Overlooking the evidence? Changes to QOF targets for 2009

Martin Hadley-Brown

Martin Hadley-Brown is a GP in Thetford, Norfolk and Chair of the Primary Care Diabetes Society.

The British Medical Association (BMA) and the Department of Health (DH) have recently announced their agreed changes to the general practice Quality and Outcomes Framework for the 2 years from 2009–2011 (NHS Employers and the General Practitioners Committee [GPC], 2008). Arguably, the biggest impact of all the clinical changes will be those made in the diabetes domain where the targets for achievement of HbA_{1c} results have been comprehensively revised. Unfortunately, in my view, it appears that the BMA negotiators failed to seek current advice from clinical leaders in either hospital or community diabetes practice, and it seems an inevitable assumption that the DH, perhaps urged on by other pressure groups determined to see tighter targets set for clinicians, set the agenda.

Changes to the QOF targets

So, from Spring 2009, the existing glycaemic control targets, which previously awarded a maximum of 11 points, for up to 90% of people with diabetes being recorded with an HbA_{1c} <10%, plus a further 17 points for up to 50% returning HbA_{1c} results of <7.5%, will be replaced. The new indicators award the previous 17 points for up to 50% of patients' results being 7.0% or less, create a new indicator worth 8 points for up to 70% of results being 8% or less, and replace the 10% indicator with one at 9%, maintaining the upper threshold at 90% and reducing the available points by one to 10. There are no achievement points for HbA_{1c} scores over 9% (NHS Employers and the GPC, 2008).

Strong representations to add an indicator that recognises not just absolute HbA_{1c} results but year-on-year improvement, have once again been rejected. This is despite the universal recognition that exists among those with an interest and knowledge in diabetes that significant reduction in HbA_{1c} results in impressive relative risk reductions from whatever starting point and that absolute microvascular risk reductions are actually higher with, for example, a reduction in HbA_{1c} from 11% to 10% than from 7.5% to 6.5% (Stratton et al, 2000).

Recent evidence regarding glycaemic control

This largely unexpected change in the targets and indicators comes at the end of a year when significant new evidence from a number of sources has become available to inform our best clinical practice regarding glycaemic management.

The UKPDS

As long ago as 1998, the UKPDS (United Kingdom Prospective Diabetes Study) provided the definitive answer that improved glycaemic control led to reduced risk of microvascular complications in type 2 diabetes including nephropathy, retinopathy and neuropathy (UKPDS Group, 1998a). It did not provide an answer as to whether macrovascular complications such as myocardial infarction (MI), stroke or peripheral vascular disease could be reduced by lowering HbA_{1c}, although subanalyses of UKPDS as well as other trials have convincingly shown the benefits of optimising blood pressure control and minimising lipid levels using statins in people with diabetes (Colhoun, 2004; UKPDS Group, 1998b). Other evidence exists showing that cardiovascular risk increases with raised HbA_{1c} levels, probably even those below the levels seen in people with diabetes (Khaw et al, 2001); what was missing was the demonstration that our current treatments could reduce macrovascular risk at the same time as the HbA_{1c}.

The recently released extension report from the UKPDS may have moved us closer to that answer (Holman et al, 2008). Following up patients from the original 15-year trial, who were, remember, recruited near to the diagnosis of their diabetes, this latest UKPDS publication shows the outcome as far as a maximum of 25 years of people who have now been out of the trial for over 10 years. Two key points emerge. First, that despite the HbA_{1c} readings of the "intensively controlled" and "standard control" groups coming together within a year of the trial's end, and not diverging again, the benefits of reduced complications for the originally better controlled group are still evident 10 years later. This is being

dubbed the "legacy effect", and describes the apparent long-term benefit of optimal (by the standards of the time) glycaemic control from the time of diagnosis.

The second point is that the reduction in MI incidence in the more tightly controlled group, which previously just failed to reach statistical significance despite showing a 16% relative risk reduction (UKPDS Group, 1998a), not only persists, but now *does* reach statistical significance as a result of the longer timescale (Holman, 2008). So, UKPDS continues to inform our practice.

This incarnation of the data would suggest that we should be aiming for optimal control of biochemical parameters from the earliest possible stage of diagnosed diabetes, and that by doing so, people with diabetes can minimise their risks, both of microvascular and macrovascular morbidity and mortality. This, of course, is not the whole story. (I will not here attempt to touch on prevention of diabetes in the first place – probably a far greater, tougher challenge, but one that we will have to face.)

On the face of it, the UKPDS findings just described, together with European Association for the Study of Diabetes (EASD) and American Diabetes Association (ADA) targets for glycaemic control of 7.0% (Nathan et al, 2009), and the International Diabetes Federation (IDF) target of 6.5% (IDF Clinical Guidelines Workforce, 2005), would seem to support the new QOF targets which I started by criticising.

The latest NICE guidelines for management of type 2 diabetes, together with the "newer agents update" currently available in draft form (available at http://www.nice.org.uk/Guidance/CG/Wave16/3), advocate an individualised HbA_{1c} level, with suggested targets of 6.5 and 7.5% specified at different stages of the blood glucose lowering algorithm (National Collaborating Centre for Chronic Conditions, 2008).

So what is my objection? Two recent trials have shown that there may be a high price to pay for the indiscriminate pursuit of tight glycaemic control.

ACCORD, ADVANCE and VADT

The ACCORD (Action to Control Cardiovascular Risk in Diabetes) study specifically set out to discover whether cardiovascular events were reduced by the achievement of optimal HbA_{1c} in people with long-established diabetes at "high vascular risk". Median HbA_{1c} levels of 6.4% and 7.5% were achieved in the comparator groups and the trial was stopped early after a mean follow-up of 3.5 years because of a significantly higher death rate in the tight control group. It equated to one extra death per 95 patients over the 3.5 years (ACCORD Study Group, 2008).

The VADT (Veterans Administration Trial; Abraira, 2008), another large study reporting in 2008 in the US, also looked at the levels of control we are being incentivised to pursue. The "intensive control" group achieved HbA_{1c} levels of 6.9% with the comparator group at 8.4%, and again the study contained a population with long-established rather than newly diagnosed diabetes. Cardiovascular event rates were only one-third of those originally predicted, reflecting modern achievements with lipid and blood pressure control. Tight glucose control only showed benefits in terms of reducing nephropathy, but the headline from this trial was the predictive value of hypoglycaemia for cardiovascular events (Abraira, 2008).

Indeed the link between ACCORD and VADT, while as yet incompletely assessed, is in my experience, widely thought to be the consequences of hypoglycaemia in provoking cardiac and all-cause catastrophe, particularly in people with long-established diabetes whose HbA_{1c} is reduced relatively rapidly. The ADVANCE (Action in Diabetes and Vascular Disease: Preterax and Diamicron MR Controlled Evaluation) trial notably did not replicate the excess death rate of ACCORD, but achieved less-tight control with less hypoglycaemia, and also failed to show a reduction in macrovascular risk over 5 years (ADVANCE Collaborative Group, 2008).

So a pattern may be emerging: the UKPDS follow-up tells us that optimal control from near diagnosis with relatively low hypoglycaemia rates is beneficial. ACCORD and VADT point to possible major dangers of trying to achieve very aggressive glycaemic control in later diabetes, particularly where that involves hypoglycaemia. NICE recognises the appropriateness of aiming for variable targets for different individuals, recommending aiming for an HbA_{1c} of 6.5% from diagnosis but up to 7.5% later. It also advocates the use of sulphonylureas as second-stage agents after metformin, and we know the potential of the former to cause hypoglycaemia if not used with great care and particularly in older people.

"So what is my objection? Two recent trials have shown that there may be a high price to pay for the indiscriminate pursuit of tight glycaemic control."

"It is up to each of us to continue to consider the best interests of each person with diabetes. Optimal diabetes care is neither mechanistic nor often easy."

Did this evidence inform the new QOF targets?

QOF now extends the financial imperative on practices to aim for indiscriminate HbA_{1c}-lowering below 7%, regardless of age or other morbidity. Exception reporting, while available, is monitored by PCTs and its use deprecated as a "cheat". In my view, this QOF change was introduced with minimal, if any, accountable expert advice, and in the face of alternatives that would have been safer and achieved more for those with the least-well controlled diabetes. The typical diabetes practice population has significant numbers of older people with longstanding diabetes and other existing morbidity, as well as its share of newly diagnosed diabetes each year. It appears that different approaches may be appropriate to each. True clinical medicine may be safer than target chasing! It is up to each of us to continue to consider the best interests of each person with diabetes. Optimal diabetes care is neither mechanistic nor often easy. ■

Abraira C (2008) The Veteran's Administration Diabetes Trial (VADT) – Results. Presented on Wednesday 10 September, at: *44th Annual Meeting of the European Association for the Study of Diabetes*. Rome, Italy, 7–11 September

Action to Control Cardiovascular Risk in Diabetes Study Group (2008) *NEJM* **358**: 2545–59

Action in Diabetes and Vascular Disease: Preterax and Diamicron MR Controlled Evaluation Collaborative Group (2007) *NEJM* **358**: 2560–72

Colhoun HM, Betteridge DJ, Durrington PN et al (2004) **364**: 685–96

Holman RR, Paul SK, Bethel MA (2008) *NEJM* **359**: 1577–89

International Diabetes Federation Clinical Guidelines Workforce (2005) *Global guideline for type 2 diabetes*. International Diabetes Federation, Brussels

Khaw K-T, Wareham N, Luben R et al (2001) *BMJ* **322**: 15–8

Nathan DM, Buse J Holman RR et al (2009) *Diabetes Care* **32**: 193–203

National Collaborating Centre for Chronic Conditions (2008) *Type 2 diabetes: national clinical guideline for management in primary and secondary care (update)*. Royal College of Physicians, London

NHS Employers and the General Practitioners Committee (GPC; 2008) *QOF Changes and New Indicators for 2009/10* Available at: http://www.nhsemployers.org/pay-conditions/primary-890.cfm (accessed 06.01.09)

Stratton IM, Adler AI, Neil HA et al (2000) *BMJ* **321**: 405–12

UK Prospective Diabetes Study Group (UKPDS; 1998a) *Lancet* **352**: 837–53

UKPDS Group (1998b) *BMJ* **317**: 703–13

QOF results 2007/08

Table 1. UK disease prevalence statistics as published in the QOF for 2004/05, 2005/06, 2006/07 and 2007/08. (all data in %)

Disease Area	England*				Wales**				Northern Ireland***				Scotland****			
	04/05	05/06	06/07	07/08	04/05	05/06	06/07	07/08	04/05	05/06	06/07	07/08	04/05	05/06	06/07	07/08
Coronary heart disease	3.6	3.6	3.5	3.5	4.3	4.3	4.3	4.2	4.1	4.2	4.2	4.1	4.5	4.5	4.5	4.5
Stroke	1.5	1.6	1.6	1.6	1.8	1.9	2.0	1.9	1.4	1.6	1.6	1.7	1.7	1.9	2.0	2.0
Hypertension	11.3	12.0	12.5	12.8	12.5	13.4	14.3	14.5	10.0	11.1	11.7	11.9	11.7	12.4	12.5	13.1
Diabetes	3.3	3.6	3.7	3.9	3.8	4.1	4.2	4.4	2.8	3.1	3.1	3.3	3.3	3.4	3.5	3.7

Table 2. QOF points achieved for 2004/05, 2005/06, 2006/07 and 2007/08.

	England*				Wales**				Northern Ireland***				Scotland****			
	04/05	05/06	06/07	07/08	04/05	05/06	06/07	07/08	04/05	05/06	06/07	07/08	04/05	05/06	06/07	07/08
Total QOF points (%)	91.3	96.2	95.5	96.8	90.2	95.6	94.9	97.3	94.2	97.9	97.8	98.7	92.5	97.7	97.1	98.2
Average QOF points/practice	958.7	1010.5	954.5	968.0	947.1	1003.3	948.6	973.5	989.0	1027.6	977.8	986.7	971.3	1026.2	971.2	982.2
Diabetes points achieved (%)	93.2	97.4	97.5	98.0	93.3	97.5	97.5	98.6	95.7	98.3	98.8	99.0	96.0	98.5	98.9	99.0
CHD points achieved (%)	95.3	98.3	98.4	98.9	93.4†	97.3†	98.1	99.2	97.0	99.2	99.5	99.7	95.0	98.7	99.0	99.5
Hypertension points achieved (%)	94.4	98.1	98.3	98.8	93.7	97.7	97.9	98.9	97.9	99.6	99.5	99.7	94.8	99.0	99.0	99.5
Stroke and TIA points achieved (%)	92.0	97.2	97.3	98.2	91.2	96.8	97.2	98.6	95.9	99.1	99.2	99.5	94.3	98.9	98.6	99.3

†*Includes left ventricular dysfunction. CHD: coronary heart disease; TIA: transient ischaemic attack*

**QOF data for England available at: http://www.ic.nhs.uk/statistics-and-data-collections/audits-and-performance/the-quality-and-outcomes-framework (accessed 19.12.08);*
***QOF data for Wales available at: http://www.statswales.wales.gov.uk/TableViewer/tableView.aspx?ReportId=4111 (accessed 19.12.08);*
****QOF data for Northern Ireland available at: http://www.dhsspsni.gov.uk/qof_data (accessed 19.12.08);*
*****QOF data for Scotland available at: http://www.isdscotland.org/isd/3305.html (accessed 19.12.08).*