

# A person-centred approach to determining target HbA<sub>1c</sub> for older people with diabetes

Barbara Jane Waterman

## Article points

1. The development of diabetes in older people is caused by a combination of environmental and genetic factors superimposed on normal age-related changes in carbohydrate metabolism.
2. Management options for this age group may require consideration of pre-existing disease or disability.
3. Current treatment options may be contraindicated, have undesirable side-effects, or little effect on disease progression.
4. An expanded Diabetic Annual Review Guide for older people is proposed, which includes psychosocial and functional considerations, enabling individual glycaemic targets to be set.

## Key words

- Older people
- Person-centred approach
- Target HbA<sub>1c</sub>
- Type 2 diabetes

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Although early, intensive glycaemic control in diabetes has been shown to reduce the long-term risk of micro- and macrovascular disease, there is uncertainty about the mechanisms responsible and the ideal glycaemic target, particularly for older people with comorbidities. In light of such uncertainty, the clinician must cast a wide net of clinical enquiry to locate the individual within the epidemiological. An expansion of current diabetes annual review consultations designed to capture elements of the psychosocial considerations of older people and incorporate them into the physiological review is proposed. Such exploration can preserve a sense of perspective that is lost when centrally set targets drive decisions, and thereby allow an individualised person-centred evaluation of competing issues.

Care of older people with type 2 diabetes is guided by NICE (2009) guidance, driven by the QOF (NHS Employers and BMA, 2009), complicated by their clinical and functional heterogeneity (American Diabetes Association [ADA], 2010) and handicapped by the scarcity of data regarding the use of pharmacological interventions in this age group (Hawthorne and Yarnall, 2009; Neumiller and Setter, 2009).

The challenge of managing people in this age group is to reflect on the past, observe the present and predict the future, using evidence-based practice to engage pharmaceutical, psychosocial and personal resources, aiming towards person-centred goals that enhance quality of life (Department of Health [DH],

2010) while reducing mortality and morbidity (NICE, 2009). This article explores some under-appreciated aspects of management and proposes an expansion of current diabetes annual review consultations to incorporate psychosocial and functional considerations in order to individualise glycaemic targets.

The author works in a general practice where the incidence of diagnosed diabetes in the adult population (7.9%;  $n=474$ ) is almost double the UK average (4.36%; Diabetes UK, 2010). Among older people (65 years and over) in the practice population, the incidence rises to 22.9% ( $n=274$ ). As the most significant demographic change in diabetes prevalence in future years will be the increase in the proportion of older people (Wild et al, 2004;

### Page points

1. Seventy-five per cent of those over the age of 65 years with type 2 diabetes have two or more comorbidities and 66% have evident macrovascular disease.
2. Diabetic retinopathy affects 40% and the risk of blindness is increased by glaucoma, cataracts and macular degeneration.
3. Fifty per cent have gastro-oesophageal reflux disease, 25% depression, 20% chronic airways disease, 16% arthritis and 15% chronic heart failure.
4. An increased number of comorbidities results in a decreased prioritisation of diabetes.
5. Cognitive decline is uniquely associated with diabetes, and prevalence of depression is doubled.

Cefalu and Cefalu, 2006; Arzumanyan et al, 2010; Caughey et al, 2010), primary care nurses must prepare for an increased demand on services, exacerbated by the surge of new diagnoses resulting from imminent changes in diagnostic criteria (John et al, 2011).

### Diabetes in older people

Older people with diabetes are a unique group (Chelliah and Burge, 2004) within which current treatment options may have contraindications, undesirable side-effects and little effect on disease progression (ADA, 2010; Piya et al, 2010).

The pathophysiology of type 2 diabetes developing in older people is different from that developing in younger people. For example, different pathophysiological mechanisms of insulin secretion may be responsible for islet sensitivity (Burcelin and Dejager, 2010) and fasting hepatic glucose production is not increased (Meneilly, 2010). Non-insulin-mediated glucose uptake is impaired and beta-cell response to gastric inhibitory polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) is reduced (Meneilly, 2010).

Diabetes developing in this age group is caused by a combination of environmental and genetic factors superimposed on normal age-related changes in carbohydrate metabolism (Meneilly, 2010).

### Impact of comorbidities

Management options for older people may require consideration of pre-existing disease or disability. Seventy-five per cent of those over the age of 65 years with type 2 diabetes have two or more comorbidities (Caughey et al, 2010) and 66% have evident macrovascular disease (Gunasekaran and Fowler, 2010). Diabetic retinopathy affects 40% and the risk of blindness is increased by glaucoma, cataracts and macular degeneration (Abbatecola et al, 2008). Fifty per cent have gastro-oesophageal reflux disease, 25% depression, 20% chronic airways disease, 16% arthritis and 15% chronic heart failure (Caughey et al, 2010).

Cognitive decline is uniquely associated with diabetes, and prevalence of depression

is doubled (Abbatecola et al, 2008). The downward spiral of frailty is activated more quickly in diabetes; those with the condition are two- to three-times less able to walk 400 m, prepare meals or do housework than their peers without diabetes (Abbatecola et al, 2008). Pain, impaired mobility, hand tremor, malnutrition, falls, fractures, incontinence and urine infections are prevalent in this group and impact on management (Vischer et al, 2010). An increased number of comorbidities results in a decreased prioritisation of diabetes (Caughey et al, 2010).

Polypharmacy is common in older people (Kennedy et al, 2006; Neumiller and Setter, 2009; Caughey et al, 2010; Vischer et al, 2010), with ensuing risks of interactions and adverse effects heightened by compromised renal and hepatic function (Neumiller and Setter, 2009), including hypoglycaemia.

Ageing is associated with changes in multiple pharmacokinetic parameters, although considerable individual variation makes predictions based on age alone insufficient (Neumiller and Setter, 2009).

Sarcopenic obesity (age-related changes in muscle composition and quality) and poorly regulated food intake may alter hormonal and neurotransmitter regulation, increasing the difficulties inherent in the control of diabetes (Abbatecola et al, 2008; Neumiller and Setter, 2009). The focus of treatment should be ensuring the safety of the individual while making changes that improve quality of life and reduce mortality where possible (Chelliah and Burge, 2004; Cefalu and Cefalu, 2006; Abbatecola et al, 2008; Arzumanyan et al, 2010; Gunasekaran and Fowler, 2010).

### Diabetes in perspective

Prioritising treatment outcomes may be a complex process within which the duration of diabetes is important. In 2005, a 65-year-old person with diabetes had a predicted life-expectancy of 17–20 years, long enough to potentially develop long-term complications of the condition (Kant et al, 2010). The benefit of glycaemic control is evident after 8 or more years, whereas blood pressure (BP) and lipid

control produce benefit after only 2–3 years (Neumiller and Setter, 2009). In older people with low functional status, cardiovascular and mortality outcomes in those with and without diabetes were similar (Cefalu and Cefalu, 2006).

Diabetes care for older people who are functional, cognitively intact and have significant life expectancy should be the same as for younger adults, including an HbA<sub>1c</sub> target of <48 mmol/mol (<6.5%) (NICE, 2009). For others, glycaemic targets may be relaxed while avoiding symptomatic hyperglycaemia (NICE, 2009; ADA, 2010).

Intensive glycaemic control in the early years of diabetes has been shown to reduce the risk of micro- and macrovascular disease in the longer term (DCCT/EDIC [Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications] Study Research Group, 2005; Holman et al, 2008; Eldor and Raz, 2009; Gore and McGuire, 2009; Gunasekaran and Fowler, 2010); however, there is uncertainty about the mechanisms responsible (Holman et al, 2008; Gore and McGuire, 2009) and the ideal glycaemic target, particularly for older people with comorbidities.

Trials powered (yet insufficiently [Nichols, 2008]) to evaluate the effects of intensive pharmacological glycaemic control on macrovascular disease outcomes in cohorts of people at high cardiovascular risk, found a significantly higher mortality risk associated with a median HbA<sub>1c</sub> level of 46 mmol/mol (6.4%) (ACCORD [Action to Control Cardiovascular Risk in Diabetes] Study Group, 2008) and no significant benefit of intensive glycaemic control in reducing macrovascular events (ADVANCE Collaborative Group, 2008; Liebson, 2008; Eldor and Raz, 2009; Gore and McGuire, 2009; VADT Investigators, 2009).

Uncertainty exists over the relative impact of oscillating glycaemia versus stable high blood glucose (Kilpatrick et al, 2009; Ceriello and Ihnat, 2010) and the presence of any natural glycaemic thresholds (Hawthorne and Yarnall, 2009; Selvin et al, 2010) in the activation

of pathways involved in the pathogenesis of diabetes complications.

In the shadow of such uncertainty, the clinician must cast a wide net of clinical enquiry to locate the individual within the epidemiological and individualise glycaemic targets. Target options and therapeutic pathways may be limited by a need to avoid the increased risk of hypoglycaemia inherent in tight glycaemic control (ACCORD Study Group, 2008; ADVANCE Collaborative Group, 2008; Neumiller and Setter, 2009; VADT Investigators, 2009; Piya et al, 2010), and there may be limited potential benefit from such control if life-expectancy is short. Clinical idealism must be tempered by the cognitive and physical ability, psychosocial environment and quality-of-life goals of the individual, and the possible lack of potential benefit.

Advanced age is itself a risk factor for hypoglycaemia (Chelliah and Burge, 2004; Kant et al, 2010); the physical risk and emotional distress associated with such episodes result in hypoglycaemia being the main limiting factor in the glycaemic control of older people (Chelliah and Burge, 2004; Alagiakrishnan and Mereu, 2010), partly due to patient-initiated medication omission to avoid them (Neumiller and Setter, 2009). Ageing modifies the counterregulatory and symptomatic responses to hypoglycaemia, and the decreased cognition, renal impairment, polypharmacy and malnutrition commonly found in this age group contribute to the increased risk (Alagiakrishnan and Mereu, 2010; Kant et al, 2010). The risk of progression to severe hypoglycaemia is high because of their altered symptom intensity and glycaemic thresholds (Chelliah and Burge, 2004), and impaired psychomotor performance when blood glucose levels are low (Nichols, 2008). The risk of unanticipated, severe or fatal hypoglycaemia associated with the use of insulin, sulphonylureas and metaglinide derivatives, such as nateglinide or repaglinide, increases exponentially with age (Chelliah and Burge, 2004). *Table 1* summarises the risk factors for hypoglycaemia in older people with type 2 diabetes.

Older people, having a high incidence of cardiovascular disease (Gunasekaran and

#### Page points

1. Intensive glycaemic control in the early years of diabetes has been shown to reduce the risk of micro- and macrovascular disease in the longer term; however, there is uncertainty about the mechanisms responsible and the ideal glycaemic target, particularly for older people with comorbidities.
2. Target options and therapeutic pathways may be limited by a need to avoid the increased risk of hypoglycaemia inherent in tight glycaemic control, and there may be limited potential benefit from such control if life-expectancy is short.
3. Clinical idealism must be tempered by the cognitive and physical ability, psychosocial environment and quality of life goals of the individual, and the possible lack of potential benefit.

**Table 1. Risk factors for hypoglycaemia in older people with type 2 diabetes.**

<b>Pharmaceutical</b>	<ul style="list-style-type: none"> <li>● Adrenergic blocking agents</li> <li>● Complex drug regimens</li> <li>● Polypharmacy</li> <li>● Secretagogues/insulin</li> <li>● Sedative agents</li> <li>● Tight glycaemic control</li> </ul>
<b>Physiological</b>	<ul style="list-style-type: none"> <li>● Advanced age</li> <li>● Autonomic neuropathy</li> <li>● Cognitive impairment</li> <li>● Endocrine deficiency</li> <li>● Hepatic dysfunction</li> <li>● Hypoglycaemic unawareness</li> <li>● Intercurrent illness</li> <li>● Renal insufficiency</li> </ul>
<b>Personal</b>	<ul style="list-style-type: none"> <li>● Alcohol consumption</li> <li>● Poor nutrition</li> <li>● Recent hospitalisation</li> <li>● Reliance on others to administer medication</li> <li>● Mistiming of medication in relation to meals</li> <li>● Poor adherence to medication regimens</li> </ul>

Fowler, 2010), are also at particular risk *from* hypoglycaemia – repeated episodes being an aggravating factor for preclinical atherosclerosis (Gimenez et al, 2010). Each episode has complex vascular effects, including acute activation of prothrombotic, pro-inflammatory and pro-atherogenic mechanisms (Gogitidze et al, 2010), which may in turn increase the risk of cardiovascular mortality (ACCORD Study Group, 2008).

Furthermore, instability during hypoglycaemia may result in falls (Hawthorne and Yarnall, 2009), with ensuing morbidity, mortality and loss of independence. Of a population aged 70 years or more, 77% of the frail and 30% of the non-frail with an HbA<sub>1c</sub> level of <53 mmol/mol (<7.0%) fell, compared with 58% of the frail and 12% of the non-frail with a higher HbA<sub>1c</sub> level (Nelson et al, 2007). Adynamic bone disease (characterised by low bone turnover without osteoid accumulation) and increased fracture risk result from the development of relative hypocalcaemia with age-related or diabetes-related renal disease, increasing the risk of injury

from falls (Seaquist and Ibrahim, 2010). A considerable number of falls and fractures could potentially be prevented by more conservative glycaemic control (Nelson et al, 2007).

Instability is particularly increased in older people with diabetic peripheral neuropathy, found in 30–50% of those with diabetes of 15–20 years' duration (Tsitouras, 2010), and the significant reduction in joint mobility found in older people with diabetes compared with those without (Abate et al, 2010). Hence duration of disease, life-expectancy and prevention of microvascular complications must be included in the risk/benefits evaluation for each older person (DCCT/EDIC Study Research Group, 2005; Nelson et al, 2007; Holman et al, 2008; ADA, 2010).

Such considerations pertain also to prevention of renal disease in older people with diabetes. Tight glycaemic control to an average HbA<sub>1c</sub> level of 48 mmol/mol (6.5%) may protect against the development of renal impairment or deterioration in renal function (ADVANCE Collaborative Group, 2008; Holman et al, 2008; Seaquist and Ibrahim, 2010). However, only 30% of people with diabetes of 15 years' duration develop renal impairment (Seaquist and Ibrahim, 2010), and the incidence and prevalence of chronic kidney disease increase markedly at older age.

The weaker association of renal impairment with diabetes at older ages suggests that it may be a different physiological condition to that seen in younger adults with diabetes (Islam et al, 2009). An estimated glomerular filtration rate (eGFR) of 59–45 mL/min/1.73 m<sup>2</sup> in an older person is generally due to age-related kidney dysfunction rather than renal pathology (Mangione and Canton, 2010).

Although proteinuria in people with diabetes is usually considered to be indicative of diabetic nephropathy, one study found that 52% of such patients had non-diabetic renal disease on biopsy (Mou et al, 2010). There are many problems associated with the accurate estimation of renal function in older people (Mangione and Canton, 2010), which makes interpretation of the clinical relevance of a reduced eGFR problematic.

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1. Diabetes has a deleterious effect on neuronal integrity, negatively impacting on cognitive functioning and memory, and adding the equivalent of some 4 years of ageing.
2. Diabetes results in more extensive vascular pathology, and doubles the incidence of dementia, cerebral infarcts and Alzheimer's disease.
3. People with diabetes have a doubled risk of comorbid depression, which is associated with impaired quality of life, micro- and macrovascular disease and mortality, partly through poor glycaemic control.
4. The coexistence of diabetes distress and depression triples the incidence of suboptimal glycaemic control.

When deciding management priorities to prevent progression of renal disease, BP management takes precedence over glycaemic control (Seaquist and Ibrahim, 2010) but does not exclude it (ADVANCE Collaborative Group, 2008). A person with diabetes and an eGFR of 50 mL/min will lose 2–5 mL/min/year if systolic BP is <130 mmHg and may not need renal replacement therapy for 10–20 years. A systolic BP of >160 mmHg will result in a loss of 12–14 mL/min/year, and the need for renal replacement in 4–5 years (Seaquist and Ibrahim, 2010). Lipid control may slow the rate of progression in less severe renal disease but has not been studied in a rigorous prospective manner (Seaquist and Ibrahim, 2010). Preventing high levels of albuminuria may delay disease progression (Liebson, 2008; Seaquist and Ibrahim, 2010).

Postprandial renal glucose uptake is more than doubled in people with diabetes compared with those without, but the impact of this on the pathogenesis of diabetic nephropathy – and therefore on pharmacological management – is unclear (Gerich, 2010). Renal protection is therefore important in preventing renal-related morbidity, including anaemia and bone disease, and reduced medication options, but achieving it in older people may not depend primarily on intensive glycaemic control.

Cognitive ageing is more rapid among older people with diabetes than in those without, although the rate of deterioration converges at 95 years of age (Verhaegen et al, 2003). Diabetes has a deleterious effect on neuronal integrity (van Elderen et al, 2010), negatively impacting on cognitive functioning and memory (Ding, 2010), and adding the equivalent of some 4 years of ageing. Diabetes results in more extensive vascular pathology, and doubles the incidence of dementia, cerebral infarcts and Alzheimer's disease (Hawthorne and Yarnall, 2009; Ahtiluoto et al, 2010).

Such impairments have an impact on self-care (Hewitt et al, 2011) and adherence to medication regimens, which may be less than 50% among older people with chronic disease, with omission of doses far outnumbering

commission (person does not remember taking dose and takes extra dose) (Vedhara et al, 2004).

The presence of diabetic retinopathy serves as a useful marker for cerebrovascular disease, as considerable homology exists between the retinal and cerebral microcirculations (Ding, 2010). A Mini-Mental State Examination of 1047 people over the age of 75 found that 20% had cognitive impairment (Hewitt et al, 2011), and the test could be used to guide decisions regarding diabetes management.

Self-rated health status is consistently associated with mortality in older people with diabetes (Landman et al, 2010), and self-care impacts negatively on depressive symptoms, even at subclinical levels (Gonzalez et al, 2008). People with diabetes have a doubled risk of comorbid depression (Abbatecola et al, 2008; van Bastelaar et al, 2008), which is associated with impaired quality of life, micro- and macrovascular disease and mortality, partly through poor glycaemic control (van Bastelaar et al, 2008).

The numbers of prescribed medications and comorbid conditions – each of which is prevalent among older people with diabetes (Abbatecola et al, 2008) – are individually significantly associated with depressive symptoms (Gonzalez et al, 2008), and the coexistence of diabetes distress and depression triples the incidence of suboptimal glycaemic control (HbA<sub>1c</sub> >69 mmol/mol [>8.5%]) (Gonzalez et al, 2008). Loneliness, common among older people, has a deleterious effect on increased central obesity and metabolic syndrome (Whisman, 2010).

Quality of life is negatively impacted by diabetes, particularly freedom to eat and drink, enjoyment of food, and worries about the future (Collins, 2009); the burden on carers can be overwhelming (Sinclair et al, 2010). However, the difficulties in measuring quality of life make this area of research problematic (Speight et al, 2009) and its application to practice even more so.

Although addressing the emotional issues related to the experience of living with diabetes should help to improve emotional wellbeing and clinical outcomes (van Bastelaar et al,



**Page points**

1. Tailored support and intervention to prevent diabetes-related complications and enhance quality of life in older people requires an exploration of, and respect for, their individual health goals.
2. Older people may not distinguish between the prevention of different complications, or between the importance of distinct treatments.
3. Lifestyle changes remain a management priority into old age despite resistance; one study found that 29% made no effort to change their diet, and 36% were not exercising at all.
4. Antidiabetes drugs should be considered for hypoglycaemic effect and also for other attributes: effect on weight, glycaemic durability, cardiovascular protection, individual experience with the drug, method of delivery and side-effects profile.

2008; Landman et al, 2010), and such support is deemed lacking by patients (Diabetes UK and NHS Diabetes, 2010), psychosocial interventions with clinically relevant benefit have yet to be identified (Harkness et al, 2010; Landman et al, 2010).

Awareness of the impact of personality on attitude to health and self-management may enable management to be appropriately focused and motivational strategies used to encourage and empower people with diabetes, supporting behavioural changes that could be instrumental in improving metabolic control (Mosnier-Pudar et al, 2010). In their survey of French people with diabetes with a mean age of 66 years, Mosnier-Pudar et al identified five patient types: committed (25%), carefree (23%), bitter (19%), disheartened (19%) and overwhelmed (15%) (*Appendix 1*), with no differences associated with age. Improvements resulting from repeated consistent information and encouragement (Frosch et al, 2010) may become self-reinforcing (Zeber and Parchman, 2010).

Malnutrition is a significant clinical issue. One study found malnutrition in 13.9% of older people with diabetes, and “risk of malnutrition” in a further 75%, despite 39% of them being obese. Many of these were over-medicated: 59% of those on oral medication in this study had it stopped, and the proportion of those without pharmacological treatment increased from 8% to 39% (Vischer et al, 2010).

Tailored support and intervention to prevent diabetes-related complications and enhance quality of life in older people requires an exploration of, and respect for, their individual health goals (DH, 2010). Older people may not distinguish between the prevention of different complications, or between the importance of distinct treatments (Huang et al, 2005). Older people in the study by Huang et al cited the desire to remain independent in their activities of daily living as the most common healthcare goal (71%) and for 43% this was their primary healthcare goal; the second goal was staying alive and healthy.

To be effective and inclusive, clinicians should use motivational interviewing techniques (Jansink et al, 2010) and frame discussions in

the individual’s own language of global and functional terms (Huang et al, 2005).

**Individualising glycaemic targets**

The author has developed a Diabetes Risk Review Guide (*Appendix 2*), currently being trialled at her workplace (*Appendix 3*), for use before and during diabetes annual review consultations with older people, in an attempt to capture elements of the psychosocial and incorporate them into the physiological review. Such exploration can preserve a sense of perspective that is lost when centrally set targets drive decisions, thereby allowing an individualised, person-centred evaluation of competing issues (Abbatecola et al, 2008).

The guide incorporates the concept of a risk/benefit assessment to determine a target HbA<sub>1c</sub> level, to be located within a comprehensive management plan that includes aggressive treatment of dyslipidaemia and hypertension (Eldor and Raz, 2009; Hawthorne and Yarnall, 2009; Gunasekaran and Fowler, 2010). The standard HbA<sub>1c</sub> target is set at 53 mmol/mol (7.0%) (Eldor and Raz, 2009), with further adjustments based on the criteria shown in *Figure 1*. Thereafter, glycaemic management options may be individually determined.

Lifestyle changes remain a management priority into old age (Kennedy et al, 2006) despite resistance; one study found that 29% made no effort to change their diet, and 36% were not exercising at all (Huang et al, 2005). Yet diet and exercise-induced weight loss can increase beta-cell function due to reduced glucotoxicity, increase insulin-stimulated glucose disposal (Solomon et al, 2010) and improve cardiovascular risk factors (Look AHEAD [Action for Health in Diabetes] Research Group and Wing, 2010).

Antidiabetes drugs should be considered for hypoglycaemic effect and also for other attributes: effect on weight, glycaemic durability, cardiovascular protection, individual experience with the drug, method of delivery and side-effects profile.

Medication options may be limited in older people with diabetes and comorbidities, owing to adverse drug interactions (Abbatecola et al,

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1. The author recommends that primary care nurses use the Diabetic Annual Review Guide before consultation in order to highlight problematic biochemistry results and alert the clinician to comorbidities and medication omissions or potential interactions.
2. During the consultation, personal health attitudes and goals, cognition, depression, frailty and instability can be evaluated with pertinent questioning, and a risk/benefit assessment made.
3. The completed Review Guide may be scanned into the individual's computer record, providing documented evidence of the decision-making process.

2008). Reduced renal clearance may result in lactic acidosis with metformin, hypoglycaemia with nateglinide and sulphonylureas, and oedema formation with glitazones (Sampanis, 2008). Insulin doses should be reduced (Sampanis, 2008), and careful use is warranted in older people to minimise the risk of adverse effects (Chelliah and Burge, 2004; Cefalu and Cefalu, 2006; Neumiller and Setter, 2009; Kant et al, 2010).

Evidence for the use of metformin where possible is strong (Holman et al, 2008; Gore and McGuire, 2009; NICE, 2009; Boyle et al, 2010). Where it is contraindicated, insufficient or not tolerated, incretin-based therapies are becoming an attractive option (Abbatecola et al, 2008; NICE, 2009; Pala et al, 2010), particularly dipeptidyl peptidase-4 (DPP-4) inhibitors.

DPP-4 inhibitors are a well-tolerated, efficacious oral treatment option for older people with diabetes (Brazg et al, 2007; Abbatecola et al, 2008; Arzumanyan et al, 2010; Aschner et al, 2010; Engel et al, 2010; Seck et al, 2010), with potentially favourable cardiovascular implications (Fadini et al, 2010) and possibly a more sustained response to treatment in older than in younger people (Burcelin and Dejager, 2010). Dosage

may need to be reduced in people with renal impairment (Piya et al, 2010)

**Recommendations and conclusion**

The author recommends that primary care nurses use the Diabetic Annual Review Guide before consultation in order to highlight problematic biochemistry results and alert the clinician to comorbidities and medication omissions or potential interactions. Pathophysiology can be anticipated and the BP target determined.

During the consultation, personal health attitudes and goals, cognition, depression, frailty and instability can be evaluated with pertinent questioning, and a risk/benefit assessment made. Thereafter, a safe and person-centred decision can be made regarding the glycaemic target. The nurse will be sufficiently well informed to negotiate management strategies in keeping with the individual's physiological and psychosocial position, and may use appropriate motivational techniques to achieve lifestyle and medication adherence. The completed Review Guide (*Appendices 2 and 3*) may be scanned into the person's computer record, providing documented evidence of the decision-making process.

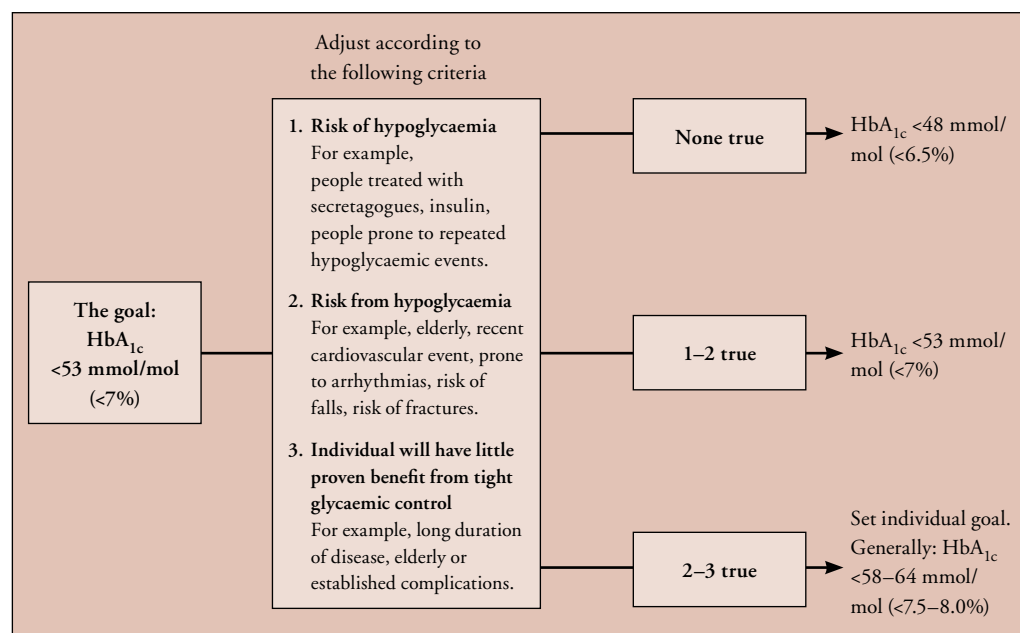


Figure 1. Determining the optimal HbA<sub>1c</sub> goal for the older person with diabetes. The algorithm shows the sequence of criteria according to which the treatment should be tailored to the individual (adapted from Eldor and Raz, 2009, with permission).

This article highlights the multi-dimensional nature of the experience of diabetes in older people, acknowledges that interventions based on correlations and uncertainty fall short of the gold standard, but offers a framework for individualised decision-making and supported disease management in line with DH intentions. ■

### Appendices 2 and 3

These will be available online at: [www.diabetesandprimarycare.co.uk](http://www.diabetesandprimarycare.co.uk).

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**“This article highlights the multidimensional nature of diabetes in older people, acknowledges that interventions based on correlations and uncertainty fall short of gold standard, but offers a framework for individualised decision-making and supported disease management in line with Department of Health intentions.”**



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Appendix 1. Characteristic behaviours and attitudes towards type 2 diabetes summarised by patient type (Mosnier-Pudar et al, 2010).	
<p><b>Committed</b></p> <ul style="list-style-type: none"> <li>● Take action on self-management</li> <li>● Take responsibility for lifestyle</li> <li>● More likely to exercise</li> <li>● View physicians as partners</li> <li>● Describe family and friends as supporters</li> <li>● Diabetes is not perceived as a burden</li> </ul> <p><b>Carefree</b></p> <ul style="list-style-type: none"> <li>● Express little concern about diabetes</li> <li>● Do not perceive a need to modify lifestyle</li> <li>● Have high levels of drug compliance</li> <li>● Perform minimal self-care</li> <li>● Require little support from family and friends</li> <li>● Diabetes is not perceived as a burden as it is disregarded</li> </ul> <p><b>Bitter</b></p> <ul style="list-style-type: none"> <li>● Express unfairness and revolt against diagnosis</li> <li>● Take no responsibility for previous poor lifestyle</li> <li>● Complain frequently about diet, exercise and adverse medication events.</li> <li>● Exhibit low levels of compliance and poor glycaemic control</li> <li>● Require much family support and make frequent use of healthcare professionals</li> <li>● Diabetes is perceived as an undeserved burden</li> </ul> <p><b>Disheartened</b></p> <ul style="list-style-type: none"> <li>● Low motivation to change lifestyle, erratic effort</li> <li>● May shown obsession with diet but easily discouraged</li> <li>● Complain frequently of adverse events with medication</li> <li>● Overweight and low motivation are key considerations for management</li> <li>● Diabetes is not perceived as a significant burden as effort is limited.</li> </ul> <p><b>Overwhelmed</b></p> <ul style="list-style-type: none"> <li>● Express anxiety, fear, depression and/or guilt at diagnosis</li> <li>● Are often obsessed with diet, but have great difficulty in adhering to a diet plan</li> <li>● Exhibit poor medication adherence</li> <li>● Involve family and friends significantly in their disease experience</li> <li>● Express high levels of dissatisfaction</li> <li>● Require active support to manage their health</li> </ul>	<p>Mou S et al (2010) Prevalence of non-diabetic renal disease in patients with type 2 diabetes. <i>Diabetes Res Clin Pract</i> <b>87</b>: 354–9</p> <p>NICE (2009) <i>Type 2 Diabetes: The Management of Type 2 Diabetes. 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Available at: <a href="http://cme.medscape.com/viewarticle/578214">http://cme.medscape.com/viewarticle/578214</a> (accessed 10.01.12)</p> <p>Pala L et al (2010) Relationship between GLP-1 levels and dipeptidyl peptidase-4 activity in different glucose tolerance conditions. <i>Diabet Med</i> <b>27</b>: 691–5</p> <p>Piya MK et al (2010) Emerging treatment options for type 2 diabetes. <i>Br J Clin Pharmacol</i> <b>70</b>: 631–44</p> <p>Sampanis C (2008) Management of hyperglycemia in patients with diabetes mellitus and chronic renal failure. <i>Hippokratia</i> <b>12</b>: 22–7</p> <p>Sequist ER, Ibrahim HN (2010) Approach to the patient with type 2 diabetes and progressive kidney disease. <i>J Clin Endocrinol Metab</i> <b>95</b>: 3103–10</p> <p>Seck T et al (2010) safety and efficacy of treatment with sitagliptin or glipizide in patients with type 2 diabetes inadequately controlled on metformin: a 2-year study. <i>Int J Clin Pract</i> <b>64</b>: 562–76</p> <p>Selvin E et al (2010) Glycated hemoglobin and the risk of kidney disease and retinopathy in adults with and without diabetes. <i>Diabetes</i> <b>32</b>: 298–305</p> <p>Sinclair AJ et al (2010) Caring for older adults with diabetes mellitus: characteristics of carers and their prime roles and responsibilities. <i>Diabet Med</i> <b>27</b>: 1055–9</p> <p>Solomon TP et al (2010) Improved pancreatic beta-cell function in type 2 diabetic patients after lifestyle-induced weight loss is related to glucose-dependent insulinotropic polypeptide. <i>Diabetes Care</i> <b>33</b>: 1561–6</p> <p>Speight J et al (2009) Not all roads lead to Rome – a review of quality of life measurement in adults with diabetes. <i>Diabet Med</i> <b>26</b>: 315–27</p> <p>Tsitouras PD (2010) Microangiopathic complications of diabetes: diabetic peripheral neuropathies. <i>Clin Geriatr</i> <b>18</b>: 41–4</p> <p>VADT Investigators (2009) Glucose control and vascular complications in veterans with type 2 diabetes. <i>N Engl J Med</i> <b>360</b>: 129–39</p> <p>van Bastelaar KMP et al (2008) Diabetes-specific emotional distress mediates the association between depressive symptoms and glycaemic control in type 1 and type 2 diabetes. <i>Diabet Med</i> <b>27</b>: 798–803</p> <p>van Elderen SG et al (2010) Progression of brain atrophy and cognitive decline in diabetes mellitus: a 3-year follow-up. <i>Neurology</i> <b>75</b>: 997–1002</p> <p>Vedhara K et al (2004) Habitual prospective memory in elderly patients with type 2 diabetes: implications for medication adherence. <i>Psychol Health Med</i> <b>9</b>: 17–27</p> <p>Verhaegen P et al (2003) Relation between cardiovascular and metabolic disease and cognition in very old age: cross-sectional and longitudinal findings from the Berlin Aging Study. <i>Health Psychol</i> <b>22</b>: 559–69</p> <p>Vischer UM et al (2010) The high prevalence of malnutrition in elderly diabetic patients: implications for anti-diabetic drug treatments. <i>Diabet Med</i> <b>27</b>: 918–24</p> <p>Whisman MA (2010) Loneliness and the metabolic syndrome in a population-based sample of middle-aged and older adults. <i>Health Psychol</i> <b>29</b>: 550–4</p> <p>Wild SH et al (2004) Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Response to Rathman and Giani. <i>Diabetes Care</i> <b>27</b>: 2569</p> <p>Zeber J, Parchman ML (2010) Cardiovascular disease in type 2 diabetes: attributable risk due to modifiable risk factors. <i>Can Fam Physician</i> <b>56</b>: e302–7</p>

**Appendix 2:** The diabetes annual review guide (under development).

<b>Name:</b>		<b>DOB:</b>	
<b>Age:</b>		<b>BMI:</b>	
<b>Duration DM:</b>			
<i>HbA<sub>1c</sub>:</i>	<i>CKD</i>	<i>BP:</i>	<i>Falls</i>
<i>Chol:</i>	<i>CVD</i>		<i>DR</i>
<i>LDL:</i>	<i>Retinal Screening</i>		<i>Hypos</i>
<i>HDL:</i>	<b>Current Medication</b>		<i>Depression</i>
<i>Trigs:</i>	<b>Repeat Medication</b>		<i>Cognition</i>
<i>LFT:</i>	<i>ACEi/ARB</i>		<i>MMSE</i>
<i>TFT:</i>	<i>Statin</i>		<i>Frail</i>
<i>Creat:</i>	<i>Anticoag</i>		<i>Other</i>
<i>eGFR:</i>	<i>Metformin</i>		
<i>Microalb:</i>	<i>Sulphonylurea</i>		<b>Glycaemic target risk review</b>
<i>ACR:</i>	<i>Thiazolidinedione</i>		<b>Risk from intensive control</b>
<i>Hb:</i>	<i>Other antidiabetes drug</i>		<b>Benefit from intensive control</b>
<i>Other:</i>	<i>Beta blocker</i>		
	<i>Other</i>		<b>Target HbA<sub>1c</sub></b>
	<b>Drug alerts</b>		

*HbA<sub>1c</sub> = glycated haemoglobin; Chol = cholesterol; LDL = low-density lipoprotein; HDL = high-density lipoprotein; Trigs = triglycerides; LFT = liver function tests; TFT = thyroid function tests; Creat = creatinine; eGFR = estimated glomerular filtration rate; Microalb = microalbuminuria; ACR = albumin/creatinine ratio; Hb = haemoglobin; CKD = chronic kidney disease; BP = blood pressure; CVD = cardiovascular disease; ACEi/ARB = angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; Anticoag = anticoagulant; DR = diabetic retinopathy; Hypos = hypoglycaemic events; MMSE = Mini-Mental State Examination; BMI = body mass index.*

The guide is being developed to enable some elements to be captured electronically (name, date of birth, BMI, latest blood test results, latest blood pressure, and current medication with dosage). The guide can be printed at this point. The clinician may annotate the guide to indicate existent cardiovascular disease, chronic kidney disease stage, most recent retinal screening date and outcome, drug alerts and improvement /deterioration in blood test results. During the consultation further annotation by hand will demonstrate evaluation of psychosocial and functional elements, and the rationale for the final decision regarding target HbA<sub>1c</sub> can be indicated. The guide can be scanned into the patient's computer record as evidence of the decision-making process.

**Appendix 3:** An example of the diabetes annual review guide in use.

<b>Name:</b>	-----	<b>DOB:</b>	--/--/----
<b>Age: 79</b>	<b>BMI: 32.6</b>	<b>Duration DM: 11yrs</b>	
<i>HbA<sub>1c</sub>: 6.4</i> <i>Chol: 3.3</i> <i>LDL:</i> <i>HDL: 1.1</i> <i>Trigs:</i> <i>LFT:</i> Plasma albumin level 40 Plasma total protein 73 Plasma globulin level 33 Plasma alkaline phosphatase level 91 Plasma total bilirubin level 7 Plasma alanine aminotransferase level 13  <i>TFT:</i> Plasma free T4 level 14.3 Plasma TSH level 2.74  <i>Creat: 164</i>  <i>eGFR: 35 stable</i>  <i>Microalb: 42.2 ^</i>  <i>ACR: 10.8 ^</i>  <i>Hb: 12.6 stable</i>  <i>Other:</i>	<i>CKD yes</i> <i>BP: 125/61</i>  <i>CVD angina , AF</i>  <i>Retinal Screening 9/09 Normal</i>	<i>Falls no</i>  <i>DR no</i>  <i>Hypos no</i>  <i>Depression nil</i>  <i>Cognition ok</i>  <i>MMSE x</i>  <i>Frail moderately</i>  <i>Other</i>	
	<b>Current Medication</b>		<b>Glycaemic target risk review</b>
	<b>Repeat Medication</b> <b>ISOSORBIDE MONONITRATE tabs 20mg TAKE ONE TWICE DAILY</b>  <b>PIOGLITAZONE tabs 60mg TAKE 1 DAILY</b>  <b>Diabur Test 5000 strips [ROCHE DIAG] TEST TWICE DAILY</b>  <b>GLYCERYL TRINITRATE cfc free pump spray 400micrograms/dose ONE PUFF AS NEEDED</b>  <b>DIGOXIN tabs 250micrograms ONE to be taken DAILY</b>  <b>WARFARIN SODIUM tabs 3mg As Directed by Dr</b>  <b>WARFARIN SODIUM tabs 1mg As Directed by Dr</b>  <b>SIMVASTATIN tabs 20mg ONE to be taken at bedtime</b>  <b>NICORANDIL tabs 20mg ONE to be taken TWICE daily</b>  <b>RAMIPRIL caps 10mg TAKE ONE DAILY</b>  <b>TAMSULOSIN HCl mr cap 400micrograms TAKE ONE DAILY</b>  <b>GLICLAZIDE mr tab 30mg 3 EVERY DAY</b>  <b>Drug Alerts</b>	<b>Risk from intensive control</b>  <b>high</b>	<b>Benefit from intensive control</b>  <b>intermediate</b>
		<b>Target HbA<sub>1c</sub></b>  <b>7.0%</b>  <b>Reduce medication- Pio to 30 mg and review 3m. Check for hypos with gliclazide Monitor CKD.</b>	

*HbA<sub>1c</sub>* = glycated haemoglobin; *Chol* = cholesterol; *LDL* = low-density lipoprotein; *HDL* = high-density lipoprotein; *Trigs* = triglycerides; *LFT* = liver function tests; *TFT* = thyroid function tests; *T4* = thyroxine; *TSH* = thyroid-stimulating hormone; *Creat* = creatinine; *eGFR* = estimated glomerular filtration rate; *Microalb* = microalbuminuria; *ACR* = albumin/creatinine ratio; *Hb* = haemoglobin; *CKD* = chronic kidney disease; *BP* = blood pressure; *CVD* = cardiovascular disease; *AF* = atrial fibrillation; *DR* = diabetic retinopathy; *Hypos* = hypoglycaemic events; *MMSE* = Mini-Mental State Examination; *Pio* = pioglitazone.