aged 65 and over accounting for almost half of all hospitalisations for diabetes with eye complications in 1999–00 (Figure 4.1).

The average length of stay in hospital for diabetes with eye complications in 1999–00 was 7.5 days. Males and females tended to have a similar length of stay, 7.5 days and 7.4 days respectively.

### Aboriginal and Torres Strait Islander people

There are limited data on the prevalence of diabetic retinopathy in Aboriginal and Torres Strait Islander people. In a Western Australian study of diabetes, 31% of Aboriginal and Torres Strait Islander people were found to have diabetic retinopathy, compared with 20% of non-Indigenous Australians (Stanton et al. cited in OATSIH 2001). More recently, in a study of Indigenous Australians in a rural community, the prevalence of diabetic retinopathy was found to be 14% among those with diabetes (Keefe et al. cited in OATSIH 2001).

**Figure 4.1:** Hospitalisations for diabetes with eye complications, 1999–00



Source: AIHW National Hospital Morbidity Database.

The available data are likely to underestimate the magnitude of this problem among Aboriginal and Torres Strait Islander people. Aboriginal and Torres Strait Islander people often have compounding factors such as high blood pressure and diabetic nephropathy, both of which are associated with the development and severity of diabetic retinopathy (OATSIH 2001). It is also suggested that diabetic retinopathy may be more severe at the time of diagnosis among Aboriginal and Torres Strait Islander people as a result of delayed diagnosis of Type 2 diabetes (OATSIH 2001).

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1999–2000 National Divisions Diabetes Program (NDDP) Data Collation Project.

1995 National Health Survey (Australian Bureau of Statistics).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

### References and further reading

Carter S, Bonney M, Flack J, Burns J, Powell Davies PG & Harris MF 2000. National Divisions Diabetes Program Data Collation Project. Volume 5: Divisions of General Practice—Diabetes profiles. Quality of care and health outcomes—collated CARDIAB data. Sydney: Centre for General Practice Integration Studies, School of Community Medicine, University of New South Wales.

Cohen O, Norymberg K, Neumann E & Dekel H 1998. Complication-free duration and the risk of development of retinopathy in elderly diabetic patients. Archives of International Medicine 158:641–4.



Constable I 1999. Diabetic retinopathy: pathogenesis, clinical features and treatment. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney, 365–85.

Donnelly R, Emslie-Smith AM, Gardner ID & Morris AD 2000. ABC of arterial and venous disease. Vascular complications of diabetes. *British Medical Journal* 320:1062–66.

Klein R & Klein B 1995. Vision disorders in diabetes. In: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. *Diabetes in America*. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, 293–338.

Lee SJ, McCarty CA, Taylor HR & Keefe JE 2001. Costs of mobile screening for diabetic retinopathy: a practical framework for rural populations. *Australian Journal of Rural Health* 9:186–92.

Mensah E & Kohner EM 2002. Diagnosis and management of diabetic retinopathy. *Topical Endocrinology* 19:14–18.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

NHMRC (National Health and Medical Research Council) 1997. Management of diabetic retinopathy. Clinical practice guidelines, February 2002. Viewed 29 April 2002, <<http://www.health.gov.au/nhmrc/publications/pdfcover/cp53covr.htm>>.

OATSIH (Office for Aboriginal and Torres Strait Islander Health) 2001. Specialist eye health guidelines for use in Aboriginal and Torres Strait Islander populations: cataract, diabetic retinopathy, trachoma. Canberra: OATSIH.

Phillips P, Wilson D, Beilby J et al. 1998. Diabetes complications and risk factors in an Australian population. How well are they managed? *International Journal of Epidemiology* 27:853–9.

Taylor HR 1997. Eye health in Aboriginal and Torres Strait Islander Communities. Canberra: Commonwealth Department of Health and Aged Care.

## Kidney disease

Diabetes can affect the kidneys in a variety of ways, leading to serious and even life-threatening conditions. This section focuses on diabetic nephropathy and end-stage renal disease (ESRD), as these are the most common kidney complications associated with diabetes.

### Diabetic nephropathy

Diabetic nephropathy results from high blood glucose levels damaging the blood-filtering capillaries (glomeruli) in the kidneys. The glomeruli's filtering efficiency declines and blood proteins such as albumin leak into the urine (albuminuria).

In the early stages of diabetic nephropathy, small quantities of albumin leak into the urine (called microalbuminuria). As diabetic nephropathy progresses, the kidneys leak larger amounts of albumin (called macroalbuminuria or proteinuria). Microalbuminuria is a strong predictor of developing proteinuria, ESRD, high blood pressure and cardiovascular disease. Proteinuria indicates a substantial decline in kidney function and is associated with high mortality, particularly from ESRD. Individuals with proteinuria are also known to be at an increased risk of developing high blood pressure, coronary heart disease, peripheral vascular disease and retinopathy.

Diabetic nephropathy is often symptomless until late in the disease when therapeutic interventions are ineffective. However, early detection and intervention may slow or halt its progression.

Diabetic nephropathy can be readily detected by urine testing for albumin. Identifying and treating individuals with microalbuminuria, before there is a substantial decline in kidney function, is very important. Tight control of blood glucose and blood pressure may prevent microalbuminuria progressing to proteinuria or ESRD.

### End-stage renal disease (ESRD)

ESRD is the final stage in the worsening of kidney function, when the kidneys lose the ability to remove waste products such as creatinine and urea from the blood. Thereafter, dialysis (filtering of the blood by machine) or kidney transplantation is necessary to maintain life. Diabetic nephropathy is the second most common cause of ESRD in Australia (Russ 2001).

Measuring the glomerular filtration rate and the quantity of creatinine in the blood can indicate the severity of kidney damage.

### Risk factors

Factors that may determine whether diabetic nephropathy develops and progresses to ESRD include long duration of diabetes, poor blood glucose control, high blood pressure, genetic susceptibility to diabetic kidney disease and smoking.

### How many Australians with diabetes also have kidney disease?

During the 1995 National Health Survey, 6.1% of respondents who reported ever having had diabetes also reported having kidney disease, more than four times the rate reported among persons without diabetes. Similar proportions of males and females with diabetes reported kidney disease—6.3% for females and 5.9% for males.

According to the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab), 11.2% of Australians aged 25 or over with self-reported diabetes reported being treated for or suffering from kidney disease. Significantly more women with self-reported diabetes reported being treated for or suffering from kidney disease than men (18.4% compared with 5.2%).

## Diabetic nephropathy

Examining the prevalence of diabetic nephropathy in populations is problematic due to differences in methods of measurement for albuminuria and lack of standardised terminology. Health outcomes can only be assessed when the type of urine collection is recorded, allowing the appropriate units and reference limits to be applied.

Data on the prevalence of diabetic nephropathy based on urinary albumin measurements are available from the 1999–2000 AusDiab study. The prevalence of proteinuria was found to be more than four times higher in those with diabetes compared with those without (8.7% versus 1.9% respectively) (Chadban et al. unpub.). The prevalence of low glomerular filtration rate was also three times higher in those with diabetes compared with those without (27.6% compared with 9.8%) (Chadban et al. unpub.).

Data on the prevalence of albuminuria among patients attending diabetes clinics are available from the Australian National Diabetes Information Audit and Benchmarking collection (ANDIAB). Of those who had a urinary albumin assessment in 2000 (41.2% of patients) 66.4% had normal albumin levels (normoalbuminuria), 27.9% had microalbuminuria and 5.7% had macroalbuminuria (NADC 2000).

These data are also available from the National Divisions Diabetes Program (NDDP) Data Collation Project. In 1999–00, 1,284 of the 4,359 registered NDDP patients had albuminuria assessed. Of those, 76% had normoalbuminuria, 20.9% had microalbuminuria and 3.1% had macroalbuminuria.

## End-stage renal disease (ESRD)

Evidence of the burden of ESRD caused by diabetes is available from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry, a registry of people receiving kidney dialysis or a kidney transplant. New

cases of ESRD with diabetic nephropathy as the primary cause have increased dramatically over the past decade (refer to National Health Priority Areas indicator 3.1 in Appendixes). This increase has been most evident among patients with Type 2 diabetes.

In Australia, during 2000, diabetes was the second most common cause of primary kidney disease among ANZDATA patients, accounting for more than one in five (22%) new patients (Russ 2001). This represents a considerable increase in the proportion of ESRD cases with diabetes—from one in eight cases, or 12.5%, in 1991. The burden of ESRD from diabetes, particularly Type 2 diabetes, is likely to increase further as both the age of the population and prevalence of diabetes are projected to rise dramatically.

## Hospitalisations

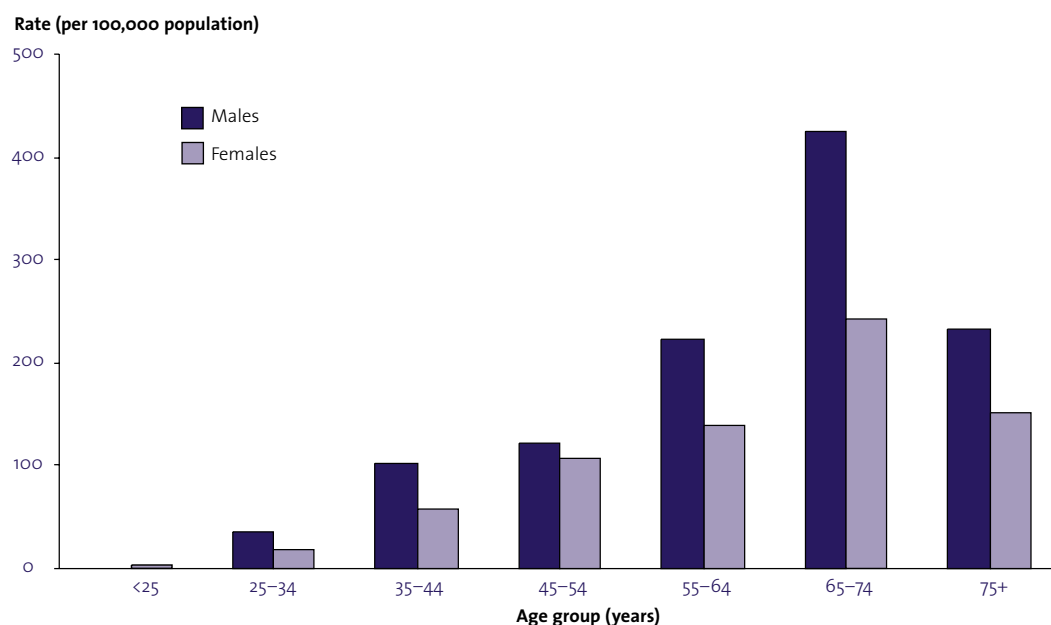
### Kidney (renal) complication

In 1999–00, there were 15,236 hospitalisations for diabetes with kidney complication; these accounted for 4.5% of hospital admissions for diabetes. Males with diabetes were much more likely to be hospitalised for kidney complication than females. Hospital use for diabetes with kidney complication increased with age and peaked at 65–74 years for both men and women (Figure 4.2).

The average length of stay in hospital for diabetes with kidney complication was 5.3 days. Females tended to have longer average lengths of stay than males (5.8 days compared with 4.9 days respectively).

Day-stay admissions for regular dialysis are extremely common for people with kidney disease (Australian Kidney Foundation 1999). After removal of day-stay admissions, the average length of stay in hospital for diabetes with kidney complication increased considerably to 10.1 days (10.2 days for males and 9.9 days for females).

**Figure 4.2:** Hospitalisations for diabetes with kidney complication, 1999–00



Source: AIHW National Hospital Morbidity Database.

### End-stage renal disease (ESRD)

In 1999–00 there were 2,691 admissions where ESRD was the principal diagnosis and diabetes was an additional diagnosis. Males with diabetes were more likely to be hospitalised for ESRD than females. The average length of stay in hospital for people with ESRD as the principal and diabetes as an additional diagnosis was 9.5 days. Length of stay in hospital was generally higher for females than for males (10.3 days compared with 8.8 days).

### Deaths

Kidney-related diseases were listed as an associated cause of death in 22.6% of cases where diabetes was listed as the underlying cause of death in 2000. In addition, diabetes was associated with 7% of deaths where kidney failure was the underlying cause of death.

### Aboriginal and Torres Strait Islander people

There are limited data on diabetic kidney complications among Aboriginal and Torres Strait Islander people. However, the available data indicate that Aboriginal and Torres Strait Islander people are proportionately much more likely to develop kidney disease and ESRD as a result of diabetes than the rest of the population.

Data from the ANZDATA Registry indicate that the yearly incidence of ESRD associated with diabetes is considerably higher among Aboriginal and Torres Strait Islander peoples. During 2000, 46% of Indigenous Australian patients had diabetic nephropathy compared with around 14% of non-Indigenous patients (Russ 2001). Nevertheless, the size of the problem of ESRD in Indigenous Australians is

likely to be underestimated by this register as a result of their poorer access to dialysis and transplant programs (Disney 1992; Russ 2001).

In a study in the Northern Territory, the incidence of ESRD associated with diabetes was 26.5 times higher in Aboriginal and Torres Strait Islander people compared with non-Indigenous Australians (90.2 per million cases in Indigenous Australians and 3.4 per million in non-Indigenous Australians) (Hoy et al. cited in de Courten et al. 1998). Moore et al. (1996) also found diabetic kidney disease to be significantly more common among Aboriginal and Torres Strait Islander patients than non-Indigenous patients (22% compared with 6% of non-Indigenous patients).

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1999–2000 National Divisions Diabetes Program (NDDP) Data Collation Project.

1995 National Health Survey (Australian Bureau of Statistics).

Australia and New Zealand Dialysis and Transplant Registry (ANZDATA).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

### References and further reading

ABS (Australian Bureau of Statistics) 2001. Causes of death, Australia, 2000. Cat. No. 3303.0. Canberra: ABS.

Australian Kidney Foundation 1999. The Australian kidney. National Epidemiological Survey of Diseases of the Kidney and Urinary Tract. Adelaide: Australian Kidney Foundation.

Chadban S, Briganti E, Kerr P, Dunstan D, Welborn T & Zimmet P (unpublished). Prevalence of kidney damage in Australian adults—The AusDiab Kidney Study.

de Courten M, Hodge A, Dowse G, King I, Vickery J & Zimmet P 1998. Review of the epidemiology, aetiology, pathogenesis and preventability of diabetes in Aboriginal and Torres Strait Islander populations. Canberra: Commonwealth Department of Health and Family Services.

Disney A (ed.) 1992. ANZDATA Registry Report 1992. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

Donnelly R, Emslie-Smith AM, Gardner ID & Morris AD 2000. ABC of arterial and venous disease. Vascular complications of diabetes. *British Medical Journal* 320:1062–66.

Hoy WE, Mathews JD & Pugsley DJ 1995. Treatment of Australian Aboriginals with end-stage renal disease in the top end of the Northern Territory: 1978–93. *Nephrology* 1:307–13.

Jerums G, Gilbert RE & Panagiotoulos S 1999. Diabetic nephropathy: recent concepts in mechanisms and management. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney, 365–85.

Moore L, Lloyd MS, Pugsley DJ & Seymour AE 1996. Renal disease in the Australian Aboriginal population: a pathological study. *Nephrology* 2:315–21.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

Russ GR (ed.) 2001. ANZDATA Registry Report 2001. Adelaide: Australia and New Zealand Dialysis and Transplant Registry.

## Neuropathy

Neuropathy (nerve damage) is a frequent complication of diabetes. Diabetic neuropathy usually manifests as either peripheral neuropathy, most commonly causing damage to the nerves in the feet, or autonomic neuropathy. The sequelae of diabetic neuropathy include pain, digestive problems, muscle weakness, non-healing ulcers and lower extremity amputation, and are associated with reduced quality of life and increased mortality. Diabetic neuropathy is generally a result of chronically high blood glucose levels which affect the metabolism of nerves. This in turn causes the accumulation of toxins which damage nerve structure and function.

### Peripheral neuropathy

Peripheral neuropathy is the presence of symptoms and/or signs of damage to the peripheral nerves (the nerves outside the brain and spinal cord) (Oyibo et al. 2002). Peripheral neuropathy can cause a diverse range of symptoms, depending on the nerve(s) affected, although some people will experience no obvious symptoms.

There are two broad types of peripheral neuropathy:

- Sensory neuropathy—affects the nerves that carry information to the brain about sensations from various parts of the body. Symptoms may include pain, tingling in the limbs or absence of feeling in the feet (which predisposes people with diabetes to foot trauma).
- Motor neuropathy—affects the nerves that carry signals to muscles to allow the muscles to move. Motor neuropathy can lead to muscle weakness, particularly in the feet, which may become deformed as a result.

### Autonomic neuropathy

Autonomic neuropathy affects the nerves that control involuntary body functions such as heart rate, blood pressure, sweating, and the action of the stomach, intestine and bladder. Symptoms may include dizziness and fainting, nausea, vomiting and diarrhoea, loss of bladder control and impotence in men.

### Risk factors

The risk of developing neuropathy increases with duration of diabetes, poor blood glucose control and age. Strict glycaemic control has been shown to reduce or prevent the development of neuropathy, and may alleviate neuropathic symptoms. Early identification is essential, especially in people with no obvious symptoms, to prevent the late sequelae of neuropathy. A combination of clinical observations and complex nerve function tests are often required to confirm the presence of diabetic neuropathy.

### How many Australians with diabetes also have neuropathy?

According to the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study, 10.3% of males and 9.4% of females with diabetes (known and newly diagnosed) had clinical signs of neuropathy. Also, 30.2% of men with self-reported diabetes reported suffering from or receiving treatment for impotence (difficulty getting or sustaining an erection).

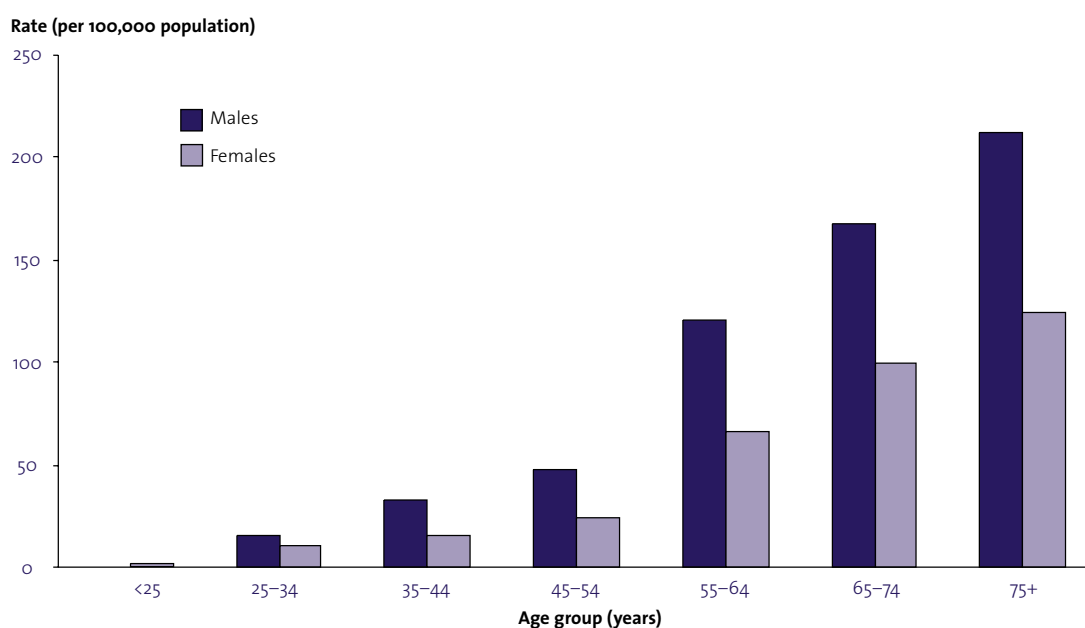
In the 2000 Australian National Diabetes Information and Benchmarking (ANDIAB) study, almost one-quarter (24.2%) of adult patients were recorded as having peripheral neuropathy following clinical assessment. However, it should be noted that ANDIAB data are obtained from specialist diabetes clinics that are likely to see more patients with complications. Impotence during the previous 12 months was also reported by 2.4% of male adult patients, with 25.7% reporting impotence before the previous 12 months.

### Hospitalisations

In 1999–00, there were 6,954 hospitalisations for diabetes-related neurological complications. These diabetes-specific neuropathies accounted for 2.1% of hospital admissions for diabetes.

Males were almost twice as likely as females to be admitted to hospital for diabetes with neurological complication. Hospital use for neurological complication increased dramatically among older people with diabetes, with around half of such cases being aged 65 and over in 1999–00 (Figure 4.3).

**Figure 4.3:** Hospitalisations for diabetes with neurological complication, 1999–00



Source: AIHW National Hospital Morbidity Database.

During 1999–00 the average length of stay in hospital for people with diabetes and neurological complication was 9.4 days. Males tended to have shorter average length of stay than females; 9.0 days compared with 10.1 days.

### Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

### References and further reading

American Diabetes Association & American Academy of Neurology 1988. Consensus statement. Report and recommendations of the San Antonio Conference on Diabetic Neuropathy. *Diabetes Care*, 11(7):592–7.

Feldman EV, Stevens MJ & Greene DA 1999. Diabetic neuropathy. In: Turtle J, Kaneko T & Osato S (eds). *Diabetes in the new millennium*. Sydney: Endocrinology and Diabetes Research Foundation, University of Sydney, 365–85.

Oyibo SO, Dang CN & Boulton AJM 2002. Diagnosis and management of diabetic neuropathy. *Topical Endocrinology* 19:10–13.

Vinik AI, Holland MT, Le Beau JM, Liuzzi FJ, Stansberry KB & Colen LB 1992. Diabetic neuropathies. *Diabetes Care* 15(12):1926–75.



## Foot complications

Diabetes is associated with nerve damage (peripheral neuropathy) and poor circulation (peripheral vascular disease—PVD) in the lower limbs. These factors increase the risk of developing foot ulcers and infections. Progression of these conditions in people with diabetes often leads to lower extremity amputations. Amputations are associated with increased morbidity and mortality and high treatment costs. Diabetes is estimated to account for approximately half of all non-traumatic amputations (DHAC & AIHW 1999).

### Foot ulcer

Over time, diabetes can damage the nerves in the feet, resulting in a loss of sensation. Reduced sensation of pain and discomfort from foreign bodies, injury or even tightly fitting shoes can predispose people to foot trauma and ulceration. Damage to nerves also causes wasting of the foot muscles, reduced joint mobility and foot deformities such as claw or hammer toes that are vulnerable to ulceration.

High blood glucose can also damage blood vessels in the lower limbs. Without a healthy supply of oxygen and nutrients, feet are predisposed to ulceration and infection.

Foot ulceration is a common reason for hospital admission for people with diabetes and is estimated to precede more than half of all diabetes-related amputations.

### Lower extremity amputation

The combination of diabetic neuropathy, PVD and foot deformity increases the risk of lower limb ulcers. Non-healing ulcers can result in gangrene (chronic infection resulting in tissue death). Amputation of the affected area may be necessary as a limb-salvaging procedure if medical treatment is unsuccessful.

Amputation is estimated to be 15 times more common in people with diabetes. Nearly half of the amputations in people with diabetes are minor

(involving toes, feet and ankles); the other half are major (below knee or above knee) (Campbell et al. 2000). Major amputations are associated with greater loss of limb function and require greater rehabilitation following amputation (Oyibo et al. 2002).

Many patients with diabetes who undergo amputation will have a subsequent amputation on the other side within a few years. The remaining limb becomes more vulnerable to ulceration and infection because it has to bear extra pressure.

### Risk factors

The risk of lower limb ulcers and amputations is higher in people who have had diabetes for 10 years or more, are male, have poor blood glucose control, have cardiovascular, visual or kidney complications, or smoke. Certain foot-related conditions are associated with an increased risk of foot ulcer and amputation: peripheral neuropathy (particularly loss of protective sensation), PVD, foot deformity, and prior history of foot ulcers or amputation.

Improved glycaemic control can prevent or reduce the development of diabetic neuropathy. Regular monitoring of the feet for early signs of diabetic neuropathy, peripheral vascular disease and foot deformities are essential. Appropriate therapeutic footwear, combined with podiatry care and footcare education, may also reduce the risk of serious foot ulcers and amputation.

### How many Australians with diabetes also have foot complications?

#### Foot ulcer

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) assessed the prevalence of various diabetes-related complications in Australia. Among people with known and newly diagnosed diabetes, 19.4% were found to be at risk of foot ulcer (defined by the presence of any one of neuropathy, PVD or history of foot ulceration). The greatest risk was



evident in those with a diabetes duration of 20 years or more (< 5 years 16.4%, 5–9 years 20.4%, 10–19 years 26.1% and  $\geq$  20 years 46.5%).

Based on Australian National Diabetes Information Audit and Benchmarking (ANDIAB) data for 2000, the prevalence of current foot ulcers among adult patients attending diabetes clinics was 3.0%. In addition, 6.2% of patients had a past history of foot ulcers. The vast majority (86.5%) of patients with a current foot ulcer had a past history of foot ulceration (NADC 2000). Also indicative of potential foot problems, peripheral neuropathy, PVD and foot deformity were recorded for a total of 24.2%, 12.6% and 5.6% of adult patients, respectively.

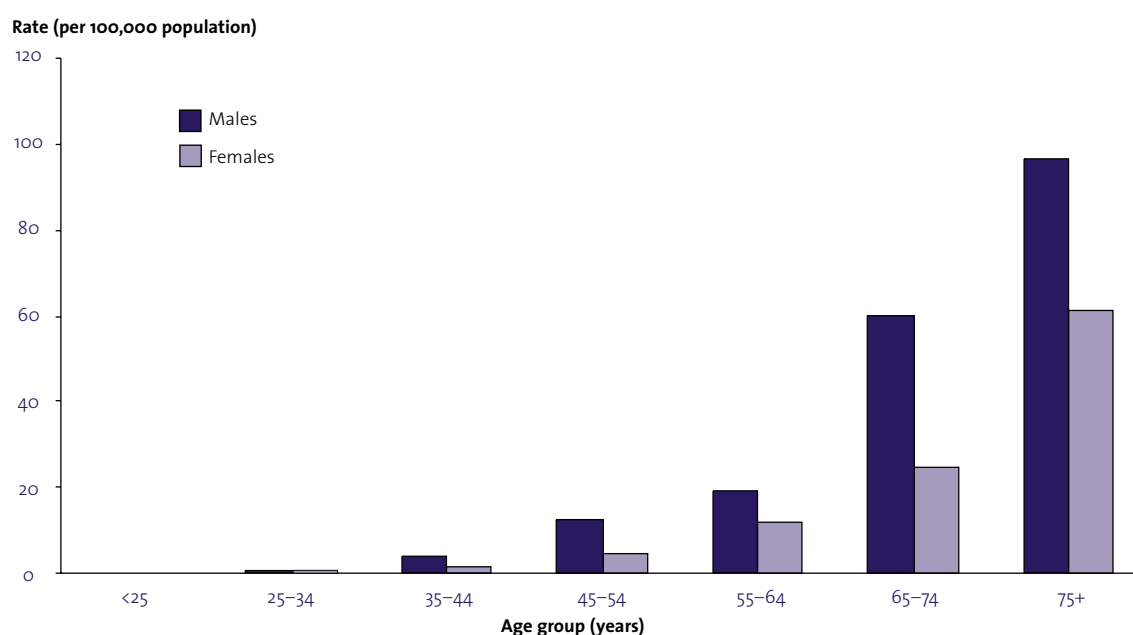
Among patients registered in the National Divisions Diabetes Program (NDDP) Data Collation Project during 1999–00, 24.9% were identified with foot risk (indicated by a history of foot problems and/or presence of peripheral neuropathy, PVD or foot deformity on examination).

### Lower limb amputation

Absence of limbs was reported by 2.1% of respondents with self-reported diabetes during the 1995 National Health Survey. This was more than four times the rate reported among persons without diabetes, despite the higher mortality of amputees with diabetes. The proportion of males with diabetes reporting absence of limbs was 3.3% compared with 0.97% of females with diabetes.

In 2000, the incidence of lower limb amputation among ANDIAB patients was estimated to be 0.8%. Further analysis revealed that 90% of patients undergoing a lower limb amputation in the previous 12 months had a past history of foot ulceration (NADC 2000).

**Figure 4.4:** Hospitalisations for lower limb ulcer as principal and diabetes as additional diagnosis, 1999–00



Source: AIHW National Hospital Morbidity Database.



## Hospitalisations

The majority of foot ulcers are treated in outpatient settings, which limits effective surveillance of the problem (DHAC & AIHW 1999). However, some information on the extent of lower limb ulcer and amputation is available from hospital separation data.

### Lower limb ulcer

In 1999–00 there were 1,859 admissions to hospital where lower limb ulcer was the principal diagnosis and diabetes was an additional diagnosis. Males were almost twice as likely to be hospitalised for lower limb ulcer as the principal and diabetes as an additional diagnosis than females. Hospital use for lower limb ulcer increased with age, with more than 70% of such cases being aged 65 and over in 1999–00 (Figure 4.4).

For people admitted to hospital with lower limb ulcer as principal and diabetes as an additional diagnosis in 1999–00, the average length of stay was 13.4 days. Females tended to have a much longer average length of stay than males, 16.5 days compared with 11.4 days.

### Lower extremity amputation

During 1999–00 a total of 3,404 amputations of lower extremities and/or limbs were performed for a diagnosis of diabetes. Males with diabetes were more than twice as likely to have a lower extremity amputation than females. Hospital use for diabetes-related amputation increases with age. For example, although men and women aged 65 and over represent only 12% of the total population, they accounted for almost 65% of hospitalisations for diabetes-related lower extremity and limb amputations in 1999–00.

Those hospitalised for lower extremity amputation tended to stay considerably longer than those hospitalised for other diabetes-related conditions. The average length of stay in hospital for diabetes-related lower extremity amputation was 27.5 days. Females tended to have a shorter average length of stay than males, 25.4 days compared with 28.4 days.

## Main data sources

2000 Australian National Diabetes Information Audit and Benchmarking (ANDIAB) (National Association of Diabetes Centres).

1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (International Diabetes Institute & Commonwealth Department of Health and Aged Care).

1999–2000 National Divisions Diabetes Program (NDDP) Data Collation Project.

1995 National Health Survey (Australian Bureau of Statistics).

National Hospital Morbidity Database (Australian Institute of Health and Welfare).

National Mortality Database (Australian Institute of Health and Welfare).

## References and further reading

Campbell LV, Graham AR, Kidd RM, Molloy HF, O'Rourke SR & Colagiuri S 2000. The lower limb in people with diabetes. Position statement of the Australian Diabetes Society. *Medical Journal of Australia* 173:369–71.

Carter S, Bonney M, Flack J, Burns J, Powell Davies PG & Harris MF 2000. National Divisions Diabetes Program Data Collation Project. Volume 5: Divisions of General Practice—Diabetes profiles. Quality of care and health outcomes—collated CARDIAB data. Sydney: Centre for General Practice Integration Studies, School of Community Medicine, University of New South Wales.

Colman PG & Beischer AD 2000. Lower-limb amputation and diabetes: the key is prevention. *Medical Journal of Australia* 173:341–342.

DHAC & AIHW (Commonwealth Department of Health and Aged Care & Australian Institute of Health and Welfare) 1999. National Health Priority Areas report: diabetes mellitus 1998. AIHW Cat. No. PHW 10. Canberra: DHAC & AIHW.

Diabetes Australia 2002. Evidence based guidelines. Identification and management of diabetic foot disease. Viewed 12 April 2002, <<http://www.diabetesaustralia.com.au/docs/Foot-Part-6.pdf>>.

NADC (National Association of Diabetes Centres) 2000. ANDIAB 2000. Australian National Diabetes Information Audit & Benchmarking. Canberra: National Association of Diabetes Centres.

Oyibo SO, Dang CN & Boulton AJM 2002. Diagnosis and management of diabetic neuropathy. *Topical Endocrinology* 19:10–13.

Payne CB 2000. Diabetes-related lower-limb amputations in Australia. *Medical Journal of Australia* 173:352–4.

Williams G & Pickup JC 1999. *Handbook of diabetes*. 2nd edn. Oxford: Blackwell Science, 159–64.

## Oral complications

Diabetes can lead to oral complications. Diabetes may manifest initially with oral symptoms other than thirst. For instance, burning tongue, gum bleeding and excessive salivation have been found in undiagnosed people with diabetes and resolved on treatment to improve glycaemic control. Oral complications are very uncommon in westernised societies but are more common in underdeveloped countries or in lower socioeconomic groups, especially where there is poor hygiene and delayed diagnosis of diabetes.

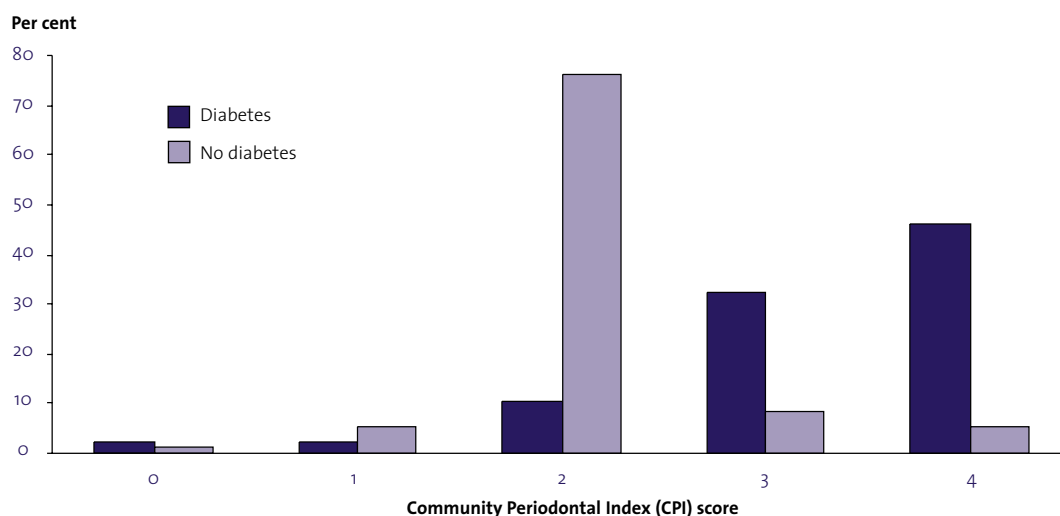
### Periodontal disease

There is growing acceptance that diabetes is associated with increased occurrence, extent and severity of periodontitis (inflammation of the tissues supporting the teeth). The risk is independent of whether the diabetes is Type 1 or Type 2. Some researchers point to a two-way connection between diabetes and periodontal disease, proposing that not only are patients with diabetes more prone to periodontal disease, but the presence of periodontal disease affects control of blood glucose.

International studies have shown that people with Type 2 diabetes are about three times more likely to have destructive periodontal disease than those without diabetes. Among Indigenous Australians in remote communities, having diabetes is significantly associated with a maximum score in the Community Periodontal Index (CPI) (Figure 4.5). CPI is a measure of periodontal disease suggested for use by the World Health Organization that combines three indicators of periodontal health: gum bleeding, calcium deposits on teeth and depth of periodontal pockets, with a score of 4 indicating worst health.

Diabetes can affect the tissues supporting the teeth (periodontium) and the treatment of periodontal diseases. Patients with long-term poor control of diabetes have increased extent and severity of periodontal disease, whereas those who maintain good metabolic control have minimal periodontal problems. Integrated medical and dental management of these conditions is essential for the general health and quality of life of patients. Treatment of periodontal infections with systemic antibiotics can contribute to the control of diabetes.

**Figure 4.5:** Community Periodontal Index score by diabetes status in remote Indigenous Australians, 2000



Source: Dental Statistics Data Collection.

## Tooth loss

Indigenous Australians with diabetes in remote areas of Australia have significantly more missing teeth than those without diabetes, indicating that periodontal disease associated with diabetes may have contributed to tooth loss (Figure 4.6). Missing teeth in older Indigenous Australians are also associated with high rates of diabetes and advanced periodontal disease.

## Other oral problems

Caries (tooth decay) in the crowns of teeth appear to be more frequent in adults with poor control of insulin-dependent diabetes. Oral infections other than dental caries and periodontal disease are often more severe in people with diabetes. Examples of these are life-threatening deep neck infections and fatal ulcers of the palate.

## Risk factors

Poor oral hygiene, poor control of blood glucose levels, smoking and inadequate nutrition increase the risk of oral complications in people with diabetes.

## Main data source

Dental Statistics Data Collection (Australian Institute of Health and Welfare).

## References and further reading

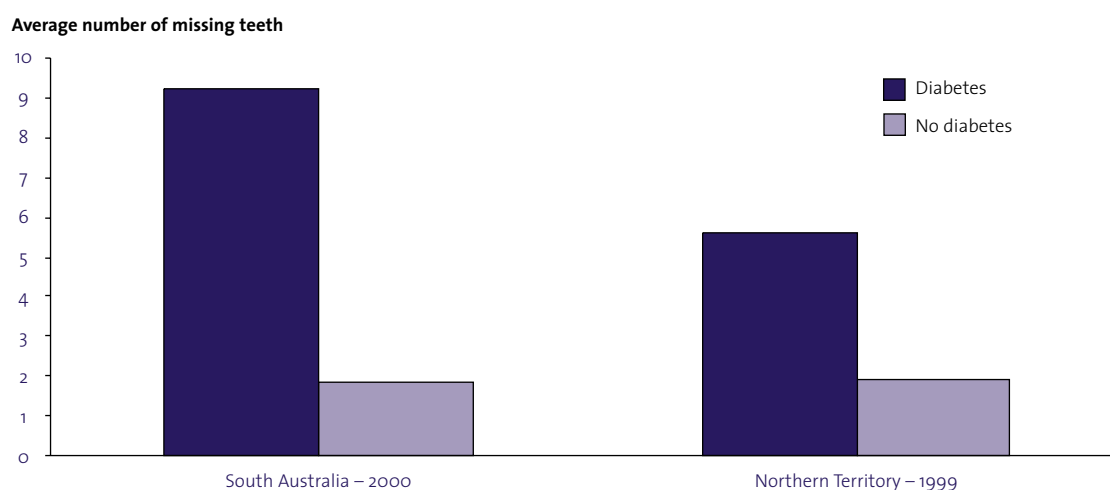
Fenesy KE 1998. Periodontal disease: an overview for physicians. *Mount Sinai Journal of Medicine* 65 (5–6):362–9.

National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health 1995. *Diabetes in America*. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.

USDHHS (United States Department of Health and Human Services) 2000. *Oral health in America: a report of the Surgeon General*. Rockville, MD: National Institute of Dental and Craniofacial Research, National Institutes of Health, USDHHS.

WHO (World Health Organization) 1997. *Oral health surveys—basic methods*. Geneva: WHO.

**Figure 4.6:** Missing teeth by diabetes status in remote Indigenous Australians, 1999 and 2000



Source: *Dental Statistics Data Collection*.

## Complications in pregnancy

Two forms of maternal diabetes may occur during pregnancy: pre-existing diabetes and gestational diabetes. Pre-existing diabetes represents about 10% of cases of maternal diabetes.

### Effects of maternal diabetes

In women with pre-existing diabetes, glycaemic control worsens during pregnancy and insulin requirements increase in Type 1 diabetes. This is because the pregnancy hormones induce insulin resistance. Maternal diabetes affects the foetus and newborn as well as the mother.

For the mother with diabetes, pregnancy can worsen kidney function in those with established nephropathy (kidney disease). Increased protein in the urine and pregnancy-induced hypertension are three to four times more common in women with pre-existing diabetes than in women without diabetes. Retinopathy may also deteriorate rapidly during gestation. Caesarean delivery is three to four times more frequent in pregnancies involving diabetes.

Pre-existing diabetes can cause major congenital malformations in the foetus, particularly in the first 8 weeks of gestation, when the major organs are forming, as well as spontaneous abortions. Defects include absence of brain, malformations of the spine, skeleton and kidneys, and heart and great blood vessel abnormalities. The malformation rate is related to the degree of hyperglycaemia (about 7–30% in poorly controlled patients) but tight metabolic control before and during early pregnancy can reduce the rate. International studies indicate that spontaneous abortions occur in 7–17% of diabetic pregnancies if diabetes is not well managed. Women with good control do not appear to have increased rates of spontaneous abortions compared with women without diabetes.

The perinatal death rate (stillbirths and newborn deaths within the first week of life) is also increased by 1.5 to 2-fold in pregnancies with pre-existing diabetes compared with those without diabetes, according to international data. The main causes of this are:

- death in the uterus in the third trimester of pregnancy;
- prematurity due to a high incidence of spontaneous premature labour and of elective premature delivery in an attempt to avoid death in the uterus late in the pregnancy;
- low birth weight due to foetal growth retardation in the uterus in some cases where the mother has diabetic nephropathy;
- congenital malformations; and
- birth trauma due to a high incidence of excessively large babies.

International studies show that large babies occur at a rate of 30% in diabetic pregnancies compared with 10% in non-diabetic pregnancies. Data from Queensland indicate that 12.3% of all babies born in 1997 weighed 4,000 g or more, compared with 17.7% when mothers had gestational diabetes and 17.9% when mothers had pre-existing diabetes.

Accelerated foetal growth, leading to large-for-gestational-age babies, is due to increased delivery of glucose and other nutrients from mother to foetus. This stimulates the pancreas in the foetus to produce extra insulin, which promotes abdominal fat deposition, growth of the skeleton and large size organs. Complications for these babies include birth trauma, and jaundice, hypoglycaemia and low levels of calcium in the newborn. Poor glycaemic control also leads to impaired lubrication of the lungs and respiratory distress in the newborn. These babies may also have a long-term greater risk of obesity.

Women with gestational diabetes may have a greater risk of foetal perinatal death and disease, and are themselves at increased risk of developing Type 2 diabetes and perhaps cardiovascular disease later in life. Infants of women who develop gestational diabetes may have newborn hypoglycaemia, jaundice, respiratory distress and birth trauma resulting from being excessively large babies, much the same as those of women with pre-existing diabetes.

### How many Australian women are affected by maternal diabetes?

Although there are no national figures on how many Australian women are affected by maternal diabetes, data from States and Territories indicate that gestational diabetes occurs in 3.0–4.5% of all women giving birth and pre-existing diabetes in 0.4%.

Reliable data on the prevalence of gestational diabetes among Aboriginal and Torres Strait Islander mothers are scarce. There are varying estimates of prevalence

ranging from less than 1% to up to 20%. However, de Courten et al. (1998) suggest that Indigenous women experience a higher risk of gestational diabetes than non-Indigenous women.

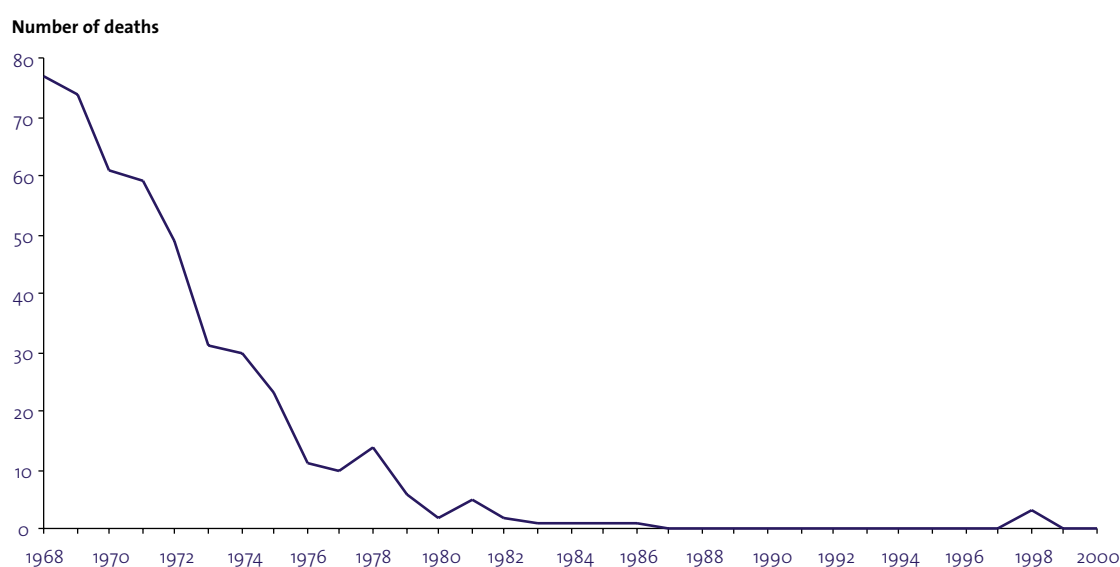
### Hospitalisations

In Australia, in 1999–00 there were 13,901 hospitalisations for gestational diabetes as a principal or additional diagnosis, and 2,330 hospitalisations for pre-existing diabetes in pregnancy as a principal or additional diagnosis.

### Infant deaths

The frequency of foetal disease and mortality in diabetic pregnancies has been dropping over the past few decades, possibly as a result of better medical care and control of diabetes in the mother (Figure 4.7).

**Figure 4.7:** Deaths among infants of a diabetic mother, 1968–2000



Source: AIHW Mortality Database.

## Main data source

States and Territories Perinatal Data Collections.

National Mortality Database (Australian Institute of Health and Welfare).

## References and further reading

de Courten M, Hodge A, Dowse G, King I, Vickery J & Zimmet P 1998. Review of the epidemiology, aetiology, pathogenesis and preventability of diabetes in Aboriginal and Torres Strait Islander populations. Canberra: Commonwealth Department of Health and Family Services.

Edwards CRW, Bouchier IAD, Haslett C & Chilvers ER (eds.) 1998. Davidson's principles and practice of medicine. 17th edn. Edinburgh: Churchill Livingstone.

National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health 1995. Diabetes in America. 2nd edn. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.

Williams G & Pickup JC 1999. Handbook of diabetes. 2nd edn. Oxford: Blackwell Science, 191–6.