5 Prevention and management of chronic kidney disease

Chronic kidney disease (CKD) is a serious and costly, but highly preventable and treatable, disease. Reducing the human and economic burden imposed by CKD relies heavily on preventing the onset and progression of the disease.

The Strategic Framework for Preventing Chronic Disease (NPHP 2001) takes a continuum of care approach to disease prevention. Intervention at various stages across the course of disease can prevent or delay progression to the next stage. The strategy identifies three broad levels of prevention: primary, secondary and tertiary.

Primary prevention measures aim to eliminate or reduce exposure to factors which cause ill health or disease. For CKD this involves strategies to reduce the incidence and prevalence of risk factors such as diabetes and high blood pressure, in order to reduce the number of people at risk of developing CKD. Secondary prevention consists of measures for early detection of disease to allow prompt and effective intervention to prevent the disease becoming established. Early detection and effective intervention in the early stages of kidney damage are essential to prevent or delay the development of CKD. Tertiary prevention strategies are focused on management of established disease to prevent progression and reduce or delay long-term complications, impairment or disability. Management of CKD aims to prevent or delay further kidney damage and loss of kidney function, and hence reduce the incidence and prevalence of ESKD and other complications. In those who do develop ESKD, good management during kidney replacement therapy not only reduces suffering and death, but also improves quality of life.

In Australia, considerable efforts have been made by government, health professionals and consumer groups to improve prevention and management of CKD. For example, successful use of public health strategies and legislative changes in Australia in 1979 have been credited with the significant decrease in incidence of treated end-stage kidney disease caused by analgesic nephropathy in the last decade. Also, improvements in management and techniques of kidney replacement therapy have led to longer survival of patients with end-stage kidney disease.

In 2000, Kidney Health Australia and the Australian and New Zealand Society of Nephrology developed the Caring for Australians with Renal Impairment (CARI) Guidelines to guide and improve the management of kidney disease in Australia (Box 5.1). The guidelines have been well accepted and have extensively influenced practice throughout Australia and New Zealand.

Although there have been successes in the past and consistent effort in recent years, the burden imposed by CKD in Australia has the potential to increase. This chapter provides an overview of the current situation with regard to the prevention and management of CKD in Australia. Where available, CARI guidelines are used against available information to illustrate the status of relevant practices. This overview provides information to inform future policies, strategies and interventions to help reduce the burden of CKD.
Risk reduction

There are some difficulties in preventing the population from developing CKD through reducing exposure to risk factors and causes of the disease, as these are not completely understood. However, there is strong evidence that many of the known risk factors that lead to developing CKD are modifiable. In Australia, widespread and consistent efforts have been made to promote healthy lifestyle in recent years. These efforts have led to a significant reduction in some risk factors, such as smoking and high blood pressure. However, not all risk factors have shown an improvement and there is potential for further gains in health through risk factor modification.

Several factors are involved in the reduction in risk of CKD in the community:

- Improving information and awareness of the disease;
- Reducing prevalence of behavioural risk factors;
- Reducing prevalence and improving control of biomedical risk factors; and
- Reducing exposure to external factors which increase risk.

Improving information and awareness of chronic kidney disease in the community

‘One of the primary barriers to reducing the incidence of CKD in Australia is the limited public awareness of the preventable risk factors that contribute to its development and progress’ (KHA 2004). Information regarding CKD risk reduction is available and accessible for most Australians. There are numerous government, non-government, pharmaceutical, and consumer web sites containing kidney disease information, and printed information is also available. In addition, an annual public awareness campaign (Kidney Awareness Week, or ‘Wee Week’) aims to improve awareness of kidney disease in the community and among health professionals.

However, national polling undertaken for the Pfizer Australia Health Report indicated that almost half of all respondents were not aware they could reduce their risk of developing kidney disease, and had little or no knowledge of how to keep their kidneys healthy (Pfizer Australia & Kidney Health Australia 2004). A recent stakeholder survey also found that less than 15% of respondents were aware that simple actions such as not smoking, following a healthy diet and having regular blood pressure checks could have benefits for the health of their kidneys (TNS Consultants 2004).

Reducing prevalence of behavioural risk factors

Recent progress in risk reduction with regard to behavioural factors is mixed. There has been a substantial reduction in the prevalence of tobacco smoking in the general population in Australia over the last 20 years, with the proportion of daily smokers aged 14 years and over nearly halving between 1985 and 2004. However, the proportion of people undertaking insufficient physical activity for health benefits has increased in recent years, and evidence suggests that the average Australian’s diet is not as healthy as it could be. These two factors have played important roles in increasing the prevalence of obesity and diabetes in Australia, thereby indirectly contributing to the prevalence of CKD.
Reducing prevalence and improving control of biomedical risk factors

Of the biomedical risk factors for CKD, two of the most common and amenable to management are high blood pressure (hypertension) and diabetes. As progressive CKD can develop as complications of these conditions, good management is essential in reducing risk. In people with established CKD, high blood pressure and poorly controlled blood glucose levels (among those who also have diabetes) may promote kidney function reduction and increase the risk of developing complications such as cardiovascular disease. These factors can therefore also be incorporated into tertiary prevention efforts.

Box 5.1: Caring for Australians with Renal Impairment (CARI) Guidelines

‘Caring for Australians with Renal Impairment (CARI) Guidelines is a national evidence-based project that commenced in 1999 with funding from the pharmaceutical industry. The two bodies responsible for the CARI Guidelines are the Council of the Australian and New Zealand Society of Nephrology (ANZSN) and Kidney Health Australia (KHA).

The aim of the CARI Guidelines is to improve the health care and outcomes of paediatric and adult renal patients by helping clinicians and nurses to adhere to evidence-based medical practice as often as possible. It is anticipated that the guidelines will serve as both a valuable educational resource and a means of enhancing the quality, appropriateness, consistency and cost-effectiveness of renal health care.’ (KHA 2005).

Existing guidelines cover the areas of chronic kidney disease, dialysis and transplantation. Each guideline comprises various subtopics and their recommendations. The first version of the guidelines was completed in 2000. It is intended that updating and revision of all guidelines will occur every 3 years, to ensure that the guideline contents are kept relatively up to date.


Blood pressure control

The prevalence of high blood pressure among people aged 25–64 years has reduced in Australia in recent years. However, blood pressure control among those who have high blood pressure is not always ideal. According to the 1999–00 AusDiab study, among 3.6 million (28.6%) Australians aged 25 years and over with high blood pressure, 53.1% were untreated. Only 18.6% had blood pressure controlled in the normal range, and the remaining 81.4% had elevations in blood pressure to varied degrees (Table 5.1) (Briganti et al. 2003).

<table>
<thead>
<tr>
<th>Elevation in blood pressure</th>
<th>Untreated hypertension</th>
<th>Treated hypertension</th>
<th>Per cent of people with hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0%</td>
<td>18.6%</td>
<td>18.6%</td>
</tr>
<tr>
<td>Mild</td>
<td>41.6%</td>
<td>16.7%</td>
<td>58.3%</td>
</tr>
<tr>
<td>Moderate</td>
<td>9.2%</td>
<td>8.3%</td>
<td>17.5%</td>
</tr>
<tr>
<td>Severe</td>
<td>2.3%</td>
<td>3.3%</td>
<td>5.6%</td>
</tr>
<tr>
<td>Total</td>
<td>53.1%</td>
<td>46.9%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Source: Briganti et al. 2003.
For those people who already have CKD, a number of studies have shown that blood pressure lowering is associated with substantial lessening in kidney function decline. CARI Guidelines currently recommend the following targets for blood pressure (ANZSN & KHA 2001):

- ≤130/85 mmHg if proteinuria is <0.25 g/24 hours
- ≤130/80 mmHg if proteinuria is 0.25–1 g/24 hours
- ≤125/75 mmHg if proteinuria is >1 g/24 hours.

Information about blood pressure control and hypertension in people with CKD is not currently available in Australia.

Blood glucose control

There is evidence that tight control of blood glucose in people with diabetes can prevent or delay onset and slow the progression of kidney damage (DCCT 1993). The percentage of glycosylated haemoglobin in the blood (HbA1c level) is a measure of how well blood glucose has been controlled over the past three months. CARI Guidelines recommend that HbA1c be maintained at less than 7.5% for primary prevention of nephropathy and prevention of progression from microalbuminuria to overt nephropathy (ANZSN & KHA 2004a).

In the 1999–00 AusDiab study, the average HbA1c level among people with diabetes was 6.7%, with 78% of people meeting the CARI Guidelines HbA1c level of less than 7.5%. People with Type 1 diabetes were much less likely to have an HbA1c less than 7.5% than those with Type 2 diabetes (26% and 80% achieving this level, respectively).

Reducing exposure to external risks

In some cases, the toxic effects of certain chemicals or drugs may cause damage to the kidneys. These include certain antibiotics, heroin, and a number of drugs used in chemotherapy. Overuse of analgesics can also cause kidney damage.

The case of analgesic nephropathy is an example of successful disease control in Australia. Analgesic nephropathy is a type of toxic injury to the kidneys which may result from long-term daily use of analgesics. It was highly prevalent among the treated ESKD population in the past, accounting for nearly 20% of all new cases of treated ESKD in Australia in the early 1970s. From 1970 to 1983, the incidence rate of treated ESKD caused by analgesic nephropathy increased from 4 to 8 per million population. In 1979, legislative restriction on the advertising and over-the-counter sale of compound analgesics took place in Australia. This policy change has had a significant impact on the incidence of treated ESKD due to analgesic nephropathy (Briganti et al. 2000). The incidence was stable from 1984 to 1993, and then consistently decreased over the following decade to reach 4 per million population in 2003. Analgesic nephropathy is now one of the less commonly identified causes of treated ESKD in Australia.

Early detection

There is substantial evidence that early diagnosis and intervention provides the greatest opportunity for kidney function preservation. While there is currently no evidence to support mass screening of the general population by urine or blood testing, opportunistic screening is justified for selected high-risk groups (Johnson et al. 2004; Boulware et al. 2003).
CARI Guidelines recommend targeted screening of individuals at risk of developing kidney disease (ANZSN & KHA 2004b), including:

- patients with vascular disease, diabetes or hypertension;
- immediate relatives of patients with diabetes, hypertension or renal disease;
- Aboriginal and Torres Strait Islander Australians; and
- patients complaining of prostatic symptoms.

Methods of screening include checking blood pressure, performing urinalysis by microalbuminuria dipstick (or albumin/creatinine ratio) in people with diabetes and proteinuria dipstick in people without diabetes, and calculation of GFR (using serum creatinine).

There is currently no nationally coordinated, standardised screening program in Australia, and information on early detection among these high-risk groups is limited. Nevertheless, the results of the BEACH general practitioner survey provide some information on testing of kidney function in patients who were treated for vascular disease, diabetes and high blood pressure by GPs.

In 2002–03, high blood pressure was the most frequently managed problem in general practice, accounting for 9% of problems managed by GPs. GPs rarely referred patients with high blood pressure to other health professionals or services, suggesting that high blood pressure is mostly handled in general practice. Of these consultations for high blood pressure, around 13% had a pathology test ordered which could be used to detect kidney damage (see Chapter 2).

About 3% of all problems managed by GPs in 2002–03 related to diabetes. Tests that could be used to detect kidney damage were ordered in around 16% of these cases.

Besides hypertension and diabetes, other high-risk group individuals are patients with vascular diseases, including atherosclerosis, peripheral vascular disease, atrial fibrillation or flutter, cerebrovascular disease, coronary heart disease, heart failure and high blood cholesterol or lipid disorders. These diseases accounted for about 4% of problems managed by GPs in 2002–03. Tests that could be used to detect kidney damage were ordered in about 9% of these cases.

Management of early chronic kidney disease

Treatment of CKD may include use of angiotensin-converting enzyme (ACE) inhibitors, tight control of blood pressure and blood glucose, treatment of comorbidities, and patient education. The aims of management in patients with early stage CKD are delaying the progression of the disease, preventing onset of complications, providing early treatment for its comorbidities and preparing patients who are reaching the end stage for kidney replacement therapy.

In Australia, there is evidence that the aims of management of early CKD can be achieved in a high-risk population. In late 1995, a kidney and cardiovascular treatment program was introduced into the Tiwi community, which has a three- to five-fold increase in death rates and a recent annual incidence of end-stage kidney disease of 2,760 per million. People with confirmed hypertension, diabetes with microalbuminuria, or overt albuminuria received treatment including blood-pressure-lowering medication, attempts to control glucose and lipid levels, and health education. After nearly three and half years follow-up, there was a
50% reduction in end-stage kidney disease and natural death among the patients who had received treatment (Hoy et al. 2003).

It is not known if this treatment regime would benefit other patients who have CKD in Australia, as there are currently no national programs for CKD prevention. It is also impossible to evaluate outcomes of the treatment of CKD at early stages due to the absence of relevant information.

Management of pre-dialysis

Patients are classified as being at the pre-dialysis stage when they have severe CKD and are expected to progress to require dialysis or transplantation in the next 6–18 months (KHA 2004). Key patient needs at this stage include:

- appropriate selection of the preferred mode of therapy, and adequate preparation;
- timely initiation of treatment;
- availability of counselling, education, and rehabilitation throughout the process; and
- appropriate management of comorbid conditions (such as anaemia, high blood pressure and bone disease) and risk factors (such as blood lipids and nutrition) (KHA 2004).

Timely referral of patients to a specialist (nephrologist), pre-dialysis education and accessing a multidisciplinary health professional team are essential approaches to address these needs.

Timing of referral to nephrologist

The CARI Guidelines recommend that approximately one year is required to optimally prepare a patient and their family/carers for kidney replacement therapy (ANZSN & KHA 2004c). In Australia, referral to a nephrologist less than 90 days prior to the date of initiation of dialysis is regarded as a late referral. Late referral not only limits the patient’s opportunity for selection of dialysis modality and for timely placement of an appropriate dialysis access, but also results in poorer patient survival and reduced likelihood of kidney transplant (Cass et al. 2003; Roderick et al. 2002).

The frequency of late referral in Australia has been between 25–30% in recent years. The reasons for such a high proportion of late referrals are not well studied. Several possible factors may influence the current situation, including:

- structural issues in the health system, such as distance from a dialysis facility and queues in outpatient clinics;
- lack of knowledge in physicians, and poor communication between GPs and nephrologists; and
- unavoidable factors, such as asymptomatic ESKD presenting at an advanced stage, or delay or refusal of the patient to seek medical care (KHA 2004).

Pre-dialysis education and training

Although various kidney units have developed pre-dialysis education programs, there are currently no national, standardised programs of pre-dialysis care and education available in Australia.
Results from a recent Australian survey suggested that patients who had been recently diagnosed as pre-dialysis had a low level of understanding as to what dialysis involved. They lacked knowledge of when they might expect to begin dialysis, how dialysis would affect them physically and how much of an impact it would have on their daily life (TNS 2004).

**Availability of multidisciplinary health professional teams**

There is evidence to suggest that patients who receive care from a multidisciplinary team (including nephrologists, dieticians, renal nurses, and social workers) have better outcomes from dialysis and survive longer (Curtis et al. 2005; Mendelssohn 2005). In Australia, there is some evidence to suggest that patients who obtain dialysis from a hospital unit have greater opportunities to access a multidisciplinary care team, compared with patients who attend satellite units or dialyse at home (Healthcare Management Advisors Pty Ltd 2004).

**Management of end-stage kidney disease**

When CKD reaches end-stage, kidney replacement therapy is the only currently available treatment to prolong life.

The number of people receiving kidney replacement therapy has more than tripled in Australia over the last 20 years. A greater number of older people are receiving treatment for ESKD, and the proportion of patients with comorbidities has increased. This has led to greater complexity in providing and managing kidney replacement therapy. However, despite these difficulties, advances in technology and better management have led to improvements in outcomes.

A profile of the general pattern of kidney replacement therapy in Australia is presented in Chapter 2. This section provides additional information on management of kidney replacement therapy, including adequacy of dialysis, transplant-related issues, and patient survival.

**Adequacy of dialysis**

**Adequacy of haemodialysis**

The outcome of haemodialysis may be influenced by many factors, particularly the urea reduction ratio (URR), nutritional status and vascular and infective comorbidities connected with dialysis.

Currently, no information has been collected nationally regarding the nutritional status and vascular and infective events during haemodialysis. However, data on some indicators of the technical aspects of haemodialysis, such as the URR, the dose of dialysis, vascular access, haemoglobin (Hb) concentration, calcium and phosphate products, are collected by the ANZDATA Registry. These indicators showed that up to March of 2004, most haemodialysis-dependent patients have received adequate treatment, and the adequacy of dialysis has improved over the last five years (Excell et al. 2005d; Kerr 2000). These data and corresponding CARI Guidelines (ANZSN & KHA 2000) are presented in Appendix 2.
Adequacy of peritoneal dialysis

Assessment of peritoneal dialysis adequacy involves various measurements including clinical assessment of wellbeing, physical measurements, small solute clearance, fluid removal and the impact of treatment on the individual’s life. The results of such comprehensive measurements are not available nationally.

Peritonitis (inflammation of the peritoneum, the lining of the abdomen) is a major complication of peritoneal dialysis. Up to 31 March 2004, median peritonitis-free survival of peritoneal dialysis-dependent patients was 19.2 months, with 30% of patients completely free of peritonitis at 3 years (Excell et al. 2005c). This was an improvement from 1999, when the median peritonitis-free survival of peritoneal dialysis-dependent patients was 16.6 months, with 23% of patients completely free of peritonitis at 3 years (Collins 2000).

Transplant issues

Kidney donor sources

There are two sources of donor kidneys: living and deceased people. Kidneys from living donors can come from relatives (parents, sisters, brothers and adult children), or unrelated persons (usually the spouse, close friends or distant relatives). Kidneys can also come from a person who has been declared brain dead while their vital organs are being maintained on a breathing machine if they or their next of kin had agreed to donate their organs after death.

In 2003, 218 (40%) of 543 transplanted kidneys were from living donors, and 325 (60%) were from deceased donors. The number of transplanted kidneys from living donors has increased by 29% over the last 5 years (Excell et al. 2005b). This increase is largely attributed to the increasing number of unrelated living donors. This phenomenon can be traced to the development of more powerful anti-rejection drugs, which have reduced the importance of tissue matching in donor selection, thereby widening the possible pool of donors (International Association of Living Organ Donors 2002). In contrast, the increase in the number of kidneys from deceased donors appears relatively low, only 14% over the same period. The slow growth in deceased donor kidneys may be partly attributed to reduction in and increased survival following traffic accidents, and improvement of intensive care in the hospital.

Pre-emptive transplant

Kidney transplant prior to the initiation of dialysis is known as pre-emptive transplantation. Pre-emptive transplantation not only avoids pre-transplant dialysis and reduces the risk of morbidity and cost associated with dialysis, but also can increase the chance of survival.

In 2003, about 25% (50 cases) of all live donor transplantations were pre-emptive, accounting for 9% of all kidney transplantations (Excell et al. 2005b).

Waiting list for transplant

At 31 March 2004, 1,591 dialysis-dependent patients were waiting for a kidney transplant. This represents 21% of the total dialysis-dependent population. The majority of patients (94%) on the waiting list were under 65 years old, and 83% were waiting for their first transplant. The average waiting time for a kidney transplant was about 4 years.
Among the states and territories, the Australian Capital Territory had the longest waiting list, where 32% of dialysis-dependent patients were waiting for a kidney transplant. This was followed by New South Wales (28%), Victoria (21%), Western Australia (20%), Tasmania (18%), South Australia (14%), the Northern Territory (12%) and Queensland (9%) (Excell et al. 2005a). The reasons for the differences in the number of patients on the waiting list between the states are not clear, but could relate to different overall numbers of end-stage kidney disease patients, different age structures or suitability for transplant among these patients, variations in the number of deceased and living kidney donors, or other factors.

**Survival**

**People receiving dialysis**

The desired outcome of dialysis is to reduce morbidity and mortality and improve quality of life of people with ESKD. At present, there is no information available regarding morbidity of dialysis patients. According to the ANZDATA Registry, the survival of dialysis patients in Australia has improved across most age groups over the past 20 years in Australia. However, long-term survival rates for those aged 65 years and over are still low compared with people at younger ages. The survival rates at 1, 5 and 10 years after commencement of dialysis are shown in Table 5.2.

**People with a kidney transplant**

Quality of life and survival of kidney transplant patients are generally better than dialysis-dependent patients. The survival of kidney transplant patients has also improved over the past 20 years in Australia. The survival rates at 1, 5 and 10 years after transplantation are shown in Table 5.3.
Table 5.2: Trends in survival at 1, 5 and 10 years after commencement of dialysis, by age group and 5-year cohort

<table>
<thead>
<tr>
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<tbody>
<tr>
<td></td>
<td>Patients at risk</td>
<td>Survival rate (std error)</td>
<td>Patients at risk</td>
<td>Survival rate (std error)</td>
<td>Patients at risk</td>
</tr>
<tr>
<td>0–24 years</td>
<td>1</td>
<td>178</td>
<td>96 (± 0.01)</td>
<td>205</td>
<td>98 (± 0.01)</td>
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<tr>
<td></td>
<td>5</td>
<td>25</td>
<td>88 (± 0.03)</td>
<td>33</td>
<td>81 (± 0.05)</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>8</td>
<td>64 (± 0.11)</td>
<td>9</td>
<td>76 (± 0.07)</td>
</tr>
<tr>
<td>25–44 years</td>
<td>1</td>
<td>527</td>
<td>95 (± 0.01)</td>
<td>630</td>
<td>95 (± 0.01)</td>
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<td></td>
<td>5</td>
<td>106</td>
<td>70 (± 0.03)</td>
<td>139</td>
<td>68 (± 0.03)</td>
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<td>10</td>
<td>25</td>
<td>42 (± 0.05)</td>
<td>47</td>
<td>46 (± 0.04)</td>
</tr>
<tr>
<td>45–64 years</td>
<td>1</td>
<td>1,083</td>
<td>87 (± 0.01)</td>
<td>1,430</td>
<td>89 (± 0.01)</td>
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<td></td>
<td>5</td>
<td>321</td>
<td>42 (± 0.02)</td>
<td>362</td>
<td>41 (± 0.01)</td>
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<td></td>
<td>10</td>
<td>87</td>
<td>14 (± 0.01)</td>
<td>78</td>
<td>12 (± 0.01)</td>
</tr>
<tr>
<td>65–74 years</td>
<td>1</td>
<td>230</td>
<td>75 (± 0.02)</td>
<td>609</td>
<td>81 (± 0.01)</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>86</td>
<td>26 (± 0.03)</td>
<td>185</td>
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<td>10</td>
<td>9</td>
<td>3 (± 0.01)</td>
<td>27</td>
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<td>75 years and over</td>
<td>1</td>
<td>16</td>
<td>68 (± 0.10)</td>
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<td>76 (± 0.05)</td>
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<td>7</td>
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<td></td>
<td>10</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>1 (± 0.01)</td>
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</table>

n.y.a. Not yet available.

Source: ANZDATA Registry.
Table 5.3: Trends in survival at 1, 5 and 10 years after kidney transplant, by age at transplant and 5-year cohort

<table>
<thead>
<tr>
<th></th>
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<tr>
<td></td>
<td>Patients at risk</td>
<td>Patients at risk</td>
<td>Survival rate (std error)</td>
<td>Patients at risk</td>
<td>Survival rate (std error)</td>
<td>Patients at risk</td>
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<tr>
<td></td>
<td>(Number) (Per cent)</td>
<td>(Number)</td>
<td>(Per cent)</td>
<td>(Number)</td>
<td>(Per cent)</td>
<td>(Number)</td>
</tr>
<tr>
<td>0–24 years</td>
<td>1</td>
<td>320 (97 (±1.0))</td>
<td>328 (98 (±0.8))</td>
<td>324 (97 (±1.0))</td>
<td>320 (99 (±0.6))</td>
<td>193 (95 (±1.7))</td>
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<tr>
<td></td>
<td>5</td>
<td>311 (91 (±1.6))</td>
<td>320 (92 (±1.5))</td>
<td>314 (95 (±1.2))</td>
<td>313 (94 (±1.5))</td>
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<td>291 (84 (±2.1))</td>
<td>299 (87 (±1.9))</td>
<td>308 (90 (±1.8))</td>
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<tr>
<td>25–44 years</td>
<td>1</td>
<td>656 (94 (±1.0))</td>
<td>734 (96 (±0.8))</td>
<td>738 (97 (±0.6))</td>
<td>887 (98 (±0.5))</td>
<td>590 (99 (±0.3))</td>
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<td>5</td>
<td>613 (82 (±1.5))</td>
<td>702 (88 (±1.2))</td>
<td>715 (91 (±1.1))</td>
<td>860 (94 (±0.9))</td>
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<td>539 (70 (±1.8))</td>
<td>643 (78 (±1.5))</td>
<td>668 (79 (±1.6))</td>
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<td>n.y.a.</td>
</tr>
<tr>
<td>45–64 years</td>
<td>1</td>
<td>601 (89 (±1.3))</td>
<td>795 (88 (±1.1))</td>
<td>807 (93 (±0.9))</td>
<td>925 (94 (±0.8))</td>
<td>638 (95 (±0.9))</td>
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<td>537 (67 (±1.9))</td>
<td>703 (75 (±1.5))</td>
<td>750 (80 (±1.4))</td>
<td>870 (84 (±1.3))</td>
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<td>400 (43 (±2.0))</td>
<td>597 (52 (±1.8))</td>
<td>642 (58 (±1.8))</td>
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<td>65 years and over</td>
<td>1</td>
<td>2 (50 (±35.4))</td>
<td>30 (87 (±6.2))</td>
<td>59 (88 (±4.2))</td>
<td>50 (92 (±3.8))</td>
<td>76 (94 (±3.0))</td>
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<td></td>
<td>5</td>
<td>1 (0.0)</td>
<td>26 (73 (±8.1))</td>
<td>52 (71 (±5.9))</td>
<td>46 (73 (±6.7))</td>
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<tr>
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<td>. .</td>
<td>22 (37 (±8.8))</td>
<td>42 (36 (±6.6))</td>
<td>n.y.a.</td>
<td>n.y.a.</td>
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Not applicable.

n.y.a. Not yet available.

Source: ANZDATA Registry.
References


DCCT (Diabetes Control and Complications Trial Research Group) 1993. The effect of intensive treatment of diabetes on the development and progression of long-term


