

part 2

Diabetic Retinopathy

1 | Introduction

Diabetes is an important cause of morbidity among all Australians, but it also poses some problems that are specific to Aboriginal and Torres Strait Islander communities.

In 1997 the National Health and Medical Research Council (NHMRC) published clinical practice guidelines for the management of diabetic retinopathy. Part of that document contained information relevant to Indigenous communities. Using as references the NHMRC's work, other work published since 1997, and specific data on Indigenous Australians, the Guidelines presented here describe the central elements of treating and managing diabetic retinopathy in Indigenous communities. The purpose is to encourage 'best practice' by providing information that is relevant to the health care professionals who work in these communities.

This part is divided into two broad sections: Sections 2 and 3 provide background and epidemiological information on diabetic retinopathy; Sections 4 to 6 deal with detection and management of the condition.

2 | Background

2.1 Definitions

2.1.1 Diabetes mellitus

Diabetes mellitus is a condition resulting from impairment of the body's ability to tolerate glucose. It is commonly classified into two types: insulin-dependent diabetes mellitus (IDDM, or type 1 diabetes); and non-insulin dependent diabetes mellitus (NIDDM, or type 2 diabetes). Because the distinction between IDDM (type 1 diabetes) and NIDDM (type 2 diabetes) is not always obvious, the National Health and Medical Research Council used the following definition:

Cases with diabetes onset prior to age 30 and treated with insulin (younger-onset) will be considered to have IDDM, while people with diabetes diagnosed from age 30 (older-onset), and treated with either diet alone, oral therapy or insulin, will be considered to have NIDDM.²⁸

For the purpose of these Guidelines the terms type 1 and type 2 diabetes will be used in favour of the terms IDDM and NIDDM. Type 2 is by far the most common form of diabetes found in Aboriginal and Torres Strait Islander people.²⁹

Both types of diabetes can lead to diabetic retinopathy.

2.1.2 Diabetic retinopathy

The NHMRC defined diabetic retinopathy as the typical retinal microvascular lesions²⁸ that occur in nearly all people having diabetes over a long period.

Among the lesions that can occur are microaneurysms, haemorrhages, hard exudates, cotton-wool spots, intra-retinal microvascular abnormalities, venous beading, new vessels and fibrous tissue. None of these is specific to diabetes, but with diabetic retinopathy there is a characteristic pattern, symmetry and evolution of the lesions.²⁸

The degree and rate of change to the retina in people with diabetes varies. Diabetic retinopathy is one of the most serious complications of diabetes-if the condition is left unmonitored and untreated, progressive damage to the retina leads to decreased visual acuity and ultimately blindness.

2.2 The patient population

The patient population is Aboriginal and Torres Strait Islander people who have diabetes mellitus, particularly those who live in rural and remote parts of Australia.

2.3 The purpose

The primary purpose of these Guidelines for evaluating and managing diabetic retinopathy is to prevent, retard or reverse visual loss, thus maintaining or improving vision-related quality of life.

2.4 The goals

These Guidelines are designed to encourage ‘best practice’ on the part of health care professionals dealing with diabetic retinopathy in Aboriginal and Torres Strait Islander communities. Underlying this seeking of ‘best practice’ is the knowledge that almost all people with diabetes eventually develop diabetic retinopathy, that blindness caused by the condition can be prevented with appropriate screening and treatment, and that regular eye examinations are needed if retinopathy is to be detected early.³⁰

There are thus six goals:

- ▶ to identify all Aboriginal and Torres Strait Islander people who have diabetes;
- ▶ to educate and manage people who have diabetes and in this way retard the development of complications of diabetes such as diabetic retinopathy;
- ▶ to identify Indigenous Australians at risk of blindness by providing regular screening for diabetic retinopathy;
- ▶ to provide laser treatment for patients identified as being at risk of visual loss from diabetic retinopathy;
- ▶ to minimise the negative consequences of treatment in order to maintain or improve vision and thus improve vision-related quality of life;
- ▶ to achieve all of the above in a manner that is sensitive to the needs of and cultural differences among Indigenous Australians.

3 | Epidemiology

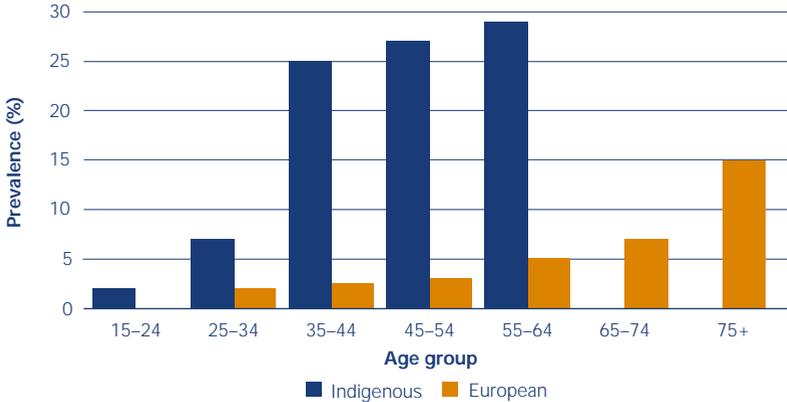
3.1 Diabetes

There is only limited information available on the incidence and prevalence of diabetes among Aboriginal and Torres Strait Islander communities. The information available on diabetic retinopathy is even more limited.

It is estimated that diabetes occurs in 20 to 50 per cent of adults in many Indigenous communities where the diet has changed rapidly from traditional foods to the foods of an affluent, Westernised society.³¹ Prevalence data show that the lowest rate of diabetes occurs in communities that have maintained a traditional diet and lifestyle. Overall, Indigenous communities across Australia have much higher prevalence rates for diabetes, and a much younger average age of onset, than non-Indigenous Australians.³²

Figure 1 shows the contrast by age group.

Figure 1: Diabetes prevalence among Australians of European origin and Indigenous Australians from 10 communities in northern and central Australia, by age, 1983 to 1995^{33,34}



Note: There were insufficient numbers in the Indigenous cohort to include data for subjects aged 65 or more years.

3.2 Diabetic retinopathy

Among Australians aged 20 to 65 years, diabetic retinopathy is now the leading cause of blindness.³⁵ Among the diabetic population, it is estimated that the prevalence of diabetic retinopathy ranges from 8 to 35 per cent.²⁹

As noted, there is very limited data on Indigenous Australians and diabetic retinopathy. A Western Australian study found, however, that 31 per cent of Indigenous people with diabetes had retinopathy, compared with 20 per cent of non-Indigenous people.³⁶ There was a higher proportion of type 2 diabetes among the Indigenous sample and a tendency towards an earlier age of onset. Additionally, diabetic retinopathy within 10 years of onset of diabetes was more common in the Indigenous sample population than in the non-Indigenous sample. Although the study had only 134 participants, it does demonstrate that prevalence rates for diabetic retinopathy among Indigenous Australians are likely to be higher than in the non-Indigenous community.

One study of Indigenous Australians in a rural community showed that 83 per cent of community members with diabetes had type 2 diabetes. Diabetic retinopathy was evident in 14 per cent of those with diabetes. The mean glycohaemoglobin (HbA_{1c}) was 8.5 (SD=2.1) in the diabetic population, compared with 5.4 (SD=0.5) among community members without diabetes.³⁷

In addition, Aboriginal and Torres Strait Islander people often have compounding factors such as renal disease and hypertension.

3.3 Risk factors

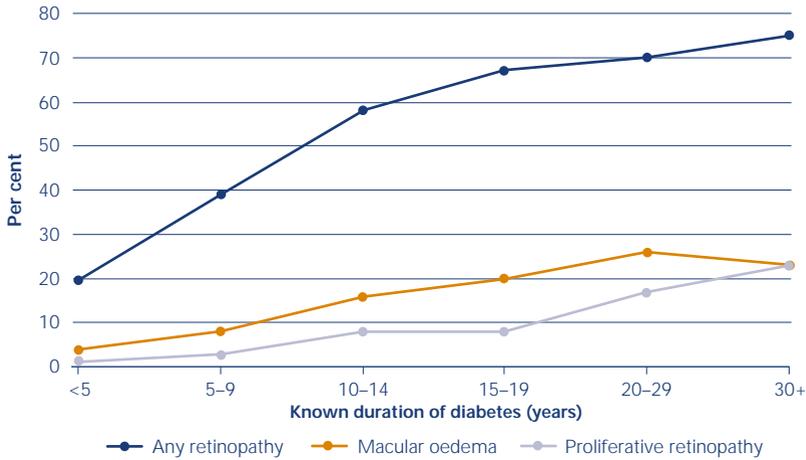
Many risk factors for the development of diabetic retinopathy have been suggested. The two main ones are the duration of diabetes and inadequate glycaemic control. Other important factors are hypertension, elevated serum lipid levels, and pregnancy.

3.3.1 The duration of diabetes

There is a strong association between the duration of diabetes—either type 2 or type 1 diabetes—and the development and severity of diabetic retinopathy. This has been demonstrated by many studies, including one involving 5500 patients seen in Newcastle, New South Wales.³⁸ As noted, type 2 diabetes is by far the most common type of diabetes among Aboriginal and Torres Strait Islander people. The Newcastle study found that, in patients diagnosed with type 2 diabetes, almost 15 per cent had signs of retinopathy at diagnosis, 55 per cent after 10 years, and 70 per cent after 15 or more years. Although the Newcastle study involved mainly non-Indigenous Australians, it is likely that late diagnosis of type 2 diabetes in Indigenous Australians would result in increased severity of diabetic retinopathy at the time of diagnosis.²⁹

Figure 2 provides details of the Newcastle study's findings for the prevalence of any retinopathy, proliferative retinopathy and macular oedema, by known duration of diabetes.

Figure 2: Type 2 diabetes cases: prevalence of any retinopathy, proliferative retinopathy and macular oedema, by known duration of diabetes, in the Newcastle study³⁸



3.3.2 Glycaemic control

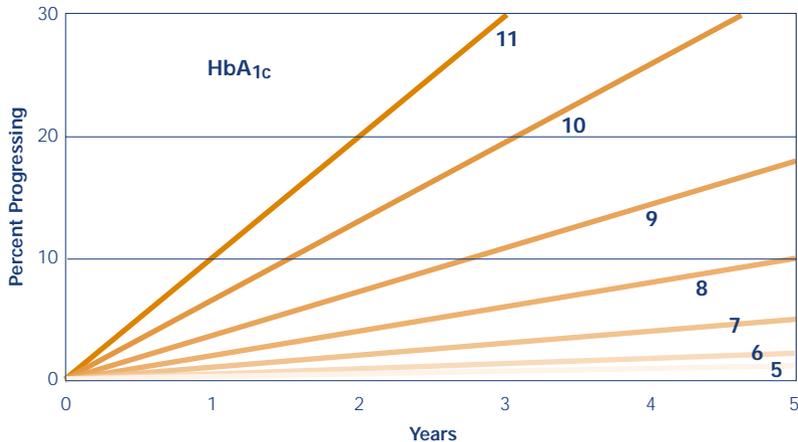
In patients with type 1 diabetes, strict glycaemic control reduces the risk of developing diabetic retinopathy and retards its progression once the disease is established.³⁹ The Diabetes Control and Complications Trial demonstrated that the risk of developing diabetic retinopathy was reduced by 76 per cent if strict glycaemic control was maintained. In patients with early-stage diabetic retinopathy, the risk of progression of the disease was reduced by 54 per cent. There is strong evidence for this risk factor—in different communities and in varying ethnic groups²⁸—so, despite the lack of specific studies, it is likely that Indigenous Australian communities would be similarly affected.

Evidence about the effects of controlling hyperglycaemia in patients with type 2 diabetes was gathered in the UK Prospective Diabetes Study⁴⁰, which involved a randomised controlled clinical trial of blood-glucose control in 3867 patients with newly diagnosed type 2 diabetes. As with type 1 diabetes in the Diabetes Control and Complications Trial, it was found that in patients with type 2 diabetes, strict glycaemic control using either sulphonylureas or insulin reduced the risk of microvascular complications. The need for retinal photocoagulation in the intensively treated group was reduced by 29 per cent compared with those receiving conventional treatment.

Glycaemic control and glycohaemoglobin

The NHMRC Guidelines state that glycohaemoglobin (HbA_{1c}) is considered a better measure of diabetic control than blood glucose because it is less variable and provides a measure of control over the last two or three months.²⁸ The Wisconsin Epidemiologic Study of Diabetic Retinopathy⁴¹ examined the relationship between glycohaemoglobin level at baseline and the incidence and progression of diabetic retinopathy over 10 years. People with glycohaemoglobin levels in the highest quartile at baseline were about three times more likely to have progression of retinopathy than people with levels in the lowest quartile.²⁸ A similar relationship was found in the Diabetes Control and Complications Trial⁴²—see Figure 3.

Figure 3: Absolute risk of sustained retinopathy progression as a function of the updated mean glycohaemoglobin level during the Diabetes Control and Complications Trial and the years of follow-up⁴²



Note: Estimated from Poisson regression models.

3.3.3 Hypertension

The UK Prospective Diabetes Study examined hypertension as an independent risk factor for diabetic retinopathy in patients with type 2 diabetes. Anti-hypertensive treatment with either captopril (an angiotensin-converting enzyme inhibitor) or atenolol (a beta-blocker) was given to 1148 patients with both diabetes and hypertension.⁴³ Unlike previous studies, the UK study found tight blood pressure control produced a clinically significant decrease in the risk of deaths related to diabetes and in the progression of diabetic retinopathy.

3.3.4 Elevated serum lipid levels

The Early Treatment Diabetic Retinopathy Study found that patients with elevated serum lipid levels were twice as likely to have retinal hard exudates as patients with normal cholesterol levels.⁴⁴ Increasing hard-exudate deposition appeared to be independently associated with an increased risk of visual impairment. This association is based on observational data, and as yet there are no completed interventional trials evaluating whether lowering the serum lipid level would reduce the risk of retinal changes in diabetes.

3.3.5 Pregnancy

Pregnancy increases the rate of progression of diabetic retinopathy.⁴⁵ For women with no or minimal non-proliferative diabetic retinopathy (NPDR) before pregnancy, increased NPDR occurred in 12 per cent of cases, most changes regressing postpartum. Similarly, an increased progression was observed among women with NPDR before pregnancy: 47 per cent developed increased NPDR and 5 per cent developed proliferative diabetic retinopathy (PDR); of these two groups, 29 per cent regressed postpartum and 50 per cent required laser treatment. Among the group of women with PDR before pregnancy, 46 per cent progressed during pregnancy.⁴⁶

Factors associated with the risk of diabetic retinopathy⁴⁷

- ▶ Age
- ▶ Age at diagnosis
- ▶ Alcohol use
- ▶ Blood pressure or hypertension
- ▶ Body mass index or obesity
- ▶ Cigarette smoking
- ▶ Contraception and pregnancy
- ▶ Duration of diabetes
- ▶ Ethnicity
- ▶ Glycaemic control
- ▶ Insulin
- ▶ Nutritional factors
- ▶ Serum lipids
- ▶ Socio-economic status

Ways of reducing the risk of developing diabetic retinopathy

- ▶ Tight control of blood glucose
- ▶ Effective treatment of hypertension
- ▶ Lowering serum lipid levels

3.4 The natural history of diabetic retinopathy

Although almost 15 per cent of patients first diagnosed with type 2 diabetes and less than 5 per cent of patients first diagnosed with type 1 diabetes show signs of diabetic retinopathy³⁸, after 20 or more years of having diabetes almost all have some degree of retinopathy.

3.4.1 Definitions

Non-proliferative diabetic retinopathy is the earliest stage of diabetic retinopathy; it is visible using retinal imaging techniques and its main clinical characteristics are:

- ▶ microaneurysms
- ▶ retinal haemorrhages
- ▶ hard exudates
- ▶ cotton-wool spots
- ▶ venous beading
- ▶ intraretinal microvascular abnormalities (IRMA).

Proliferative diabetic retinopathy is characterised by the growth of new vessels—neovascularisation—and is indicative of more advanced diabetic retinopathy. The new vessels tend to be fragile, so they are prone to bleed, causing vitreous haemorrhage. If the new vessels fibrose and contract, this can lead ultimately to retinal detachment.

Macular oedema results from increased permeability of retinal vessels. It is called clinically significant macular oedema if the centre of the macula is involved or threatened and non-clinically significant macular oedema if the centre of the macula is not involved or threatened.³⁰

As the disease progresses, the retinal microvasculature gradually closes, resulting in impaired perfusion and retinal ischaemia. Among the signs of increasing ischaemia are venous abnormalities (various types of beading and loops), IRMA, and more severe and extensive vascular leakage characterised by increasing retinal haemorrhages and exudation.³⁰

Macular oedema, vitreous haemorrhage and retinal detachment all cause impaired vision. Macular oedema is the dominant cause of visual impairment resulting from diabetic retinopathy among Indigenous Australians. Prevention of visual deterioration is the main way of preserving vision since, once vision is lost, only rarely can it be restored.

3.4.2 Grading diabetic retinopathy

In order to monitor the disease's progress and to plan management, non-proliferative and proliferative diabetic retinopathy are classified according to their degree of severity. Non-proliferative diabetic retinopathy is classified as minimal, mild, moderate and severe; proliferative retinopathy is classified as early (non-high risk) or high-risk. Table 1 lists the various stages of diabetic retinopathy and the corresponding clinical features. It should be noted, however, that the data are for Caucasian Americans and may underestimate the rate of progression of diabetic retinopathy among Indigenous Australians.

Table 1: Diabetic retinopathy: classification into stages (Wisconsin level) and predictive value of retinal lesions²⁸

Retinopathy stage	Clinical signs	Rate of progression (%)			
		To PDR		To high-risk stage	
		1 yr	3 yrs	1 yr	5 yrs
Minimal NPDR (level 20) (fig. 1)	Isolated microaneurysms only (m)	Not documented			
Mild NPDR (level 30) (fig. 2)	Microaneurysms (m) + retinal haemorrhages (h)	5	14	1	15
Moderate NPDR (level 40) (fig. 3)	Haemorrhages and microaneurysms (h,m) in at least 1 quadrant + cotton-wool spots (w) or venous beading in 1 quadrant only	12–26	30–48	8–18	25–39
Severe NPDR pre-proliferative (level 50) (fig. 4)	One of the following: <ul style="list-style-type: none"> ▶ Intra-retinal microvascular abnormalities (IRMA) in 1 or more quadrants ▶ venous beading (b) in 2 or more quadrants ▶ haemorrhages/microaneurysms (h,m) in all 4 quadrants 	52	71	15	56

Table 1: (continued)

Retinopathy stage	Clinical signs	Rate of progression (%)			
		To PDR		To high-risk stage	
		1 yr	3 yrs	1 yr	5 yrs
PDR (level 60) (fig. 5)	One or more of the following: <ul style="list-style-type: none"> ▶ peripheral new vessels (NVE) (v) ▶ disc new vessels (NVD) less than 1/3 of disc diameter ▶ vitreous or preretinal haemorrhage with NVE less than 1/2 disc area 			46	75
High-risk PDR (level 70) (fig. 6)	One or more of the following: <ul style="list-style-type: none"> ▶ *NVD\geq1/3 disc area (v) ▶ *NVD with vitreous or preretinal haemorrhage ▶ *NVE\geq 1/2 disc area with vitreous or preretinal haemorrhage 	Severe visual loss (VA <5/200) develops in 25–40% within 2 years			
Macular oedema (fig. 2) Clinically significant macular oedema (fig. 9)	Retinal oedema or thickening within 2 disc diameters of the macular centre Retinal oedema, thickening or hard exudates within 500 μ m of macular centre (1/3 diameter of optic disc) or Retinal oedema or thickening 1 disc diameter or larger, any part of which is within 1 disc diameter of the centre of the macula	Can occur at any stage of diabetic retinopathy. Can occur at any stage of diabetic retinopathy.			

Note: Data are for Caucasian Americans and may underestimate the rate of progression among Indigenous Australian.

Retinopathy Chart

Prepared by the Diabetic Retinopathy Working Party of the NHMRC in conjunction with the Australian Diabetes Society Retinopathy Sub-Committee.

Material derived from NHMRC “*Clinical Practice Guidelines for the Management of Diabetic Retinopathy*”.

Classification	Clinical Signs	Referral Recommendations
Minimal NPDR (fig. 1)	Isolated microaneurysms only (m)	Referral may not be needed. Review annually with dilated fundus exam.
Mild NPDR (fig. 2)	Microaneurysms (m) + retinal haemorrhages (h)	Routine referral to an ophthalmologist. Review with ophthalmologist at least annually.
Moderate NPDR (fig. 3)	Haemorrhages and microaneurysms (h,m) in at least 1 quadrant + cotton-wool spots (w) or venous beading in 1 quadrant only	Refer to an ophthalmologist as soon as possible.
Severe NPDR (fig. 4)	One of the following: <ul style="list-style-type: none"> ▶ Intra-retinal microvascular abnormalities (IRMA) (i) in 1 or more quadrants ▶ venous beading (b) in 2 or more quadrants ▶ haemorrhages/microaneurysms (h,m) in all 4 quadrants 	Refer to an ophthalmologist urgently. PRP may be indicated.
PDR (fig. 5)	One or more of the following: <ul style="list-style-type: none"> ▶ peripheral new vessels (NVE) (v) ▶ disc new vessels (NVD) less than 1/3 of disc diameter ▶ vitreous or preretinal haemorrhage with NVE less than 1/2 disc area 	Refer to an ophthalmologist urgently. PRP may be indicated.
High-risk PDR (fig. 6)	One or more of the following: <ul style="list-style-type: none"> ▶ NVD \geq 1/3 disc area (v) ▶ NVD with vitreous or pre-retinal haemorrhage ▶ NVE \geq 1/2 disc area with vitreous or preretinal haemorrhage 	Refer to an ophthalmologist urgently. PRP may be indicated.
Macular oedema (fig. 2)	Retinal oedema or thickening within 2 disc diameters of the macular centre	Refer to an ophthalmologist as soon as possible.
Clinically significant macular oedema (CSME) (fig. 9)	Retinal oedema, thickening or hard exudates within 500 μ m of macular centre (1/3 diameter of optic disc) or Retinal oedema or thickening one disc diameter or larger size, any part of which is within a disc diameter of the centre of the macula	Refer to an ophthalmologist urgently. macular laser indicated.

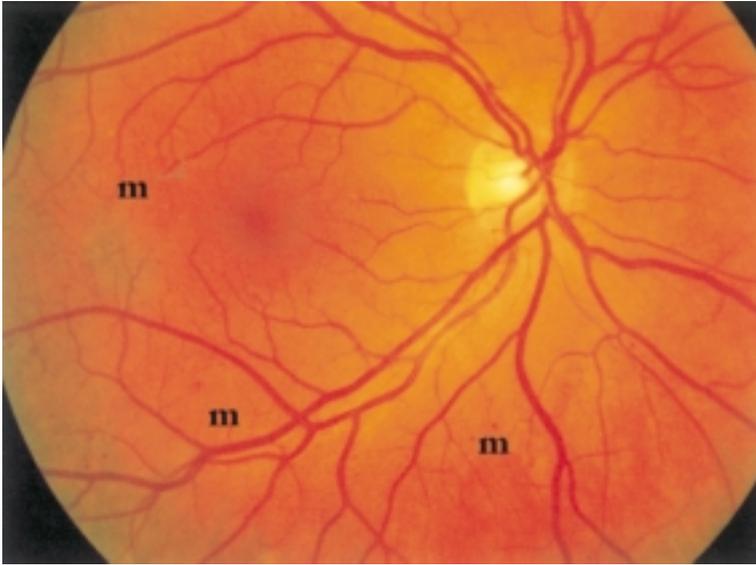


Figure 1: Minimal non-proliferative diabetic retinopathy (NPDR) - few scattered microaneurysms (**m**) only, the remainder of the fundus is normal.

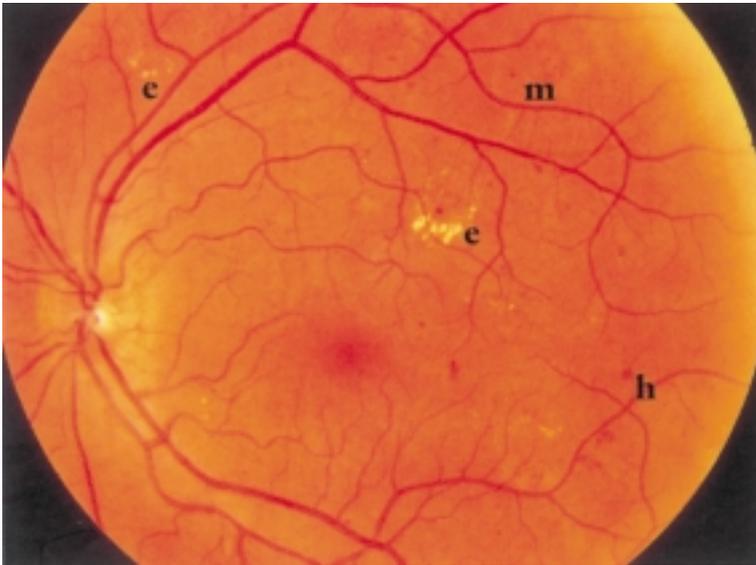


Figure 2: Mild non-proliferative diabetic retinopathy - microaneurysms (**m**) and dot haemorrhages (**h**). Also demonstrates macular oedema with small amount of lipid exudate (**e**) - not clinically significant.

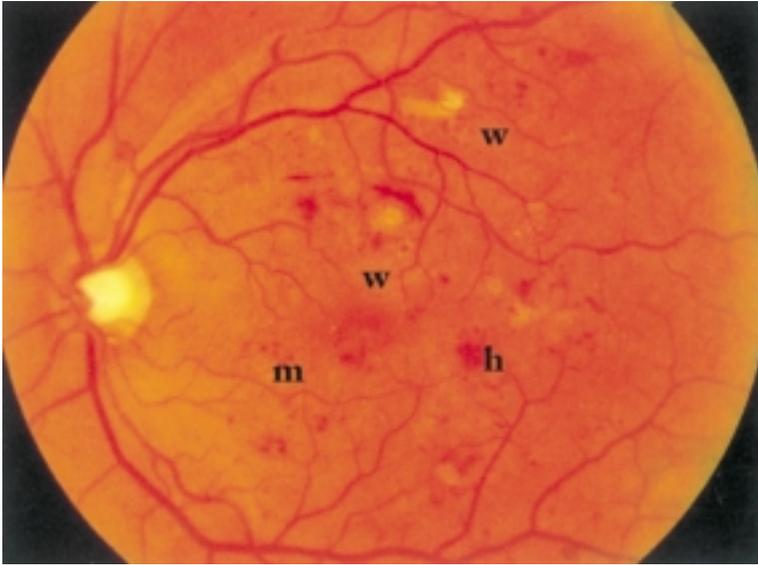


Figure 3: Moderate non-proliferative diabetic retinopathy - cotton wool spots (**w**), more retinal haemorrhages (**h**) and microaneurysms (**m**).

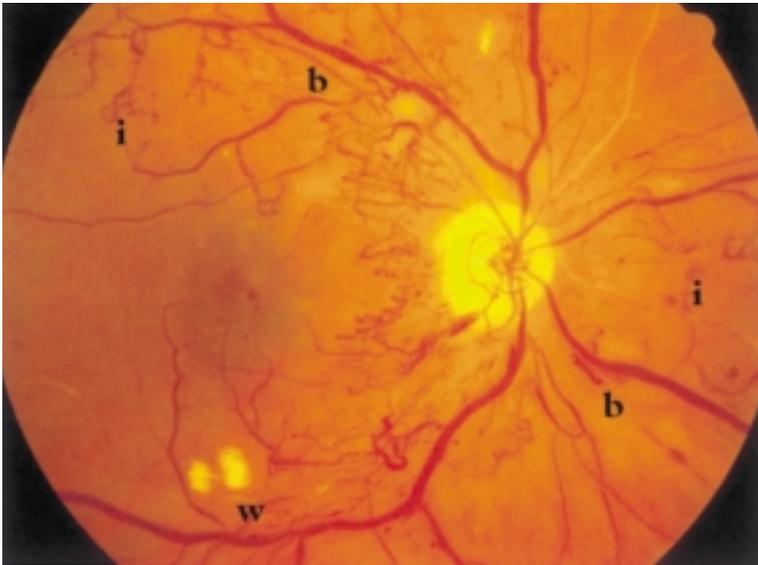


Figure 4: Severe non-proliferative diabetic retinopathy—intraretinal microvascular abnormalities or IRMA (**i**) venous beading (**b**) or venous calibre changes, widespread retinal ischaemia and cotton-wool spots (**w**)—beginning of new vessel on optic disc.



Figure 5: Proliferative diabetic retinopathy—peripheral new vessel (v), retinal haemorrhages (h) and no vitreous or pre-retinal haemorrhage—note lack of other retinopathy features.

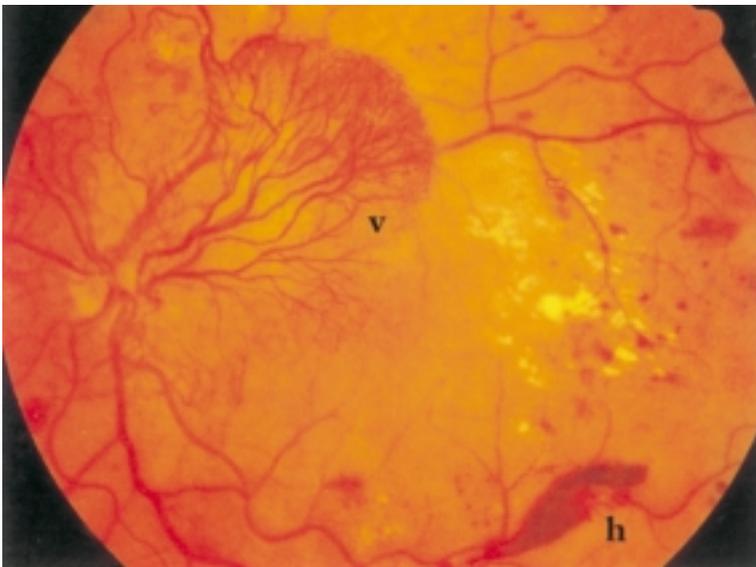


Figure 6: High-risk proliferative diabetic retinopathy—large frond of disc new vessels (v) and pre-retinal haemorrhage (h).

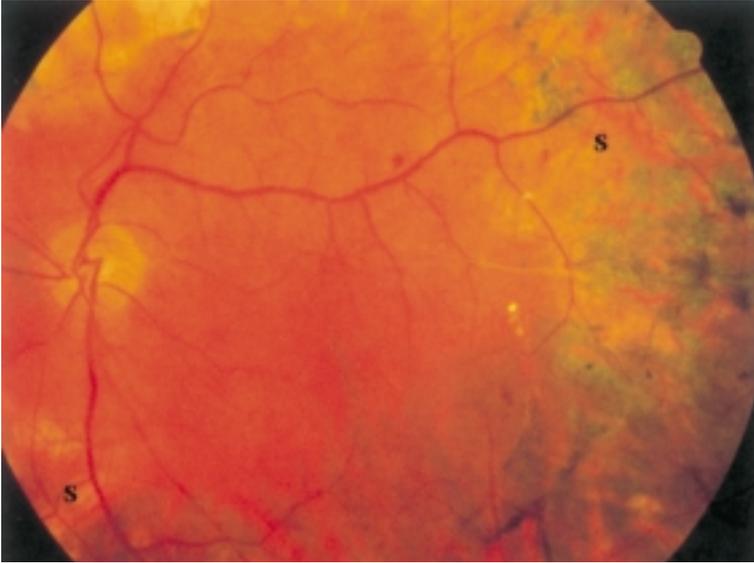


Figure 7: High-risk proliferative diabetic retinopathy—post treatment with pan-retinal laser photocoagulation scars (s) temporary and nasally—disc new vessels regressed.

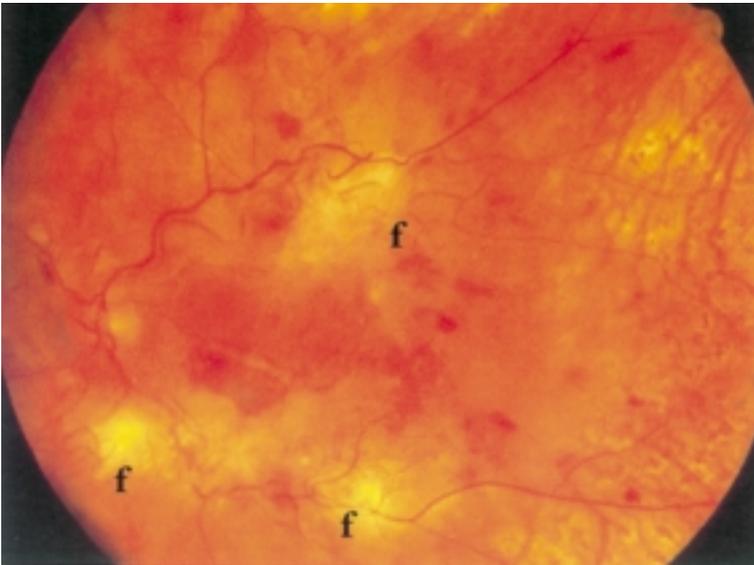


Figure 8: Advanced proliferative diabetic retinopathy—pre-retinal fibrovascular tissue producing traction on retina (f) across the macular region.



Figure 9: Clinically significant macular oedema—localised area of retinal oedema surrounded by lipid exudate (e) extending to the macula.

4 | Prevention and early detection

4.1 Prevention

4.1.1 Primary prevention: diabetes

As discussed, diabetic retinopathy occurs in people with either type 1 or type 2 diabetes mellitus. Primary prevention should therefore aim at decreasing the prevalence of diabetes within Aboriginal and Torres Strait Islander communities.

To decrease the prevalence of diabetes, dietary modification and physical activity should be encouraged in the entire Aboriginal and Torres Strait Islander population, and interventions designed to achieve this should be introduced at an early age (after age 13 years).⁴⁸ A recent study of a Central Australian Aboriginal community, in which a community-based nutrition awareness and healthy lifestyle program had been implemented between 1988 and 1990, showed that this intervention led to an improvement in dietary habits but not to a reversal of the trend towards a growing prevalence of obesity and diabetes.⁴⁹ In communities where healthy food choices are limited, the role of regular physical activity in improving metabolic fitness may also need to be emphasised.⁴⁹

Obtaining healthy food can be difficult in rural and remote communities. Because of the distances involved, food is more expensive than in metropolitan areas and transport can be problematic.

Primary prevention of diabetes: a summary

- ▶ Dietary education and modification
- ▶ Weight loss
- ▶ Increased physical activity

4.1.2 Primary prevention: diabetic retinopathy

The Diabetes Control and Complications Trial results for type 1 diabetes and the UK Prospective Diabetes Study results for type 2 demonstrated that strict glycaemic control delayed the development of diabetic retinopathy. One of the main problems associated with maintaining strict glycaemic control is the occurrence of hypoglycaemic episodes. In a rural setting where facilities are limited, this can result in increased morbidity, and perhaps mortality.

The Prospective Diabetes Study also showed that treatment of hypertension delayed the onset of diabetic retinopathy in people with type 2 diabetes. The study results emphasise the need for good control of both blood pressure and blood glucose in such people.

Primary prevention of diabetic retinopathy: a summary

- ▶ Strict glycaemic control
- ▶ Effective control of hypertension
- ▶ Lower serum lipid levels

4.1.3 Secondary prevention: diabetic retinopathy

The Diabetes Control and Complications Trial and the UK Prospective Diabetes Study demonstrated that, once the signs of diabetic retinopathy appear, maintaining good control of blood glucose and blood pressure retards the progression of retinopathy.

Early monitoring and laser treatment of retinal changes may be up to 98 per cent effective in preventing severe loss of vision.⁵⁰

4.2 Screening

It is currently recommended that all Australians with diabetes have a dilated fundus examination and a visual acuity assessment at least every two years. This is usually performed by an ophthalmologist, optometrist or other suitably trained health professional. Except in the rare instance of diabetes onset before puberty, initial assessment should occur at the time of diagnosis.

It is recommended that screening for retinopathy in type 2 diabetes be done at the time of diagnosis and every one to two years thereafter.⁵¹

For Aboriginal and Torres Strait Islander communities in rural and remote areas, it is often difficult to comply with these recommendations. Among the particular problems are the lack of suitably trained staff to perform dilated fundus examinations, the transient nature of the health care workforce and the workers' varying levels of skill, as well as the acceptability of these methods to Aboriginal and Torres Strait Islander people. Problems also arise because of the high prevalence of concurrent eye disease.⁵²

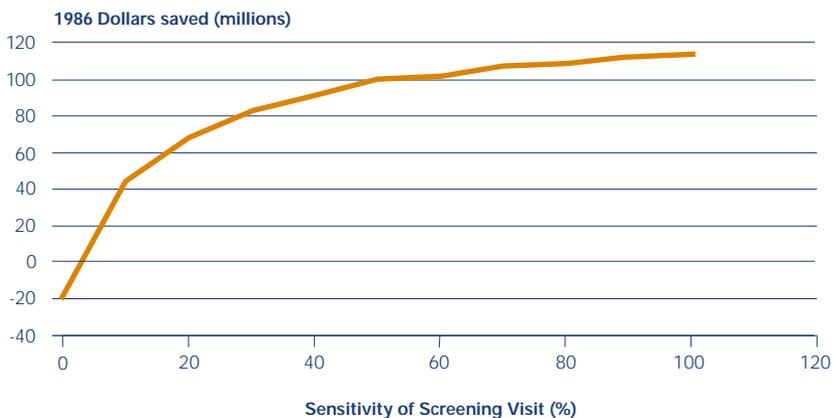
An alternative screening method for diabetic retinopathy involves the use of non-mydratric retinal cameras. Although the photos need to be read by suitably trained staff, the photographing can be done without dilation of the

pupil and the camera can be operated after minimal training. This is a major benefit of this form of screening. Aboriginal and Torres Strait Islander people can do the screening themselves in remote parts of Australia where professional services are limited. As a result, acceptance of and compliance with screening may improve, thus increasing the number of people screened.⁵¹

A screening test needs to have greater than 60 per cent sensitivity to be most effective (see Figure 4). Lower sensitivity is compensated for by regular (yearly or two-yearly) examinations. Minor early lesions not requiring treatment may be missed at the initial examination but picked up on subsequent examinations as the disease slowly progresses.

The non-mydratric retinal camera's sensitivity in detecting diabetic retinopathy has been the subject of numerous studies. Its sensitivity is at least 80 per cent and is reported to be greater than 90 per cent in ideal circumstances.^{53,54,55} A single Polaroid photograph is obviously non-stereoscopic and will not reveal the subtle retinal thickening that it is necessary to see for a diagnosis of clinically significant macular oedema, but it will almost certainly show accompanying non-proliferative changes (lipid and microaneurysms) that would be the trigger for referral of such a patient. In addition, checking visual acuity in all patients will uncover those who have visual loss as a result of maculopathy in the absence of visible non-proliferative changes.⁵⁶

Figure 4: Changes in the sensitivity of a single screening visit in detecting diabetic retinopathy: 1986 dollars saved.⁵²



Note: This US model was based on use of dilated ophthalmoscopy annually for patients with no retinopathy and every six months for those with retinopathy.

It has been estimated that 8 to 15 per cent of patients have diabetic retinopathy that is present only outside the central 45-degree field of the non-mydriatic retinal camera⁵⁴; they may therefore be missed by a single photograph. But these peripheral changes alone would rarely represent high-risk retinopathy, and this shortcoming would be compensated for by regular screening. Javitt et al.⁵² calculated that any detection method with greater than 60 per cent sensitivity is adequate for screening purposes, provided that screening is repeated at regular intervals. Using this criterion, the non-mydriatic retinal camera is more than adequate.⁵⁶

Diamond et al.⁵⁷ recently examined the effectiveness of the non-mydriatic retinal camera for identifying diabetic retinopathy among Aboriginal patients in rural Western Australia. The authors concluded, 'The Canon CR5-45NM non-mydriatic fundus camera was relatively good at identifying diabetic retinopathy and could usefully be applied within a screening programme for treatable disease within this population'. In a separate study in rural Victoria⁵⁸, Aboriginal Health Workers were trained to use the camera and produced gradable photos in 87 per cent of patients.

If it is not possible to take adequate non-mydriatic photographs—if, for example, a dark room is not available to allow for physiological dilation—it may be useful to dilate the pupils to obtain better photographs.⁵⁷

4.2.1 Barriers to screening

The National Health and Medical Research Council's recommendation on screening for diabetic retinopathy in the general diabetic population calls for a visual acuity test and fundus examination at least every two years. In the Aboriginal and Torres Strait Islander diabetic population screening is recommended annually because of the higher risk in this group.

Data from the Melbourne Visual Impairment Project showed that only 43 per cent of diabetics in the general population complied with these screening recommendations.³⁵ This poor rate of compliance suggests that there are deficiencies in primary health care recall systems, in general practitioner's examination skills, or in the referral system.⁴⁸

For Aboriginal and Torres Strait Islander communities in rural and remote Australia, there are additional barriers to screening. Among these are distance from facilities and referral systems that are more likely to falter because of long delays between visiting ophthalmologists. Further, all Indigenous Australians, regardless of their location, face cross-cultural barriers.⁴⁸

Screening for diabetic retinopathy is part of a comprehensive primary health care approach to the management of diabetes. The examination should be part of a yearly health assessment.

Screening recommendations for diabetic retinopathy in Aboriginal and Torres Strait Islander communities: a summary

- ▶ The initial examination should be conducted at the time of diabetes mellitus diagnosis.
- ▶ The annual examination should include:
 - visual acuity (Snellen chart) assessment and
 - dilated fundus examination by a general practitioner, physician, optometrist or ophthalmologist or
 - retinal photography by health care workers-the photos should be read by suitably trained personnel.

Findings on ocular examination	Recommendations
Normal screen	Repeat eye examination annually
Decreased visual acuity (<6/12) with normal fundus	Non-urgent referral to ophthalmologist
Mild or moderate non-proliferative diabetic retinopathy	Referral to ophthalmologist but can wait until next regional visit in remote areas
Severe non-proliferative diabetic retinopathy, proliferative diabetic retinopathy or macular oedema	Immediate referral to ophthalmologist for laser treatment
Ungradable photos	Non-urgent referral to ophthalmologist
Unexplained visual loss	Non-urgent referral to ophthalmologist
Media opacities	Non-urgent referral to ophthalmologist

5 | Management

Management of diabetic retinopathy begins when a patient is diagnosed with diabetes mellitus. This initial contact provides the opportunity to develop a trusting relationship with the patient and to offer advice and support, as well as make an initial assessment of their eyes. It is important to clarify how regular screening will be done and to discuss the importance of annual screening to the person's future vision. For Aboriginal and Torres Strait Islander people living in rural and remote areas of Australia, the screening options are dilated fundus examination by visiting specialists or photography of the retina with non-mydratic retinal cameras by Aboriginal Health Workers or other health care workers.

Patients should be told that it is possible to treat diabetic retinopathy effectively and that the prognosis for their long-term vision is very good if early action is taken. They should also be told how to prevent and retard the development of diabetic retinopathy through close liaison with the primary health care team. Blood glucose levels should be kept near normal; blood pressure and serum lipids should be monitored and controlled.

It is also important to inform patients that—unlike cataract surgery, which will improve vision—laser treatment for diabetic retinopathy will not produce an immediate improvement in vision; rather, the purpose is to prevent continuing loss of vision.

Audiovisual aids need to be developed and used to teach both patients and paramedical personnel about the disease.

5.1 The medical history

The initial assessment of the patient diagnosed with diabetes mellitus should include a thorough eye examination, with particular attention to those aspects relevant to diabetic retinopathy. As Figure 2 shows, 15 per cent of patients with type 2 diabetes have some signs of retinopathy at the time of diagnosis. It may be that referral to an ophthalmologist needs to occur very early for Aboriginal and Torres Strait Islander people, who tend to be diagnosed with type 2 diabetes at a later stage in the disease process.

In taking a medical history the following elements should be considered:

- ▶ the duration of diabetes—the longer the patient has had diabetes, the greater the chance of diabetic retinopathy;

- ▶ glycaemic control—glycohaemoglobin (HbA_{1c}) is a better indicator for long-term control than blood glucose levels;
- ▶ blood pressure control—hypertension should be effectively treated to delay the onset and retard the progression of diabetic retinopathy;
- ▶ serum lipid levels;
- ▶ obesity—weight reduction will aid in the control of diabetes and microvascular disease;
- ▶ smoking; and
- ▶ renal disease.

5.2 The examination

The eye examination should be comprehensive, with emphasis on best corrected visual acuity and the fundus examination.

Examination of the eye allows for the grading of any diabetic retinopathy present, so that further treatment can be determined. The aim is to intervene early to prevent visual impairment, and the presence of either of the following calls for routine referral:

- ▶ diabetic retinopathy—mild non-proliferative diabetic retinopathy or worse;
- ▶ an unexplained decrease in visual acuity.

Immediate referral is required for the following:

- ▶ macular oedema;
- ▶ neovascularisation—proliferative diabetic retinopathy;
- ▶ severe non-proliferative diabetic retinopathy—extensive retinal haemorrhages/microaneurysms, venous beading, and IRMA.

5.3 Fluorescein angiography

Fluorescein angiography involves the injection of fluorescein into the circulation so as to outline the retinal vessels. It has been used in research, and recently in randomised controlled trials, to diagnose patients, to document the adequacy of laser treatment, to identify the type and source of leakage on the retina, and to assess compliance with treatment protocols.⁵⁹

The National Health and Medical Research Council's Guidelines for diabetic retinopathy²⁸ suggest that the routine use of fluorescein angiography in managing retinopathy should be guided by clinical experience because there is

little available evidence on which to base firm Guidelines. The technique is recommended if macular oedema is present, to identify the source of perimacular leakage and to guide focal and grid laser treatment.

Aboriginal and Torres Strait Islander people in rural and remote parts of Australia may have difficulty gaining access to fluorescein angiography. In any case, experienced clinicians can manage patients without the need for this form of investigation.

Further, although fluorescein angiography is a reasonably safe procedure, the following side-effects may occur:

- ▶ nausea;
- ▶ vomiting;
- ▶ allergic skin reactions—urticaria;
- ▶ allergic reaction to fluorescein dye—resuscitation equipment should always be on hand when performing the angiogram;
- ▶ dizziness;
- ▶ chest pain;
- ▶ myocardial infarction;
- ▶ asystole;
- ▶ death.

5.4 Management and treatment

The grading of the retinal changes that are seen on fundus examination determines the management and treatment of patients with diabetic retinopathy. In general, if the changes are minimal, annual screening is all that is required until the retinopathy worsens.

5.4.1 Equipment and facilities

Assessment and treatment of diabetic retinopathy by laser is best done in a darkened room that has an adequate and continuous electrical supply. It is highly recommended that there be available a high-quality slit lamp with high-quality and robust optics combined with a compatible laser-delivery system. Patient and health professional should be seated, preferably on comfortable, adjustable stools.

Nevertheless, it is possible to provide safe treatment under sub-optimal conditions.

5.4.2 Laser treatment

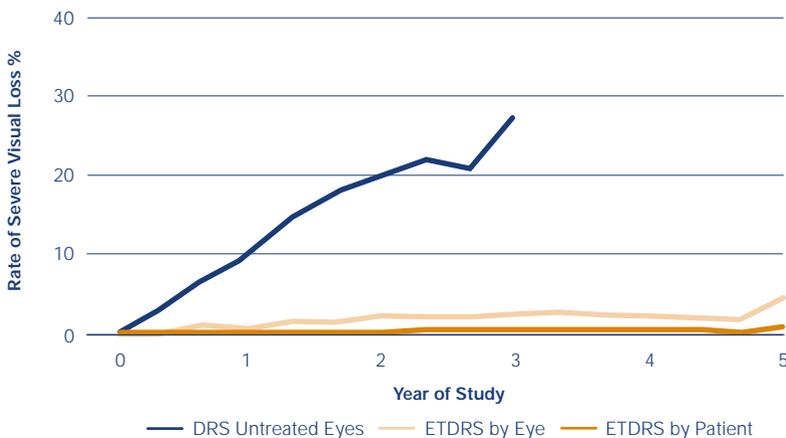
Immediate treatment is necessary for patients with macular oedema or proliferative diabetic retinopathy, or both. Treatment should also be considered for patients with severe non-proliferative diabetic retinopathy. In addition, if compliance with follow-up is likely to be poor, or if the patient has cataracts or is pregnant, treatment should not be delayed.

Laser surgery (retinal photocoagulation) is the main treatment used for diabetic retinopathy. The Diabetic Retinopathy Study (DRS) and Early Treatment Diabetic Retinopathy Study (ETDRS) trials have shown conclusively that timely laser treatment is effective in patients with both proliferative diabetic retinopathy and clinically significant macular oedema.²⁸ The significant improvement in outcome demonstrated in the ETDRS was achieved by stringent adherence to the laser-treatment recommendations as well as close follow-up with re-treatment as needed.

The patient's consent should be obtained before laser therapy.

Figure 5 shows rates of severe visual loss (visual acuity less than 5/200), assessed at each study visit after proliferative diabetic retinopathy was diagnosed, for untreated eyes in the DRS compared with treated eyes (or patients) in the

Figure 5: Proliferative diabetic retinopathy: proportion of untreated eyes in the Diabetic Retinopathy Study developing severe visual loss compared with treated eyes and patients in the Early Treatment Diabetic Retinopathy Study.⁵⁰



Note: Severe visual loss = visual acuity less than 5/200.

ETDRS. Although the risk of severe visual loss for untreated DRS eyes at three years approached 30 per cent, only 4 per cent of treated eyes in the ETDRS had reached severe visual loss by five years and only 1 per cent of patients had this degree of visual loss in both eyes.⁵⁰

5.4.3 The type and extent of laser treatment

Focal treatment for clinically significant macular oedema

For macular (focal) treatment, use small spot-sized (100-micron) focal laser burns applied directly to leaking microaneurysms and in a grid pattern to areas of diffuse leakage or retinal thickening, or both. Direct treatment of microaneurysms should result in a colour change (whitening or darkening) of the microaneurysms. Grid-pattern burns should be of mild intensity, spaced more than one burn-width apart, and no burns should occur closer than 500 microns from the centre of the macula.

Panretinal photocoagulation treatment for proliferative retinopathy or severe non-proliferative diabetic retinopathy

For panretinal photocoagulation treatment (PRP), the Early Treatment Diabetic Retinopathy Study recommended 500-micron moderate-intensity burns placed approximately half a burn-width apart, from the posterior fundus to the equator. PRP is usually divided into two or more sessions per eye. Standard treatment should total 1200–1600 burns, not closer than two disc diameters from the centre of the macula.

If both clinically significant macular oedema and proliferative diabetic retinopathy are present in the same eye, it is important to apply focal treatment for the former before starting PRP. If clinically significant macular oedema and high-risk proliferative diabetic retinopathy are present in the same eye, both focal treatment and panretinal photocoagulation treatment should be applied in the first session.

5.4.4 Side-effects and complications of laser treatment

Patients should be advised that not all their treatment can be carried out at one time or in one place.

The most frequent side-effect of laser therapy is discomfort or pain during PRP; in some cases peribulbar anaesthesia is necessary.

After treatment, transient blurring of vision, for days or weeks, is also common.

Longer term visual reduction may result from exacerbation of macular oedema in some patients. This effect can be minimised by treating any macular oedema before starting PRP, as recommended by the Early Treatment Diabetic Retinopathy Study.⁶⁰ There is also a slight risk of damage to the macula from inadvertent foveal contact or from subsequent migration of laser-treatment scars.⁶¹

Increased sensitivity to glare and difficulty with light-dark adaptation are also common in patients with diabetic retinopathy: these problems may become more severe after laser treatment.

No increased risk of cataract has been reported from laser treatment.²⁸

Be wary of attempting to 'overtreat' in one session: exudative retinal detachment and other complications can occur.

5.4.5 Vitreoretinal surgery

Patients requiring vitreoretinal surgery should be appropriately referred.

5.5 Follow-up

Close follow-up, and re-treatment as necessary, after laser treatment for diabetic retinopathy were important factors in achieving the significant improvement in outcome observed in the Early Treatment Diabetic Retinopathy Study.

Laser treatment follow-up: a summary

- ▶ After focal treatment:
 - Review at two to four months.
 - Repeat focal treatment if significant retinal thickening persists at four months.
- ▶ After panretinal photocoagulation treatment:
 - Review at two to four months.
 - If new vessels are stable or regressing, treatment may be adequate and the patient should be reviewed at four months.
 - If new vessels worsen, further panretinal photocoagulation treatment is necessary.

Table 2: Management recommendations²⁸: a summary

Retinopathy stage	Focal or grid laser	Panretinal laser	Follow-up (months)
Mild or moderate non-proliferative diabetic retinopathy			
No macular oedema	No	No	6–12
Early macular oedema	Sometimes	No	4–6
Clinically significant macular oedema	Yes	No	2–4
Severe non-proliferative diabetic retinopathy			
No macular oedema	No	Sometimes ^a	2–4
Early macular oedema	Sometimes	Sometimes, after focal or grid laser	2–4
Clinically significant macular oedema	Yes	Sometimes, after focal or grid laser	2–4
Proliferative diabetic retinopathy			
No macular oedema	No	Yes	2–4
Early macular oedema	Yes	Yes, after focal or grid laser	2–4
Clinically significant macular oedema	Yes	Yes, after focal or grid laser	2–4
High-risk proliferative diabetic retinopathy			
No macular oedema	No	Yes	2–4
Early macular oedema	Yes	Yes	2–4
Clinically significant macular oedema	Yes	Yes	2–4

a. Consider panretinal photocoagulation treatment if compliance with a follow-up regime is likely to be poor or if the patient has cataracts or is pregnant.

6 | Cataract surgery and diabetic retinopathy

For people who have both cataracts and diabetic retinopathy, current opinion recommends adequate laser treatment of significant retinopathy before cataract surgery. Treatment of any macular oedema or threatened maculopathy should be with focal or grid laser.²⁸

The reason for treating diabetic retinopathy before cataract surgery is that pre-operative retinopathy, particularly maculopathy, influences the visual outcome after cataract surgery as a result of asymmetric retinopathy progression in the operated eye. This leads to an increased risk of rubeosis iridis or neovascular glaucoma.²⁸

Sometimes it is necessary to remove the cataract to complete the laser treatment. The laser treatment should be completed as soon as possible after cataract surgery.