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Developing a Quality Measure for Clinical Inertia in Diabetes Care

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Objective. To develop a valid quality measure that captures clinical inertia, the failure to initiate or intensify therapy in response to medical need, in diabetes care and to link this process measure with outcomes of glycemic control.
Data Sources. Existing databases from 13 Department of Veterans Affairs hospitals between 1997 and 1999.
Study Design. Laboratory results, medications, and diagnoses were collected on 23,291 patients with diabetes. We modeled the decision to increase antiglycemic medications at individual visits. We then aggregated all visits for individual patients and calculated a treatment intensity score by comparing the observed number of increases to that expected based on our model. The association between treatment intensity and two measures of glycemic control, change in HbA1c during the observation period, and whether the outcome glycosylated hemoglobin (HbA1c) was greater than 8 percent, was then examined.
Principal Findings. Increases in antiglycemic medications occured at only 9.8 percent of visits despite 39 percent of patients having an initial HbA1c level greater than 8 percent. A clinically credible model predicting increase in therapy was developed with the principal predictor being a recent HbA1c greater than 8 percent. There were considerable differences in the intensity of therapy received by patients. Those patients receiving more intensive therapy had greater improvements in control ($p < .001$).
Conclusions. Clinical inertia can be measured in diabetes care and this process measure is linked to patient outcomes of glycemic control. This measure may be useful in efforts to improve clinicians management of patients with diabetes.

Key Words. Diabetes mellitus, outcomes assessment, quality of health care

Central to improving clinical practice is reliable and valid measures of the quality of care. While process and outcome measures each play an important role in quality measurement, it has long been recognized that the development of process measures that are linked to outcomes is an important

health services research goal (Brook, McGlynn, and Cleary 1996). The presence of such links helps validate the process measure and quality improvement resources may be redirected to those processes shown to have the greatest impact on patient outcomes (Hammermeister et al. 1995). Yet establishing links between process and outcome measures in observational studies is not easy and paradoxical results in which more intensive care produces worse outcomes often arise because of confounding by indication (Rubin, Pronovost, and Diette 2001). We now attempt to develop a quality measure for describing clinicians' practices in the pharmacological management of diabetes mellitus and to link this process measures to important intermediate outcomes of glycemic control.

Diabetes is a common medical problem that often has a significant negative impact on a patient's health status. Studies of the quality of diabetes care have frequently demonstrated a wide gap between recommended medical practices and the care that diabetes patients actually receive (Saaddine et al. 2002). This is particularly true for the management of hyperglycemia in which many patients have inadequate glycemic control (O'Connor et al. 1996; Chin et al. 2000; Harris 2000; El-Kebbi et al. 2001). Guidelines published by the American Diabetes Association (2003) identify hemoglobin A1c (HbA1c) of less than 7 percent as desirable and have long recommended that clinicians take action when HbA1c levels are greater than 8 percent. Diabetes treatment protocols typically encourage stepped intensification of pharmacological therapy until goals for glycemic control are achieved (Mazze et al. 1994; Abraira et al. 1995; El-Kebbi et al. 1997). Yet medical practice studies find that clinicians often do not follow these

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recommendations, failing to increase antiglycemic medications despite persistently elevated blood glucose and HbA1c levels (El-Kebbi et al. 1997; Wetzler and Snyder 2000).

Recently, the term clinical inertia has been proposed to define the phenomenon by which health care providers fail to initiate or intensify therapy when indicated (Phillips et al. 2001). Moreover, it is increasingly recognized as among the most important barriers to achieving control of chronic medical conditions (Javors and Bramble 2003). Efforts to improve care by identifying and minimizing clinical inertia may then have a significant impact in enhancing outcomes for patients with diabetes. Before this can be accomplished, however, one must be able to measure clinical inertia.

Two measures of clinical inertia in diabetes care have been used previously: the proportion of patients having an intensification of pharmacological therapy at visits with elevated measures of glycemic control (El-Kebbi et al. 1997; Wetzler and Snyder 2000) and the proportion of patients with elevated HbA1c levels on a greater-than-starting dose of medications (Grant et al. 2002). These measures have typically examined only a short time-frame and do not reflect the many clinical factors that influence treatment decisions. Furthermore, they have not been linked to outcomes, perhaps because such attempts are likely to suffer from confounding by indication (Rubin, Pronovost, and Diette 2001). Confounding by indication refers to the fact that poor glycemic control is simultaneously an indication for treatment intensification and a predictor of poor outcome. Thus, unless we adjust for variations in patient status during the course of treatment, we are likely to see more intense therapy associated with worse glycemic control. Recently, we proposed an alternate approach to measuring intensity of pharmacological therapy in hypertension care that compares the observed number of increases in therapy over an extended time period to the number predicted by empirically modeling factors that affect the probability of an increase in therapy at individual visits (Berlowitz et al. 1998). Improved performance on this process measure was associated with better blood pressure control, reversing the negative association (likely because of confounding by indication) that appeared when predicting outcomes from aggregated measures of treatment intensity adjusted only for baseline patient characteristics. This intensity score approach has since been placed within the general framework of causal modeling for the effect of a time-varying treatment via structural nested mean models (Brumback et al. 2003).

We now develop a similar quality measure for measuring clinical inertia in diabetes care by evaluating the pharmacologic management of diabetes in

patients with ready access to clinicians and medications through the Department of Veterans Affairs (VA). To accomplish this, we perform the following three tasks. First, we identify factors associated with the decision to increase antiglycemic medications at individual patient care visits. Second, using the resulting model, we describe the intensity of antiglycemic medication therapy received by patients over an approximately 16-month period. Finally, we examine the link between this process measure and outcomes by determining whether those patients receiving more intensive therapy had improved glycemic control.

METHODS

Data Sources

Study data were obtained from two sources. First, we used two national VA databases, the Outpatient Clinic File (OPC) and the Patient Treatment File (PTF), which contain information on all outpatient and inpatient medical encounters. This included diagnoses described as ICD-9-CM codes, dates of encounters, and types of outpatient clinic visits. Second, we used the Computerized Patient Record System, available through each VA medical center, to obtain pharmacy records and the results of laboratory studies. Pharmacy records described the name, dosage, and refill date for each dispensed medication and diabetes-related equipment. Because these treatments are available to eligible veterans either for free or a small copayment, the VA is likely to be their primary source for diabetes-related medications.

Study Subjects

We studied veterans with diabetes mellitus receiving regular medical care at 13 VA medical centers located in two Veterans Integrated Service Networks, New England and Florida–Puerto Rico. Study patients met the following criteria. First, they had to have at least two primary care visits during the 6-month period beginning October 1, 1997. We randomly selected one primary care visit from this period as the "index" visit. Second, patients had to have at least one primary care visit between 1 and $1\frac{1}{2}$ years after the index visit. If more than one such visit was available, we randomly selected one as the "outcome" visit. Third, patients needed a diagnosis of diabetes mellitus. Diabetes was considered present if a diabetes ICD-9-CM code (250.x [diabetes mellitus], 357.2 [polyneuropathy in diabetes], or 362.0x [diabetic retinopathy]) was recorded in VA databases on two occasions at least 7 days apart during the

1-year period beginning April 1, 1997. At least one diabetes diagnosis had to be from an outpatient encounter and at least one had to be no later than the index visit. Ninety-five patients with more than 40 visits per year were excluded from this sample. Since onset of diabetes as a child would preclude military service, most patients had type 2 diabetes.

Variable Selection and Statistical Analyses

Analyses were performed in three steps. For the initial analytic step, we considered each individual visit contributed by patients in our study sample. We used visits as the focus of our analysis because we consider each visit as an opportunity for the clinician to assess the patient and make an informed judgment about whether or not to alter medication therapy. We constructed a model to predict the probability that an individual patient-care visit would result in an increase in antiglycemic medications based on characteristics at the time of that visit. We considered only those visits in which diabetes management was likely to occur, specifically visits to all primary care clinics and selected subspecialties of internal medicine such as diabetes/endocrinology. These visits were identified using the clinic stop variable from the OPC. An increase in antiglycemic medications was considered present at a visit if a new antiglycemic medication was started or the dosage of an existing medication was increased. Since increases in medications identified in the pharmacy database may not occur on the same day as a visit, we assigned any increase to the prior visit if it was within 14 days after the visit; otherwise it was assigned to the next visit. We chose a 14-day window after examining dates of increases in relation to visit dates. Use of a window acknowledges that clinicians may order laboratory tests at a visit and then subsequently contact a patient to change his medications. Each visit was thus classified as antiglycemic medication "increase" or "no increase." Visits occurring after a patient was prescribed an insulin "sliding scale," as indicated by this specific designation in the *sig* statement of the prescription, were not considered as these patients were likely adjusting their own medications. This was present in 594 patients.

We used clinical judgment to identify available factors likely to influence the decision to increase antiglycemic medication therapy. These potential predictors included demographic characteristics of the patients; results of laboratory tests such as measures of glycemic control, lipid levels, renal function, and urinalysis; measures of health care utilization including recent hospitalizations and the time since the patient's last outpatient visit; pharmacy inform-

ation including steroid use, insulin, and prescription of glucose monitoring equipment; and diagnoses. Diagnoses were used to identify the presence of specific diabetic complications (neuropathy, retinopathy, nephropathy, or peripheral vascular disease), other cardiovascular risk factors (hypertension, hyperlipidemia, tobacco abuse disorder), presence of any psychiatric or substance abuse disorders, and a summary measure of comorbidity burden as described by the Deyo modification of the Charlson index (Charlson et al. 1987; Deyo, Cherkin, and Ciol 1992). Diagnoses were considered present if there was a single ICD-9-CM code indicating the appropriate condition in the year prior to the index visit, an approach that we have previously found to have good agreement with medical records (Borzecki et al. 2004). Predictors were coded in alternate ways before deciding on the exact variation used in modeling. For example, for HbA1c at the time of a visit we considered different ways of coding whether any value was available, how long since the last determination, the results of the most recent determination, and results of the second most recent determination. We settled on eight different categories of availability and level (not available, ≤ 6, > 6 but ≤ 7, > 7 but ≤ 8, > 8 but ≤ 8.5, > 8.5 but ≤ 9, > 9 but ≤ 10, and > 10), in addition to a separate variable for time since last determination. Of note, VA databases do not accurately differentiate fasting from nonfasting laboratory values.

We modeled the decision to increase antiglycemic medications at a visit using classification and regression trees (CART) (Breiman 1984), a recursive partitioning algorithm as implemented in *S-plus* software version 3.3. This procedure repeatedly splits the data to create a division into subgroups of visits that are internally similar with respect to their probability of a medication increase. At each step, the procedure splits an existing group into two based on the values of a single independent variable. The variable to be split is chosen to result in the greatest degree of separation in the outcome. The model-estimated probability of a medication increase at a visit is the empirical frequencies of the outcome in each terminal group. The choice of the number of terminal groupings was guided by a cross-validation procedure in which we divided the sample into five equal-sized subsets. Then, for each possible number of terminal groups, ranging from 2 to 150, a model of that size was fit to each combination of four of the five subsets, and applied to the remaining subset. Based on the actual outcomes and estimated probabilities, *c*-statistics were calculated (Altham 1973). The final number of terminal groupings was chosen by examining models with high cross-validated *c*-statistics that conform to clinical judgments about appropriate explanatory variables.

We were concerned that many increases in insulin therapy might not be recorded in the pharmacy database. Thus, we initially developed separate models for visits in which the patient was receiving insulin and for noninsulin visits. However, as the models were similar, and increases in insulin therapy were frequently evident in the pharmacy database, we combined all visits to develop a single model.

For the next step in our analysis, we aggregated information from each individual visit for a patient to create a patient-specific measure of the intensity of medication therapy. This measure is norm-based in that it compares the intensity any given patient receives relative to the experience of other patients. We defined treatment intensity as a ratio with the numerator equal to the actual number of medication increases from the index to outcome visit minus the expected number of increases as predicted by the model for each visit. The denominator was the total number of visits. Scores on this treatment intensity measure must lie between -1 and $+1$; positive values indicate more increases in therapy than expected.

In the final step, we examined the association between this process measure describing treatment intensity and two outcome measures of glycemic control. We used a linear regression model to explain the change in HbA1c levels between the index and outcome visit. The index visit HbA1c was the determination closest in date to the index visit during the 6 months surrounding the visit. As laboratory data after the outcome visit were not available, the outcome visit HbA1c was the determination closest to the outcome visit during the 6 preceding months. We also used a logistic regression model to explain the dichotomous outcome of "outcome visit HbA1c >8 percent" yes/no, adjusting for index visit HbA1c. For both models, we adjusted for other variables likely associated with glycemic control (Zhang et al. 2000). Patients without a HbA1c determination for both the index and outcome visit were excluded from these regression analyses.

RESULTS

The study sample consisted of 23,291 patients with 266,309 visits. Patient characteristics are described in Table 1. Nearly 29 percent of patients were on insulin at baseline, and 27 percent were on insulin for the entire study period. Among the 15,437 patients with a HbA1c determination at the index visit, the mean was 7.8 ± 1.9 percent; 39.1 percent had a value above 8 percent. Patients were frequent utilizers of VA health care. Yet following 15.8 ± 1.9

Table 1: Characteristics of the Study Patients*

	Total Sample (N= 23,291)	*With Index and Outcome HbA1c (N= 12,523)*	*Without Both Index and Outcome HbA1c (N= 10,768)*	*p-Value*†
Age (years)	65.3 ± 10.4	65.3 ± 10.1	65.3 ± 10.8	.69
Male gender (%)	97.2	97.4	97.0	.05
Diabetic complication (%)	41.6	43.1	39.7	<.0001
Additional cardiovascular risk factor (%)	78.2	79.5	76.7	<.0001
Charlson index‡	1.09 ± 1.57	1.06 ± 1.52	1.14 ± 1.63	<.0001
On insulin at baseline (%)	28.6	32.2	24.5	<.0001
Index HbA1c§	7.8 ± 1.9	7.8 ± 1.9	7.6 ± 1.9	—
Index HbA1c>8% (%)§	39.1	39.8	36.0	—
Outcome HbA1c¶	7.8 ± 1.6	7.8 ± 1.6	7.7 ± 1.7	—
Outcome HbA1c>8% (%)¶	39.4	40.3	36.6	—
No. of visits	11.4 ± 6.6	12.2 ± 6.7	10.6 ± 6.4	<.0001
Months between index and outcome visits	15.8 ± 1.9	15.9 ± 1.6	15.7 ± 2.2	<.0001

*Continuous variables described as mean ± standard deviation.

†*p*-value compares group with index and outcome HbA1c to group without.

‡Charlson index excludes diabetes.

§*N*= 15,437 for total sample and *N*= 2,914 for without both sample.

¶*N*= 16,800 for total sample and *N*= 4,277 for sample without both.

HbA1c, hemoglobin A1c.

months of care, with 8.8 ± 5.1 visits per year, the mean HbA1c level among the 16,800 patients with a determination was unchanged and 39.4 percent had a value above 8 percent.

An increase in antiglycemic medication therapy occurred at 9.8 percent of visits. An increase occurred at 13.1 percent of the visits when a patient was on insulin and 7.8 percent of visits with no insulin therapy. Insulin was started on 1,884 patients during the study period.

The strongest predictor of an increase was the most recent HbA1c being greater than 8 percent (Figure 1). Other factors associated with an increase included higher serum glucose, longer interval since the last visit (when several visits are close together, an increase is less likely at any individual visit), HbA1c being performed within 3 months, being on insulin, and having received self-monitoring of blood glucose supplies. An increase occurred 32.0 percent of the time among the 10,581 visits in which the most recent HbA1c was greater than 8 percent, that HbA1c had been performed within the past

Figure 1: Model Used to Describe Factors at Individual Visits Associated with the Decision to Increase Antiglycemic Medications

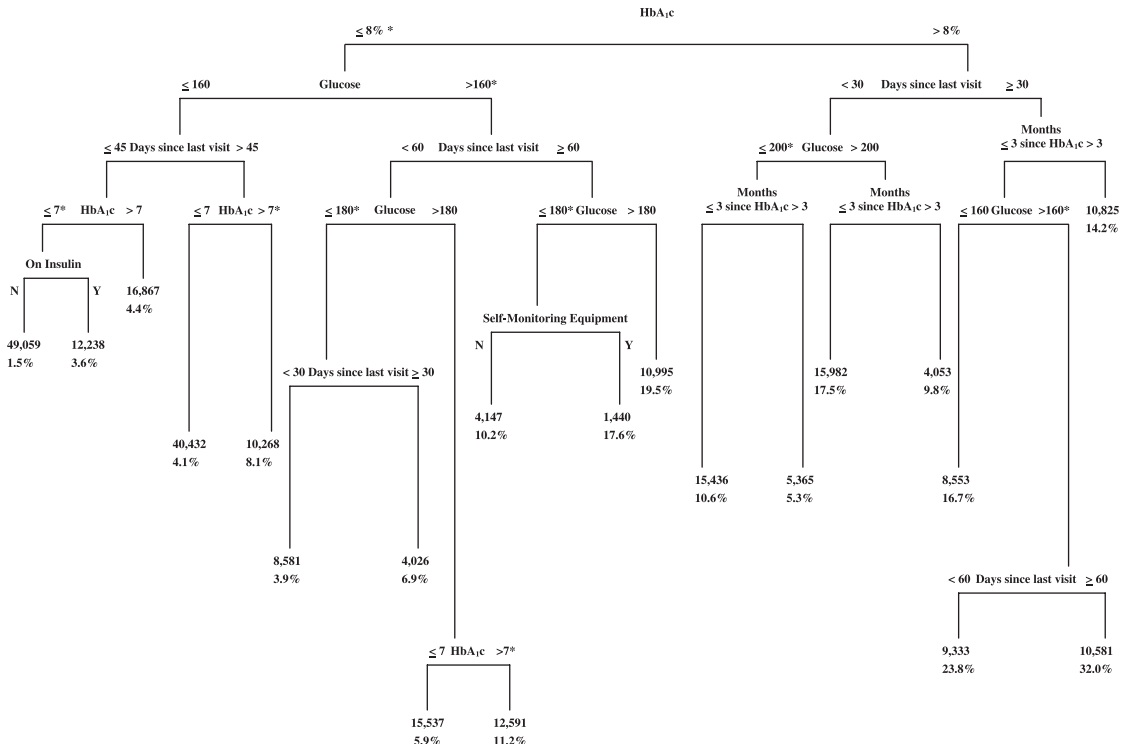

Table 2: Multivariate Regression Models* Relating Intensity of Antiglycemic Medication Therapy to Glycemic Control

	Change in HbA1c		*Outcome HbA1c > 8%*	
Variable	*Coefficient*	*p-Value*	*Odds Ratio*	*95% CI*
Intensity of therapy (per increase of 0.1)	− 0.064	<.0001	0.91	0.88, 0.98
Index visit HbA1c	—	—	1.76	1.72, 1.81
Age (per increase of 10 years)	0.013	.39	0.78	0.75, 0.81
Male gender	0.147	.13	1.12	0.86, 1.45
Any diabetes complication	− 0.131	<.0001	1.29	1.18, 1.41
Charlson index	0.026	.01	1.02	0.99, 1.05
Psychiatric diagnosis	0.156	.0002	1.03	0.92, 1.15
Alcohol/substance abuse diagnosis	− 0.169	.06	0.79	0.62, 1.00
Oral steroid use	− 0.022	.75	0.83	0.69, 1.00

*A linear model was used when modeling outcome visit HbA1c minus index visit HbA1c; a negative coefficient indicates improved glycemic control. A logistic model was used to predict odds of an outcome HbA1c > 8%; an odds ratio less than 1.0 indicates better control.

HbA1c, hemoglobin A1c; CI, confidence interval.

three months, the serum glucose was greater than 160 mg/dl, and the prior visit was more than 60 days previously. In contrast, an increase occurred at only 1.5 percent of visits when the most recent HbA1c was less than 7 percent, the serum glucose was less than or equal to 160 mg/dl, the prior visit was within the past 45 days, and the patient was not on insulin therapy.

The expected number of increases per patient varied with some patients having all their visits in the group with the lowest predicted probability of an increase (1.5 percent), and others having each visit with a 32 percent predicted probability of an increase. Patient treatment intensity scores also varied among patients with a range from − 0.32 to 0.96. Twenty-eight percent of patients had intensity scores greater than 0.05, values we viewed as more intensive therapy than the norm, and 34 percent had values less than –0.05, indicating less intensive therapy. Mean intensity scores were slightly higher for patients with an index HbA1c > 8 percent compared with those with one < 8 percent (0.02+0.15 versus 0.01+0.11, $p < .001$).

More intensive antiglycemic medication therapy was significantly associated with improved glycemic outcomes. An index and outcome HbA1c was available in 12,523 patients (54 percent of the sample). These patients differed from the 10,768 patients without these determinations (Table 1). Among patients in the highest quintile of treatment intensity scores, with scores greater than 0.09, HbA1c values declined by 0.12 percent between the index

and outcome visit. In patients receiving less intensive therapy, the change in HbA1c values worsened progressively. In those patients in the lowest quintile, with intensity scores less than −0.08, HbA1c values increased by 0.09 percent. Fifty-seven percent of patients in the lowest quintile had an outcome visit HbA1c greater than 8 percent, as compared with 36 percent among the remaining patients. These associations were significant in regression models that adjusted for other patient characteristics (Table 2). Each 0.1 increase in the intensity score, equivalent to one additional visit during which there was an increase in therapy beyond that expected over 10 visits, was associated with an additional 0.06 percent decline in the HbA1c and an odds ratio of 0.91 that the outcome HbA1c would be greater than 8 percent. Regression models were almost identical when limited to the subsample of patients on insulin therapy for the entire study period.

DISCUSSION

Clinical trials have convincingly demonstrated that intensive medication management of diabetes improves glycemic control and prevents the development of microvascular disease (The Diabetes Control and Complications Trial Research Group 1993; U.K. Prospective Diabetes Study [UKPDS] Group 1998). Despite these facts, many patients with diabetes do not achieve recommended levels of glycemic control. Clinical inertia is now recognized as an important barrier contributing to inadequate glycemic control (Phillips et al. 2001). We now propose a quality measure that can be used to measure this phenomenon. This measure works by interpreting the total treatment received in the context of the level of treatment expected as a result of a sequence of conditional decisions. Thus, the same amount of treatment is viewed as more intense for a person with better glycemic control during the treatment period than for a person who was seen repeatedly with poor control.

Our approach to measuring clinical inertia in diabetes care has numerous advantages over past efforts (El-Kebbi et al. 1997; Wetzler and Snyder 2000; Grant et al. 2002). First, by evaluating care over an extended time period, our method recognizes that at any single visit, not increasing pharmacological therapy might be an appropriate action. However, over a longer time period, several increases might be indicated if treatment goals are not met. Second, our approach recognizes that many clinical factors might be considered in the decision to increase therapy, including not only the level of hyperglycemia, but also the recency of these lab tests, the types of therapy

being received, and the time since the previous visit. Third, our measure has face validity in that it is based on widely accepted clinical practice recommendations from the time frame of this study. The strongest predictor of an increase in therapy was a HbA1c greater than 8 percent. This is consistent with the American Diabetes Association recommendations that encourage action when the HbA1c is above this level, and also reflects many clinicians' beliefs that lifestyle modifications will not be sufficient to achieve tight control at these higher levels. Interestingly, HbA1c levels further above 8 percent were not associated with a greater likelihood of an increase although higher blood glucose levels were an important predictor. Fourth, our measure appears to be sensitive to differences in practice. There were large differences in the intensity of therapy that patients received with some patients receiving many more increases in therapy than the norm.

Finally, and most importantly, our process measure was linked to outcomes in that patients who received more intensive management were more likely to achieve better outcomes in glycemic control. We evaluated two measures of glycemic control and found for each that more intensive therapy was associated with significantly better results. Establishing such links between process and outcome measures is an important goal in quality assessment. It further validates our measure of intensity of therapy and suggests that we are capturing an important aspect of care. These results also emphasize that interventions to improve care should focus on the problem of clinical inertia.

Past studies in diabetes care have generally failed to establish such links between process and outcomes (Williams et al. 1967; Romm and Hulka 1979, 1980). Considerable advances have been made since these studies in developing methods, especially those that use a "propensity to receive treatment" score, to reduce bias when using observational studies to determine treatment effects (D'Agostino 1998). Approaches that only adjust for baseline covariates, however, do not work well when examining a sequence of treatment encounters, such as in diabetes care, where there may be time-dependent confounding. A number of analytic strategies have been described for this type of problem (Robins 1997; Robins, Hernan, and Brumback 2000) that recognize the need to interpret the care given at each encounter in the context of the patient's status at that encounter. As recently discussed, our intensity score is a readily interpreted measure in the spirit of G-estimators, which represent the effect of a "generalized treatment regime," for structural nested models (Brumback et al. 2003). Our diabetes intensity of medication therapy measure was able to associate more care with better outcomes, as was a similar intensity measure that we developed in hypertension (Berlowitz et al. 1998). These

findings are consistent with numerous studies showing that normative practice in these areas is insufficiently aggressive. While we cannot prove that the measures are bias-free, they are helpful in addressing the problem of confounding by indication.

Our intensity of therapy measure may be used in a variety of ways. Quality improvement programs in diabetes care could profile clinicians' practices not only on the basis of their glycemic control but also in their pharmacological management of diabetes. Clinicians and settings with higher intensity scores and better glycemic control could be identified for benchmarking. Interventions to improve care could be developed that provide clinicians with feedback on their performance on this measure. Such interventions that focus on how clinicians decide to increase therapy may be particularly successful (Cook et al. 1999) and could be directed at that subset of clinicians whose patients have poor glycemic control in the setting of low intensity therapy. As our intensity measure relies on data available from existing databases, such efforts would be relatively easy to implement. The measure may also be used to study care provided to important subgroups of patients such as ethnic minorities and individuals with mental health illnesses. For example, we recently studied hypertension control in patients with diabetes and found not only did they have worse blood pressure control than patients without diabetes, but they also were receiving less intensive therapy (Berlowitz et al. 2003). Thus, we were able to conclude that poor blood pressure control in patients with diabetes is not solely related to more difficult to treat disease, but also to the fact that clinicians were treating these patients differently.

Our results provide additional evidence regarding the presence of clinical inertia. They are similar to past studies in that many patients had inadequate glycemic control. Over 39 percent had an initial HbA1c greater than 8 percent, and following an additional 16 months of care with more than 11 visits on average, glycemic control in the population was essentially unchanged. Despite less than optimal control, increases in antiglycemic medication therapy were relatively infrequent, occurring in only 9.8 percent of visits. Even among those visits with the greatest indication for an increase, an actual increase occurred only 32 percent of the time. Thus, many opportunities for increasing therapy to achieve better glycemic control were being missed. In deciding on whether to increase therapy, our results suggest that clinicians tend to focus on relatively few factors.

The process measure that we propose is norm-based; it compares clinicians to the usual performance of other providers. While this is often the standard approach when comparing outcomes such as risk-adjusted mortality,

it is used less frequently for process measures that are often absolute. As with many HEDIS-type measures, either the appropriate process is performed or it is not. The specific situation in which performance of the process is indicated is usually well identified, for example "when the HbA1c is elevated at a visit, therapy should be increased." This approach has a number of advantages including being easy to comprehend, indicating specific subgroups in which intensification is indicated, and being less susceptible to gaming by increasing therapy in patients with well-controlled diabetes. However when examining a complex behavior such as medication intensification, which typically takes place over several visits and in which there is significant provider discretion with many factors contributing to the decision, we believe that a norm-based approach has many advantages. Because our approach is norm-based, care is required in using the model to calculate intensity scores for non-VA settings.

Unique to our approach of measuring clinical inertia is that we combine information from multiple visits. We determine an expected probability for an increase in therapy at each individual visit and then sum the expected probabilities over all visits. It is this value we compare with the observed number of increases. In calculating these expected probabilities, we used CART modeling because of our belief that clinicians' actions are very much guided by clusters of signs and symptoms as well as discrete thresholds for action as emphasized in national guidelines. Additionally, CART models are useful for identifying interactions among predictors. However, CART models have also been criticized because subgroups may be difficult to interpret clinically and simple rules may not be uncovered (Marshall 2001). We also evaluated a logistic model and found it had similar discriminative ability as the CART model.

Many factors go into the clinical decision on increasing therapy at an individual visit; our model captures only a few such factors. We cannot determine whether individual decisions to increase or not increase therapy might be appropriate for a patient. Such a determination would require a careful weighing of the risks and benefits of changes as well as patient preferences. This level of detailed information could not be obtained from existing databases. This emphasizes the importance of applying our measure to studying groups of patients rather than the decisions made for individuals.

We did not measure adherence to medications in our study. Clinicians may not be intensifying therapy because of concerns that patients were nonadherent. In at least one small study, however, this was not found to be the case (Javors and Bramble 2003). Further work should examine this issue. It could be that even a stronger link between intensity of therapy and glycemic

control would be demonstrated after considering adherence. However, the fact that we did demonstrate such a link without considering adherence suggests the validity of our approach.

Several additional limitations should be noted. First, our study population was highly selected, consisting mostly of elderly men with type 2 diabetes and access to medical care. Second, we had no information on non-VA medical care. However, most patients are likely to be using the VA for obtaining medications, and the population had high rates of utilization. Third, we relied on pharmacy databases to identify increases in therapy. While these databases are likely to capture most episodes of initiating a new therapy, increases in dosage of a prescribed medication, particularly for insulin, may be missed. Fourth, HbA1c levels were missing for many patients and these patients differed from patients with such information. We cannot be certain how this affected our results. Finally, the magnitude of changes in HbA1c associated with increased treatment intensity were not large. However, we followed patients for relatively short periods of time with some patients having as little as 6 months between determinations. This may not have been adequate time to see large changes.

It should also be noted that we focused on the decision by clinicians to increase antiglycemic medication therapy. We were interested in studying whether clinicians, when confronted with poor glycemic control, changed pharmacologic management. We did not capture other important aspects of care such as which medication was selected or how much of an increase in dosage occurred, as well as other actions that might be appropriate including enhanced patient education, efforts to improve dietary and medication adherence, or referral to a diabetes team. Additionally, we did not capture information related to potential overmedication, side effects, and risks of hypoglycemia. This may be a particular concern in an older population.

We do not know why clinicians did not increase therapy more frequently. Phillips et al. (2001), in describing clinical inertia, ascribed this phenomenon to three problems in clinical practice: physician overestimation of care provided, use of soft reasons to rationalize decisions not to increase therapy, and lack of training and organizational focus necessary to achieve therapeutic goals. However, El-Kebbi et al. (1999) have also described how failures to intensify therapy in a diabetes clinic are related to clinicians' perceptions that glycemic control is improving or that patients are nonadherent to therapy. Whatever the cause, overcoming clinical inertia is not likely to be easy, but it is essential if we are to substantially improve health outcomes for patients with diabetes. Our measure of treatment intensity may

be a first step in improving how clinicians prescribe medications used in the management of diabetes.

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