Final Report

MOVES: Patient-Based Strategy to Reduce Errors in Diabetes Care

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1. ABSTRACT

Purpose: To assess whether providing medical error information to patients and physicians reduces diabetes medical errors.

Scope: Diabetes medical errors are frequent and lead to many preventable adverse events. Effective interventions to reduce such errors are needed.

Methods: Overall, 123 primary care physicians and 3,703 eligible patients with diabetes were randomly assigned to customized feedback of medical error information to patient only, to physician only, to both the patient and physician, or control group with no feedback. Analysis included hierarchical models with covariate adjustment.

Results: Among patients with errors at baseline, 59.3% of errors were resolved by the end of the study follow-up period. However, study interventions had a negative effect on A1c testing within 6 months of intervention (p=0.01) that resolved by 12 months (p=0.35). When baseline A1c was at least 7%, interventions had no effect on A1c values (p=0.10); when baseline A1c was 8% or greater, interventions unfavorably affected A1c values (p<.01). Interventions negatively affected LDL test ordering (p<.001) but had no effect on LDL values (p=.64), which improved overall. Interventions failed to reduce errors of commission (inappropriate use of medications; p>0.05) or errors of omission (failure to intensify therapy when indicated; p>0.05). Numerous interaction effects were observed. Economic evaluation identified some effect of error status on subsequent healthcare utilization. Some conclusions were that customized feedback interventions to patients and physicians failed to reduce errors of omission or commission related to diabetes care.

Key Words: diabetes, medical errors, patients, feedback

2. PURPOSE AND OBJECTIVES OF STUDY

Specific Aim 1. Evaluate in a randomized trial whether feedback of specific clinical information to patients only (Group A), physicians only (Group B), or both patients and their physicians (Group C) reduces diabetes medical error rates compared to a usual-care control group (Group D).

Hypothesis 1. Diabetes medical error rates will be lower in Group (C) than in Group (D), lower in Group (A) than in Group (D), and lower in Group (C) than in Groups (A or B) after adjustment for relevant patient and physician characteristics.

Specific Aim 2. Demonstrate that routinely collected clinical data can be used to map diabetes medical errors and costs associated with errors across physicians and their patients.

Hypothesis 2. Patients with diabetes medical errors will have higher long-term total healthcare charges than will patients without diabetes medical errors, after adjustment for relevant patient and physician characteristics.

Hypothesis 3. Post-intervention short-term total healthcare charges will not differ for patients of physicians in Group (A), (B), or (C) compared to (D) after adjustment for patient and physician characteristics.

3. SCOPE

The Need to Improve Diabetes Care

There is ample evidence that diabetes care quality in the United States is unsatisfactory. Recent studies indicate that 30-60% of adults with diabetes have good levels of glycemic control (A1c < 7%), 40% have good lipid control (LDL < 130 mg/dL), and only 64-74% of recommended services such as eye exams and foot exams were delivered.[1] The proportion of adults with diabetes who simultaneously meet multiple clinical goals is even lower. Internal data from a leading healthcare plan from 25 medical groups showed that only 10-20% of adults with diabetes simultaneously had A1c < 7% and LDL < 130 mg/dL. Even in published reports from best practice settings, less than 20% of patients meet both A1c and LDL criteria; error rates are 80% or higher.[2-6] Although diabetes medical error is found in both subspecialty and primary care settings, this proposal addresses the problem in outpatient primary care settings, because more than 80% of the nation's

diabetes care is delivered by primary care physicians in office settings.

In this proposal, we define diabetes medical error as the failure of pharmacotherapy or behavioral therapy to achieve the aim of normalizing A1c or LDL within a defined period of time, or the use of inappropriate pharmacotherapy to achieve A1c or LDL control. There is incontrovertible evidence that diabetes medical errors, including failure to control A1c[7, 8] or LDL,[9] often lead to adverse clinical events and adverse outcomes. Adverse events are defined as undesirable or unintended incidents in care that may result in adverse outcomes or may require additional care efforts to thwart an adverse outcome. Adverse events in diabetes patients include macrovascular complications (heart attacks, strokes, peripheral vascular disease) and microvascular complications (nephropathy, neuropathy, and retinopathy). Acute metabolic derangements (hypoglycemia, nonketotic hyperosmolar coma, and diabetic ketoacidosis) and susceptibility to infections are also related to poor glycemic control. These many adverse events translate in turn to numerous adverse outcomes, defined as undesirable and unintended outcomes of care, such as death, disability, or temporary disability.

It has been shown that, for every 1% absolute increase in A1c over 7%, the likelihood of diabetes complications increases 25-30%.[7, 8] Thus, for every 20 people who fail to reach acceptable levels of glycemic control, there is an average of one adverse microvascular event plus one adverse macrovascular event per 5-year period.[10] Diabetes affects over 16 million Americans and prevalence is increasing at a rate of 3-6% a year. In the US, over 150,000 potentially preventable microvascular adverse events at an average cost of $6,000 per event,[11] and an additional 150,000 macrovascular adverse events at an estimated cost of $20,000 per event[11, 12] occur each year. These adverse events lead to an estimated $3.9 billion in excess healthcare charges in diabetes patients each year.[13-17] If error rates could be reduced by roughly 50%, nearly $2 billion per year in medical charges could possibly be saved, because recent studies demonstrate that reduction in A1c[18] or LDL[12] reduce short-term costs of care. However, the cost of efforts to reduce error must be balanced against these averted costs. For this reason, an important aspect of the current proposal is to assess short-term costs associated with reduction in diabetes medical error.

Root Causes of Diabetes Medical Error

Leading models for the improvement of chronic disease care all postulate three important domains that must be addressed to improve care: the patient, the provider, and organization of care.[19-21] The most successful improvement efforts to date have been directed at improving the organization of care.[3-5, 22, 23] Although the gains have been impressive, no setting has yet reported more than 20% of patients at both A1c goals and LDL goals. Furthermore, improvements in A1c and LDL have sometimes been accompanied by increased variation in quality of care across physicians. These data suggest that "breakthrough" reductions in diabetes medical errors must go beyond "organization of care" and take on the challenge of changing both physician and patient behavior, especially behaviors related to pharmacotherapy.

Additional support for the importance of addressing patient and provider behaviors comes from unpublished data from Project QUEST, an AHRQ-sponsored hierarchical analysis of patient-level, physician-level, and clinic-level factors that account for variance in A1c and LDL. Pilot data obtained in this study show that a significant component of the observed variance in A1c is attributable to patient-level and physician-level factors, especially to intensification of drug therapy for A1c or LDL. However, variance in A1c and LDL at the clinic level was not significant when other levels (patient, physician) were included in the model. This analysis is confirmed in part by that of Hofer et al[24] who showed substantial variance in A1c at both the patient and physician levels, especially at the patient level. Published studies by our group[22, 23, 25] and others[3, 4] show that organizational improvements at the clinic or medical group level are capable of improving care. However, after the low-hanging fruit of improvement by organizing care is harvested, even more of the substantial residual variation in A1c and LDL is related to patients and physicians variables, and especially to drug intensification.[7, 9, 26] Data from randomized trials[7, 8] confirm that intensification of appropriate pharmacotherapy predicts improvement in both glycemic control (A1c) and lipid control (LDL). Thus, even as most current improvement strategies focus on organizational change, sustained and continued improvement in diabetes care will not be possible without developing effective, inexpensive, and clinic-congruent interventions to address physician and patient behaviors, especially those related to failed or inappropriate pharmacotherapy.

The Importance of Appropriate Pharmacotherapy and Drug Intensification

It is clear that physician factors, patient factors, and factors related to the doctor-patient interaction may influence drug prescribing. Many studies indicate that physicians[27] are often reluctant to initiate or up-titrate medications, even when they recognize that patients are not at desired clinical goals. However, there are also data that show "academic detailing" or "opinion leaders" may positively influence physician prescribing practices.[28, 29]

Although much of this under-treatment may be related to physician factors, qualitative studies clearly suggest that a portion of the error is related to patient nonadherence, patient reluctance to take prescribed medications, or patient denial of the seriousness of diabetes. Studies of adults with diabetes confirm that patient factors are related to under-treatment of diabetes[30,31] and that patient nonadherence to pharmacotherapy and behavioral therapy often contributes to failure to achieve desired clinical goals. Randomized trials have also shown "patient activation" interventions can lead to reduced A1c and better quality of life.[32, 33]

Thus, the most potent intervention may be to coordinate interventions targeted to physicians with interventions targeted to their patients. The current interest in reduction of medical error makes this a particularly opportune time to launch such a trial, especially with an intervention that is simple, conceptually coherent, inexpensive, and easily assimilated into clinic routines. Consumers are increasingly aware of suboptimal outcomes in today's healthcare system and may be inclined to act on news of suboptimal care. Providers are aware that they must openly evaluate and improve their care in ways that were generally unimagined just a few years ago.

Factors that contribute to physician variation in quality of diabetes care are only beginning to be understood.[24,34] Preliminary data from our group suggest that failure to intensify drug treatment accounts for most diabetes errors and that different physicians have different rates at which they use optimally effective combination therapies to achieve glycemic control or titrate to higher doses of statins to achieve recommended LDL goals.[2]

Important physician sources of diabetes medial error include failure to (a) set clinical goals, (b) initiate appropriate treatment, (c) titrate treatment, (d) reach the clinical goal, and (e) recognize and appropriately manage important comorbid conditions, such as depression, that may interfere with reaching desired clinical goals.[35, 36] Our provisional data suggest that physician specialty, age, and gender are not related to diabetes medial error rates. This conceptual framework, elaborated more in section 3, suggests an intervention strategy that matches specific behavioral interventions to each step of the decision-making process. Examples of inappropriate pharmacotherapy that we have noted in our pilot work include use of metformin in patients with renal insufficiency and use of metformin or TZDs in patients with diagnosed or suspected congestive heart failure. Failed pharmacotherapy often was attributable to delayed initiation or titration insulin, and substituting metformin for a sulfonylurea rather than using combination therapy.

Automated review of A1c or LDL trends in specific patients with diabetes errors often indicates a specific problem related to initiation, titration, or intensification of pharmacotherapy. It also appears that a given physician makes the same error recurrently. Once such a problem is identified, individual, tailored feedback based on a specific patient case can be provided to the physician, emphasizing strategies to reduce diabetes medical errors. Such feedback has been shown in previous studies to change physician prescribing habits in some practice settings.[37, 38]

Review of Patient Interventions To Reduce Diabetes Medical Error

Modern behavior change interventions can substantially improve patient self-care and physician behaviors related to chronic disease care.[33, 39-43] A recent review[40, 44, 45] of patient strategies to improve diabetes care concluded that effective interventions are available. Specific studies have shown that successful interventions directed to patients with diabetes are potent enough to substantially reduce diabetes medical error if they are widely used.[33, 46, 47] For example, Greenfield et al[32] showed in 1988 that a patient activation intervention could lead to better diabetes care outcomes. However, that intervention was so expensive and unwieldy that it has never been implemented in a sustained way in any practice setting in the past 15 years.

Lorig's intervention[33, 46] is also expensive (about $300 per patient), can reach only a small fraction of patients, and is not easily integrated into primary care practice settings. It is clear that evaluations of newer customized approaches that aim to reduce diabetes errors are warranted. The customized interventions proposed in this study are simple, are inexpensive, have the potential of being acceptable to up to 85% of patients, and fit easily into routine primary care practice settings.

Few studies have assessed the effectiveness of coordinated interventions targeted simultaneously to providers and to the provider's patients.[21, 23, 48, 49] A recent Cochrane Collaborative Review of strategies to improve diabetes care reports that interventions that combine patient components with other components (physician, organization of care) are the most potent intervention strategies. Such a coordinated strategy would likely have both practical advantages and theoretical strength. One classic study[50, 51] showed that coordinated patient and physician interventions were more effective than those aimed at one or the other group. However, this dated study used a very expensive intervention model and lowered A1c only from 11% to 10%. A recent report of a coordinated patient and physician intervention[52] showed that intensive (and very expensive) non-customized feedback to patients and their physicians reduced A1c 0.5%, to 8.3%. Importantly, the coordinated feedback strategy was acceptable to physicians. However, most physicians received feedback on only one of their patients, so the study did not take advantage of reinforcement to physicians when they receive customized feedback on multiple patients.

An important factor that limits the broad implementation of these "patient-direct" interventions is their relatively high cost and their lack of fit into primary care practices. Most of these interventions cost about $100 to $400 per patient, and those that require face-to-face contact with patients outside the clinic present difficult logistical and scheduling challenges. Although there is currently a great deal of interest in evaluating telephone, web-based, web-TV, or e-mail interventions, less than half of patients type 2 diabetes are adept at web-based communication. Telephone or mail may present intervention vectors that are more acceptable and familiar to many patients, rather than using internet-based vectors.

In addition to low cost, population penetration, and "goodness of fit" with primary care practice, studies reviewed here suggest several characteristics that would maximize the potency of customized patient-direct interventions designed to reduce diabetes medical error: (1) target both patients and their physicians,[34, 53] (2) vividly describe the problem and the benefits of change to each participant,[54] (3) address and minimize risks or drawbacks to the participant,[47] (4) focus on specific behaviors, rather than knowledge,[44, 45, 47] (5) use data-based feedback,[28, 44, 45, 47, 55] (6) activate patients to maximize their input into physician decision making,[32] and (7) address patients' and physicians' explanatory model of diabetes.[31]

Setting

This study was conducted at HealthPartners Medical Group (HPMG), a multispecialty group practice in Minnesota, where primary care physicians provide most of the care to about 7,000 adults with diabetes at 18 clinics in the Twin Cities metropolitan area. About 10% of diabetes patients were seen by an endocrinologist or endocrine nurse clinician each year, most often for a single visit. About 30% of diabetes patients had an encounter with a diabetes teaching nurse or dietitian each year, most often for a single one-on-one visit.

Sampling Frame, Recruitment, and Retention of Primary Care Physician Participants

All **physicians** participating in the study were (a) either general internists or family physicians, and (b) currently provide ongoing care for adults with diabetes. The number of physicians assigned to each study arm, and the number of associated eligible diabetes patients, is shown in Figure 1.

Sampling Frame and Identification of Adult Study Subjects with Diabetes Mellitus

To be eligible for the study, **patients** were required to have an established diagnosis of diabetes based on either (a) two or more ICD-9 diagnosis codes for diabetes in a 12-month period of time or (b) a filled prescription for a diabetes-specific drug within a 12-month period of time. We have validated this method of diabetes identification, which has an estimated sensitivity of 0.91 and positive predictive value of 0.94 in the study population.[56] Patients who met eligibility criteria were identified from automated laboratory and pharmacy data.

4. METHODS

4.1 Study Design

The study was a group-randomized trial. Details of group criteria are outlined below.

4.2 Data Sources and Data Collection

Definition and Measurement of Dependent Variables

Glycated hemoglobin (A1c) values and A1c test rates were used to assess glycemic control. A1c tests were done at a centralized, accredited clinical chemistry laboratory using a standard high-pressure chromatography assay method[57] with a coefficient of variation of 0.058% at A1c of 8.8%. There were no changes in the assay method during the study period.

LDL cholesterol levels and test rates were used to assess lipid control. Cholesterol tests, including total, HDL and LDL cholesterol, and triglycerides, were done using standard assay methods at a single centralized accredited clinical chemistry laboratory. LDL cholesterol (LDL) values were calculated using the Friedewald equation TC = LDL + HDL + TRIG/5. The equation was solved only for measured triglycerides < 400 mg/dL. Furthermore, no lipid assay results were reported unless the patient had been fasting for at least 10 hours.

All A1c and LDL values and test dates were recorded for a defined 12-month period before and after the date of intervention for all diabetes patients under the care of study physicians. The dates of the defined time period were defined based on the date of interventions. Only patients cared for in both 12-month period prior to and after the intervention date were included in the post-intervention analysis, to ensure that the patient had adequate exposure to the study physicians' post-intervention care. For A1c and LDL tests, the value chosen for analysis was usually the latest (most recent) test in each defined time periods.

D.5. Definition and Measurement of Diabetes Medical Error and Other Dependent Variables

Diabetes medical error is the main dependent variable of interest for Hypothesis 1. To be classified as NOT having a diabetes medical error, the patient must either (a) have achieved both the A1c and LDL goals, and have no inappropriate pharmacotherapy, OR (b) not yet be at A1c or LDL goal, but have received appropriate intensification of pharmacotherapy in the past 4 months.

Appropriate Pharmacotherapy was defined as (a) initiation of therapy, or (b) increase in doses of appropriately chosen agents, or (c) addition of an appropriately chosen agent in response to A1c or LDL values that are not at goal. Dose increases for specific glycemic control agents and lipid control agents are assessed based on dose in mg per day, as calculated for each drug for each patient based on number of tablets dispensed, size of pills, and days supply. Insulin dose is calculated as the number of units of insulin per day dispensed in serial defined 6-month periods.

Inappropriate Pharmacotherapy includes (a) lowering doses, withdrawing medications without substitution of an alternative agent, or failing to initiate or increase pharmacotherapy in the setting of A1c or LDL values not at goal or (b) inappropriate use of a medication in circumstances including (1) use of metformin in a patients with CHF (ICD-9 code 428), with creatinine > 1.5 mg/dL, or ALT > 4x normal; (2) use of TZDs in patient with CHF or ALT > 2.5x normal, (3) use of a statin in a patient with ALT > 4x normal, or (4) use of both statin and fibrate in a patient with CPK > 2x normal.

Accurate classification of diabetes medical error depends upon accurate A1c and LDL tests. The A1c and LDL values chosen for analysis were the latest (most recent) test in each of two consecutive defined 12-month pre- or post-intervention periods. All A1c tests were done at a single accredited clinical chemistry laboratory using a standard liquid chromatographic assay[57] with a normal range of 4.5% to 6.1% and a coefficient of variation of 0.58% at a A1c level of 8.8%. LDL is calculated from standard assays of total cholesterol, HDL cholesterol, and triglycerides only when samples have been obtained after a minimum 12-hour fast and the triglyceride level is < 400 mg/dL. There were no changes in assay methods for either test during the study period.

Definition and Measurement of Independent Variables

In addition to the randomly assigned intervention group, several patient characteristics were included in the analysis. Patient-level independent variables included age, gender, pre-intervention test values (A1c or LDL), CHD status, eligibility for glucose or lipid intervention, insulin use, and any significant interactions between

these patient characteristics and intervention group. All patient-level data needed for the analysis were available from computerized clinical databases, and no surveys or manual chart audits were done. This strengthens the study because the quality of the data is high and there were little missing data.

4.3 Description of Interventions

Control Group
All study physicians practiced under **usual care** conditions. This includes a diabetes clinical guideline, access to clinic-based diabetes education nurses, and referral to endocrinologists, other medical and surgical subspecialties, and CDEs or dietitians as needed. Note that all these care components have previously been studied and shown not to be related to improved glycemic control, thus justifying their use as "usual care" as well as justifying the evaluation of other more potent interventions. For physicians in the control group, no other support for their diabetes care was provided beyond usual care.

Customized Physician Intervention.
Physicians assigned to Physician Intervention groups (Groups B and C) received feedback interventions in the form of a list of their patients with diabetes who had errors or commission and/or omission and who met other study inclusion criteria (age and comorbidity scores). The feedback included specific clinical information and suggestions customized both to their individual patients and to the care those individual patients have previously received. Moreover, the patients were prioritized based on clinical grounds into one of nine groups related to errors of omission. Errors of commission were separately listed in a distinct category.

The list provided each patient's past data values and medication history with respect to two health markers, A1c and LDL, only if a particular patient was not at goal on one of these two markers. Specific suggestions for alterations in pharmacotherapy were provided, such as 'increase metformin dose to maximum effective dose," or "initiate therapy with Lantus insulin 10-15 units subcutaneously each evening." All suggestions to physicians were proposed as contingent upon a thorough review of the case, examination of the patient, and updated laboratory testing. An initial list was sent at the time of intervention and an updated list with updated clinical profiles and recommendations was sent 4 months later to reinforce the intervention. Specific suggestions (customized feedback) to each physician focused on one or more of the following:

1) Failure to initiate appropriate pharmacotherapy. The list suggested the importance of initiating treatment regimens when health markers were above specific thresholds and pointed out that Patient 'X' is above the threshold for either A1c or LDL (or both) and no medication moves have been made in the past several months. The list also provided suggestions for beginning treatment and how to intensify treatment in order to achieve goal.
2) Failure to titrate pharmacotherapy to achieve specific goals. This list indicated the importance of setting specific treatment goals for each patient and pointed out that Patient 'X' was not at the accepted clinical goal for their condition. The list went on to suggest strategies for intensifying treatment systematically over time in order to achieve targeted A1c and/or LDL goals.
3) Inappropriate Pharmacotherapy. This list pointed out the dangers of particular medications for some patient conditions and noted that Patient 'X' may have one such condition. Suggestions were made for alternative treatment strategies tailored to Patient 'X'. For example, "stop metformin because serum creatinine is >= 1.5 mg/dL and select alternative oral agent or insulin therapy."

Customized patient intervention Patients assigned to Patient Intervention groups (Groups A and C) received a customized, 4-page brochure that followed the conceptual model described above for physician feedback. Patient brochures focused on:

1) The patient's current condition and note that either A1c or LDL (or both) was above recommended levels. A graph showed their personal trends in A1c and LDL values over at least a 12-month period of time, and a note was made that the patient was above the threshold for either A1c or LDL or both.
2) The nature of the medications that are generally appropriate for achieving long-term health and quality of life goals, keyed to whether the patient is already receiving medications or not. If the patient was receiving

no medications, initiation of pharmacotherapy was suggested. If the patient was already on pharmacotherapy, titration of therapy was suggested. If inappropriate pharmacotherapy was identified, the patient was notified that caution was indicated and was advised to discuss the situation with their physician.

3) The benefits of decisions (and behaviors) to reduce A1c or LDL were outlined. A caveat indicates that benefits are based on group experience and may not apply equally to all individuals.

The patient was asked to review the brochure with their personal physician (who was named in the letter) and to discuss with their physician strategies for medication changes, exercise, or other ideas to help the patient improve their diabetes care. If a patient was already at goal for either A1c or LDL, then that section of the letter was not included. A second updated letter was sent 4 months later to reinforce the intervention.

4.4 Analytic Approach

Because physicians were group randomized to intervention group and patient outcome data was linked to these physicians, the study design can be considered a nested (patients within physicians) cohort design due to the presence of repeated measures on the patient data. Technically, randomization happened within clinics, and all physicians in the same clinic were randomized to the same intervention group. Due to the small number of clinics and small and nonsignificant variance at this level, it was dropped from the analysis, leaving a patient within provider model. The principal analytic strategies considered for the analysis of this design were (a) a time by condition analysis with covariate adjustment, and (b) an analysis of post-intervention data with regression adjustment for the pre-intervention measure of the dependent variable and other covariates.[58] This latter analytic model was chosen as the method of analysis, because providers (and their patients) were randomly assigned to condition, and there was no evidence of differences across intervention groups in any of the pre-intervention measures of the dependent variables.

4.5 Protection of Human Subjects

This study was reviewed, approved in advance, and monitored by the HealthPartners Institutional Review Board.

5. RESULTS

Table 1 compares baseline characteristics and selected post-intervention data of study participants in each of the intervention groups. Because the study was group randomized, it is not surprising to find some baseline covariate imbalance across groups at the patient level. These analyses provide information needed to guide covariate adjustment in subsequent analyses.

Table 2 shows changes in glycated hemoglobin (A1c) testing and in A1c values of adults with diabetes before and after the study intervention. The sample size varies by analysis depending on the number of participants with adequate data required for various analyses. All three intervention groups had significantly lower A1c test rates compared to control in the 6-month period post-intervention ($p < .01$); when assessing rates over the entire 12-month post-intervention period, there were no significant differences in A1c test rate associated with interventions ($p=.35$). Among the patients who did not have an observed pre-intervention A1c value (n=181), there were no significant differences across intervention groups in the proportion who had an A1c test in the 6 (47.0%, $p=.20$) or 12 (60.2%, $p=.39$) months post intervention (not shown).

Table 1. Description of patients and physicians participating in the MOVES interventions.

Patients	ALL N=3703	Intervention Group Control N=847	Patient N=869	Provider N=1041	Both N=946	p[b]
Age as of intervention date (M, SD)	56.1 (12.1)	56.3 (11.7)	57.2 (12.2)	56.3 (12.6)	54.8 (11.8)*	.001
Age ≥ 65 (% yes)	26.4	26.4	29.8	27.2	22.4*	.005
Gender (% female)	46.1	45.7	49.1	47.7	42.0	.02
CHD in 12 months pre-intervention (% yes)	11.1	12.0	11.2	11.2	9.9	.55
Charlson score in 12 months pre – intervention (% Charlson = 2)	22.3	21.5	20.5	26.2*	20.5	.005
insulin use at baseline (% yes)	30.4	31.6	28.7	27.7	34.0	.01
lipid intervention eligible (% yes)	61.9	59.3	61.9	62.8	63.2	.17
glucose intervention eligible (% yes)	66.8	61.7	68.4*	68.7*	67.8*	.005
Pre-intervention A1c[a] (median)	7.2	7.1	7.2*	7.2*	7.2*	.05
Post-intervention A1c[a] (median)	7.0	6.9	7.0*	7.1*	7.1*	.005
Pre-intervention LDL[a] (median)	103	104	102	102	104	.19
Post-intervention LDL[a] (median)	89	88	89	89	89	.76
Pre-intervention A1c[a] (M, SD)	7.53 (1.6)	7.42 (1.6)	7.53 (1.6)	7.55 (1.6)	7.60 (1.6)	.10
Post-intervention A1c[a] (M, SD)	7.36 (1.5)	7.20 (1.4)	7.38 (1.5)*	7.43 (1.6)*	7.43 (1.6)*	.01
Pre-intervention LDL[a] (M, SD)	108.0 (31.9)	108.6 (31.7)	107.4 (32.5)	106.4 (31.2)	109.6 (32.3)	.21
Post-intervention LDL[a] (M, SD)	93.2 (30.5)	92.2 (29.5)	93.3 (30.0)	94.1 (31.7)	93.3 (30.1)	.72
Physicians	N=123	N=32	N=27	N=37	N=27	
Patients per physician	30.1	26.5	32.2	28.1	35.0	.27

[a] Pre- and post-intervention A1c and LDL values are the last observed values in the 12 month period prior to or following the intervention.
[b] Omnibus test of significance for differences across intervention groups.
* p<.05 for planned comparison of intervention group relative to control group.

The middle portion of Table 2 also displays the impact of the interventions on A1c levels for patients who had any pre-intervention A1c value and for those with pre-intervention A1c ≥ 8%. Results indicate no effects of the interventions on A1c levels in the analysis that included patients with any pre-intervention A1c value. Among the patients who had pre-intervention A1c ≥ 8%, the impact of the intervention depended on the patient's age, sex, and insulin use. In the two groups that received the patient intervention, older patients tended to have lower A1c values, but age was not related to A1c values in the other two groups. Men, who had higher A1c values in general, tended to have lower A1c values in the patient intervention group, but among women, the lowest A1c values were observed in the control group. Finally, patients using insulin had higher A1c values than did those not using insulin, and their A1c values did not differ greatly across the four intervention groups; patients not using insulin in any of the three active intervention groups had higher A1c values than did those in the control group.

The lower portion of Table 2 shows the impact of interventions on LDL testing and on LDL values in adults with diabetes. Among 2,547 patients with a pre-intervention LDL test, LDL test rates in the 12-month post-intervention period were significantly lower for all three intervention groups than for the control group ($p < .001$), although this effect was attenuated for men. Among the patients who did not have an observed pre-intervention LDL value (n=508), there were no significant differences across intervention groups in the proportion who had an LDL test in the 12 months post intervention (55.1%, p=.21; not shown). In a separate model predicting post-intervention LDL levels among patients with pre-intervention LDL ≥ 100 mg/dL, the impact of the interventions depended on patients' age, pre-intervention LDL levels, insulin use, and whether they were eligible for a glucose intervention message. Older patients tended to have lower LDL values in the control and provider intervention groups, but there was no relationship between age and LDL values in the two patient intervention groups. Patients with higher pre-intervention LDL values tended to have higher post-intervention LDL values, and even more so in the two provider intervention groups. Patients using insulin tended to have higher LDL values in the two provider groups (Ms=108.8, 105.0) relative to the control (M=100) and patient (M=97) intervention groups; patients not using insulin had high equally high LDL values in all four groups (Ms=105, 103, 105, 105). Finally, patients with pre-intervention A1c levels that made them eligible to receive an intervention message tended to have higher LDL values that were similarly high in all four intervention groups (Ms=106, 104, 107, 105). Those who were not eligible for the glucose message had higher

LDL values in the two provider intervention groups (102, 106) relative to the control (99) and patient (96) intervention groups.

Table 3 presents data that reflect the impact of the interventions on proportion of patients with at least one glucose or lipid medication intensification in patients with baseline LDL >= 100 mg/dL (N=1,283) or baseline A1c >= 7% (N=1,037 not on insulin therapy, 1,683 including those on insulin therapy). Adjusted models show no significant impact of any of the three interventions on likelihood of pharmacotherapy intensification among those not at their evidence-based clinical goals for A1c or LDL. This result supports the null hypothesis that the study interventions did not reduce errors of omission in diabetes care. However, it is important to recall that, among all patients with an error of commission or an error of omission in the pre-intervention period, 40.7% had the same error during the post-intervention period, and 59.3% of errors had been resolved by 12 months post-intervention.

The data in Table 4 support this last assertion and also provide specific information on changes in errors rates for specific errors of commission related to inappropriate or risky use of pharmacotherapy for A1c or LDL control. For most of these specific errors, the frequency of occurrence is so low that it is difficult to determine whether there was an impact of the intervention. Therefore, we consolidated errors of commission into larger groups of errors but still found few significant differences. Though power may be an issue here, other studies suggest that, at this sample size and with similar error rates, other error reduction strategies may significantly reduce error rates.

Table 5 provides data on the association of pre-intervention glucose and lipid status with post-intervention resource use. It can be seen that patients who are classified as having errors of omission (above goal without moves) have higher utilization in several categories of care. On the other hand, those without recent therapy intensification in some instances have lower pharmacy charges. These data are consistent with the hypothesis that efforts to reduce errors of omission by intensifying care may increase pharmacy costs but subsequently decrease costs in other categories, such as inpatient care related to diabetes complications.

6.1 Conclusions

Eligible patients were all included in the interventions unless they had previously opted out of research studies in general. The IRB granted a waiver of consent, which would have increased the generalizability of the study, but may have led to inclusion of many patients who had little interest or motivation to improve their diabetes care. It is possible that inclusion of a large number of such patients could have diluted a potential intervention effects. On the other hand, requiring consent may have led to artificial inflation of intervention effects, which could not be replicated under routine practice conditions. Thus, we view the inclusion of all eligible physicians and patients not as a weakness of the study but as strength. In this context, the failure of the interventions to affect most of the pre-specified endpoints is a cautionary tale for other planning or exploring similar intervention strategies.

Pre-specified levels of power were achieved, and, among patients with errors at baseline, 59.3% of all errors were resolved by the end of the study follow-up period. However, the interventions failed to reduce errors beyond what was observed in the control group. All three study interventions had a negative effect on A1c testing within 6 months of intervention ($p=0.01$) that resolved by 12 months post intervention ($p=0.35$). In patients with baseline A1c >= 7% the interventions had a marginally significant negative effect on A1c values ($p=0.10$); among the subgroup of patients with baseline A1c >= 8%, the interventions had a significantly unfavorable effect on A1c levels ($p < 0.01$). All interventions had a significantly negative effect on LDL test ordering ($p<0.001$) but no effect on LDL values ($p=0.64$), which improved during the study period. The interventions failed to reduce the rate of errors of commission (inappropriate use of medications; $p>0.05$) or errors of omission (failure to intensify therapy when indicated; $p>0.05$). Numerous interaction effects were observed. Initial economic evaluations indicate sporadic effects of error status on subsequent utilization of healthcare.

We conclude that the study interventions failed to reduce errors of omission or errors of commission related to diabetes care. For some measures, tailored feedback of error information to physicians or patients had the unintended effect of worsening A1c levels.

6.2 Limitations

There are a number of factors that limit the interpretation and application of these results.

(1) Generalizability. This study was conducted at a single Minnesota medical group, and the physicians and patients may be somewhat different than those in other geographic locals in terms of demographic or other characteristics. Detailed data on patients included in this study are provided in Table 1, and additional information on the patient population from which study subjects were drawn can be found in other publications.[2, 22, 59-61]

(2) Physician and patient selection effects. These are unlikely to threaten internal validity because of the randomized study design and unlikely to threaten the external validity of the study because the IRB granted a waiver of consent for both physicians and patients. Thus, by design, all eligible physicians participated in this study. This is a strength in terms of generalizability but may have led to the inclusion of physicians who were not particularly interested in or receptive to the intervention-related activities. From the point of view of physicians, the study would have been tangible in two ways: (a) physicians in the combined intervention group may have seen patients who had received intervention letters may have come in and asked for review of their care, and (b) physicians in both physician intervention arms of the study received lists of patients not at goal (and patients with selected error of commission) and were provided specific treatment suggestions for management of these patients' A1c, LDL, or errors. Physician study subjects were not compensated financially for time spent in study-related activities.

(3) Study Design. We proposed and executed a full factorial design, and we enrolled a sufficiently large number of physicians and patients to maintain acceptable levels of power for analysis of all errors of omission. The power to detect errors of commission as presented in this report may be marginal. For this reason, we are planning more analyses that will evaluate occurrence of errors of commission for other perspectives and thus increase the breadth and power of this aspect of the analysis. The application of this expanded approach to the pre-intervention data is found in a previous publication related to this project.

(4) This study was conducted prior to electronic medical record implementation at the study medical group. This led to some significant limitations of the physician intervention and the patient intervention. First, the decision support provided to physicians was not provided at the time of the patient visit but was provided every 4 months as a list of patients with recommended treatment changes that could not be implemented until the patient was in for a visit or contacted by phone. There is little evidence that patients were contacted by phone as a result of the study interventions, in part because an expanded nurse role was not an integral part of the intervention strategy. Second, the patient intervention letters included data that could be up to 6 weeks out of date at the time the letters were received, because the data on which the letters were based were electronically updated only once a month. Third, we could not administratively capture actual blood pressure readings of patients. Thus, the study did not provide information on BP to either patients or to physicians, nor were we able to assess impact of the intervention strategies on BP control.

6.3 Significance

Intervention strategies generally did not reduce errors of commission or errors of omission in diabetes care. This unanticipated result could be explained by either conceptual problems related to the theory behind the interventions or to problems related to the effective logistical application of the intervention strategies. In addition, for the patient intervention, problems with the presentation of the results and problems related to the customization strategy used must be seriously considered.

6.3.1 Problems with the Patient Intervention.

There are a number of possible explanations for why the patient intervention did not work. First, the patient letters were customized based on A1c and LDL values but were not customized based on other factors that may influence likelihood of improved diabetes care. Among these other factors, customization to the patient's readiness to change (RTC) was prominently absent. It was not possible to estimate patient RTC based on the available clinical data. However, in other work, we have shown that patient RTC is a powerful predictor of future change in patients with diabetes who do not have another more dominant disease, such as coronary heart disease. Knowledge of patient RTC would have enabled customization of messages based on RTC, a strategy that is supported by several prior research reports.

Second, it is possible that the letters to patients overestimated the patients' health literacy or their numeral. Health literacy has been shown to be related to effective diabetes care, and the letters, although carefully constructed to avoid excessive jargon or excessively complicated messages, did included numerical data and graphs of recent A1c and LDL values. It is possible that innumeracy or limited health literacy may have impeded the interpretation of the information presented for at least some patients.

Third, patient mailings were formatted as a four-page brochure with personalized information, such as the name of the patient, the patents' personal primary care physician, the names of diabetes educators at the primary care clinic, and other information. The brochure arrived in a sealed envelope marked confidential but was unsolicited and sent without prior notification to the patient. It is possible that some patients either did not read the mailed communication or did not respond to the information in an emotionally favorable way. It is also feasible that some patients with elevated A1c or LDL levels were not recently told by their physicians that changes in care were needed or had been told that they were doing well. In such cases, unsolicited information that arrived by mail and conflicts with the patient's recollection of a different message conveyed personally by a primary physician may have undermined the credibility of the mailed information.

The information in the patient brochures was as accurate and complete as we could make it using automated clinical databases. However, in a small percentage of cases, the name of the patient's clinic or their personal physician in the database was not current or accurate. We received about 10 calls from patients (less than 1% of subjects) who notified us of this problem. If this was a problem for a large proportion of patients, it may have undermined the credibility of the enclosed clinical information.

6.3.2 Problems with the Physician Intervention.

The physician decision support that was provided had no measurable beneficial effects on care and had a significantly negative impact on subsequent changes in A1c values. This remarkable and unfortunate finding demands careful consideration, because there may be an important message here, not yet clearly elucidated in the published literature. To consider this issue carefully, we must consider a number of several potential problems with this intervention mechanism. However, we must also consider the possibility that there is a flaw in the theoretical model that is the foundation of the intervention strategy—a problem of sufficient magnitude to provoke major unintended consequences.

First, the decision support information provided to physicians was up to 6 weeks out of date when deployed. Information was provided as a list of all diabetes patients who were out of control on A1c or LDL values, categorized in order of priority into 10 categories. Physicians were encouraged to concentrate on the highest-priority patients. However, it may have been many weeks or months before some of these patients arrived in the clinic for their next visit. The likelihood that the physician would recall the specifics of the recommendations after such a time lag is low. Moreover, the information on the list would become increasingly obsolete with the passage of time, and physicians may have viewed the information provided as being generally out of date or unreliable.

Many of the factors that limited the effectiveness of the physician intervention could be eliminated if the decision support could be provided at the point of care using electronic medical records. One of the take home lessons of our project might be that sophisticated clinical decision support (what drug, what dose) designed to lead to intensified chronic disease care is not effective when not delivered at the point of care.

The physician decision support intervention failed to emphasize the importance of team approaches to using the information provided. If the physician were the only user, the likelihood of appropriate and timely action would be small. If nurses or other clinical team members were involved and assigned clear and consistent responsibilities, then the likelihood of active outreach to patients who needed more intensive therapy could be increased. The list provided prioritization of patients based on their CV risk and the degree to which their A1c or LDL (or both) was out of control. However, if the information was used only in reactive fashion (wait for the patient to come into clinic) the results would be less positive than if the information was applied proactively (call the patient and invite a clinic visit). The absence of well-organized primary care teams would reduce the value of the information provided, just as the presence of a well-functioning team of providers using basic principles of disease management would increase the impact of the information provided on patients' actual care. More emphasis on development of functional primary car teams, and on the importance of patient prioritization and active outreach, may have enhanced the intervention strategy and should be evaluated in the future.[39, 62]

6.4 Implications.

Despite unexpected results, the study has a number of potentially important implications. First, the physician intervention strategy did nothing to change the practice environment of the physicians. The intervention was designed primarily to change physician behavior. It is likely that an intervention strategy that provides cognitive clinical information to physicians in a timely way, in conjunction with other proven quality improvement designed to improve office systems of care.[22, 63] might be more effective. Prominent targets for office systems interventions would be the creation of small primary care teams that include physicians, nurses, and case managers and the deployment of electronic medical records that can deliver customized decision support at the point of care and be used by team members other than physicians.

Second, primary care physician interventions may be more effective if they are customized to patterns of care by a given physician, in addition to being customized to the needs of individual patients. Furthermore, the learning style of PCPs may be variable, and customization of learning interventions to the PCP's learning style is another enhancement that may be considered in future studies. For example, it may be possible to develop "physician archetypes" with respect to diabetes care.[64] Such a physician profiling system could be tested for its ability to predict patterns of care and predict mistakes before they occur. Physician archetypes would also be relevant for education of physicians and open up new approaches to deal with common managerial, change, and accountability challenges.

Third, the intervention may have greater impact in settings in which baseline quality of diabetes care was less optimal. However, at the time of the study, less than 20% of adults with diabetes had simultaneously reached evidence-based A1c, LDL, and BP goals.

Finally, we do not provide a formal cost-effectiveness analysis of the intervention strategies, because they were not effective. However, an informal analysis shows data are consistent with other findings on these topics in similar groups of patients.[17, 65]

7. LIST OF PUBLICATIONS AND PRODUCTS

7.1 Published Works

O'Connor PJ, Sperl-Hillen JM, Johnson PE, Rush WA. Clinical Inertia and Outpatient Medical Errors. In Advances in Patient Safety: From Research to Implementation. K. Henriksen, J. Battles, D. Lewin, and E. Marks (eds) . Agency for Healthcare Research (AHRQ) 2005; vol 2; 293-3 http://www.ahrq.gov/qual/advances/

O'Connor PJ, Sperl-Hillen JM, Johnson PE, Rush WA. Identification, Classification, and Frequency of Medical Errors in Outpatient Diabetes Care. In Advances in Patient Safety: From Research to Implementation. K. Henriksen, J. Battles, D. Lewin, and E. Marks (eds). Agency for Healthcare Research (AHRQ) 2005; vol 1; 369-380. http://www.ahrq.gov/qual/advances/

Gilmer TP, O'Connor PJ, Rush WA, et al. Predictors of health care costs in adults with diabetes. Diabetes Care 2005; 28:59-64.

O'Connor PJ, Gray RJ, Maciosek MV, et al. Cholesterol levels and statin use in patients with coronary heart disease treated in primary care settings. Preventing Chronic Dis. Jul 2005; 2(3): A05

Sperl-Hillen JM, O'Connor PJ. Factors driving diabetes care improvement in a large medical group: ten years of progress. Am J Managed Care. Aug 2005; 11(Suppl 5): S177-185.

Electronic Resources from this Study

We have developed a software program that presents customized case-based feedback to physicians in the form of text and tables that list recommended drug intensifications for LDL and A1c control. These recommendations take into account the patient's age, renal function, selected comorbidities (congestive heart failure, coronary heart disease, Charlson comorbidity score), baseline medications, and baseline A1c and LDL levels. This programming supports physician decision support and can be developed more and may be more effective if it is (a) based on data updated daily instead of weekly and (b) is presented to the physician at the

time of a patient visit, rather than in a batched list every several months. We currently have started a new project that encompasses these additional factors.

We have also developed programming that gathers patient information including current and prior A1c and LDL values, medications used, age, and comorbidity score as well as outputs graphs and text designed to be communicated directly to patients. This output format was ineffective in the current project. Future work is needed to address output formatting and presentation of information to patients and to consider whether customization on additional factors, such as readiness to change, mental models of diabetes, patient activation, numeracy, or health literacy, is indicated.

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Table 2. Impact of intervention group assignment on glycated hemoglobin (A1c) and LDL testing rates and values in the MOVES trial. A1c (LDL) testing rates and values were predicted using general or generalized mixed model regression, nesting patients within providers and specifying a random intercept for physician; all other effects were fixed. Covariates included age, sex, pre-intervention A1c (LDL) value, CHD, eligibility for lipid (glucose) intervention, insulin use, and significant interactions between covariates and intervention group.

	ALL	Intervention Group: Control	Patient	Provider	Both	p[a]
A1c testing rate – 1+ test in 6 months						
Any pre-intervention A1c value						
N	3107	715	718	895	779	
pre-intervention (%)	100	100	100	100	100	
post-intervention (%)	71.2	76.6	69.2	70.5	68.9	
predicted post-intervention (%)		76.6	69.2**	70.5*	68.9**	.01
significant covariates: pre-intervention A1c, age, insulin*pt letter, lipid intervention eligible						
A1c testing rate – 1+ test in 12 months						
Any pre-intervention A1c value						
N	3107	715	718	895	779	
pre-intervention (%)	100	100	100	100	100	
post-intervention (%)	86.1	87.8	87.2	85.4	84.2	
predicted post-intervention (%)		87.8	87.2	85.4	84.2	.35
significant covariates: age, lipid intervention eligible						
A1c values						
Any pre-intervention A1c value						
N	2673	628	626	763	656	
pre-intervention M (SD)	7.47 (1.5)	7.39 (1.5)	7.47 (1.5)	7.49 (1.5)	7.52 (1.5)	
post-intervention M (SD)	7.35 (1.5)	7.20 (1.4)	7.35 (1.4)	7.42 (1.5)	7.41 (1.6)	
predicted post-intervention (M)		7.20	7.35	7.42*	7.41+	.10
significant covariates: pre-intervention A1c, age, lipid intervention eligible						
Pre-intervention A1c ge 8						
N	658	149	152	184	173	
pre-intervention M (SD)	9.45 (1.4)	9.44 (1.4)	9.47 (1.4)	9.43 (1.5)	9.48 (1.3)	
post-intervention M (SD)	8.71 (1.7)	8.50 (1.8)	8.60 (1.6)	8.85 (1.8)	8.85 (1.8)	
predicted post-intervention (M)		8.50	8.60**	8.86**	8.85***	.16
significant covariates: baseline A1c, age*pt letter, age*pt letter*prov letter, male, male*pt letter, male*prov letter, male*pt letter*prov letter, insulin use, insulin use*pt letter, insulin use*prov letter						
LDL testing rate – 1+ test in 12 months						
Any pre-intervention LDL value						
N	2547	614	569	716	648	
pre-intervention (%)	100	100	100	100	100	
post-intervention (%)	79.3	83.2	80.1	76.3	78.2	
predicted post-intervention (%)		83.2	80.1*	76.3**	78.2**	.001
significant covariates: pre-intervention ldl, age, male, male*pt letter						
LDL values						
Pre-intervention LDL ge 100						
N	991	254	221	267	249	
pre-intervention M (SD)	128 (26)	128 (25)	128 (28)	129 (278)	129 (26)	
post-intervention M (SD)	104 (31)	104 (32)	101 (27)	106 (34)	105 (31)	
predicted post-intervention (M)		104	101	106	105	.64
significant covariates: pre-intervention LDL, LDL*prov letter, age, age*pt letter, male, glucose intervention eligible, glucose eligible*prov letter, insulin use, insulin use*prov letter						

[a] Omnibus test of significance for differences across intervention groups.

+ p<.10, * p<.05, ** p<.01, *** p<.001 for planned comparison of intervention group relative to control group.

Table 3. Impact of interventions on lipid and glucose medication initiations and titration. Medication changes were predicted using generalized mixed model regression, nesting patients within providers and specifying a random intercept for physician; all other effects were fixed. Covariates in models predicting lipid (glucose) moves included age, sex, pre-intervention LDL (A1c) value, CHD, eligibility for glucose (lipid) intervention, insulin use, any glucose (lipid) move, and significant interactions between covariates and intervention group. Glucose moves analyses omitted patients who were using insulin at the time of the intervention.

		Intervention Group				
	ALL	Control	Patient	Provider	Both	p[a]
Lipid moves – any hyperlipidemia medication initiation or titration in the 12 months post-intervention						
Pre-intervention LDL ge 100						
N	1283	310	288	352	333	
lipid med pre-intervention (%)	49.9	47.1	51.4	54.3	46.5	
lipid move post-intervention (%)	34.7	35.2	37.5	32.4	34.2	
model predicted post-intervention (%)		35.2	37.5	32.4	34.2	.64
significant covariates: pre-intervention LDL, age, any glucose move						
Glucose moves – any diabetes medication initiation or titration in the 12 months post-intervention						
Pre-intervention A1c ge 7						
N	1037	207	256	317	257	
glucose med pre-intervention (%)	89.3	88.4	89.5	89.6	89.5	
glucose move post-intervention (%)	33.8	35.3	34.4	31.9	34.6	
model predicted post-intervention (%)		35.3	34.4	31.9	34.6	.71
significant covariates: pre-intervention A1c*pt letter, lipid intervention eligible, any lipid move						
Glucose starts – any diabetes medication initiation in the 12 months post-intervention						
Pre-intervention A1c ge 7						
N	1683	360	394	501	428	
glucose med pre-intervention (%)	.934	.933	.931	.934	.967	
glucose start post-intervention (%)	.181	.197	.183	.170	.178	
model predicted post-intervention (%)		.197	.183	.170	.178	.43
significant covariates: pre-intervention A1c*pt letter, lipid intervention eligible, insulin use, any lipid move						

[a] Omnibus test of significance for differences across intervention groups.

Table 4. Impact of intervention group assignment on errors of commission related to diabetes care. Analyses for each error included patients with specified errors pre-intervention. Adjusted logistic regression models included age and sex as covariates and are not adjusted for nesting within physicians. For errors with sample sizes smaller than n=30, exact p values are presented.

		Intervention Group				
	ALL	Control	Patient	Provider	Both	p[a]
O1: high protein in urine and not on ACE or ARB. No pre-intervention errors.						
T1: high ALT/AST in 12 months and on statin. No pre-intervention errors.						
T2: high ALT/AST in 12 months and on TZD. No pre-intervention errors.						
T3: On metformin and no creatinine or creatinine clearance in last 12 months						
pre-intervention errors N	250	49	67	57	77	
post-intervention resolved (%)	90.4	85.7	91.0	91.2	92.2	
post-intervention persisted (%)	9.6	14.3	9.0	8.8	7.8	.66
T4: On statin and fibrate with no CK test						
pre-intervention errors N	173	45	35	38	55	
post-intervention resolved (%)	43.9	44.4	34.3	55.3	41.8	
post-intervention persisted (%)	56.1	55.6	65.7	44.7	58.2	.32
X1: On metformin and high ALT or AST						
pre-intervention errors N	2			1	1	
post-intervention resolved (%)	100			100	100	

	ALL	Intervention Group Control	Patient	Provider	Both	p[a]
post-intervention persisted (%)	0			0	0	na
X2: On TZD and high ALT or AST						
pre-intervention errors N	1		1			
post-intervention resolved (%)	100		100			
post-intervention persisted (%)	0		0			na
X3: high ALT or AST and on statin. No pre-intervention errors.						
X4: 80+ and on metformin and no creatinine clearance in 12 months						
pre-intervention errors N	66	15	23	16	12	
post-intervention resolved (%)	80.3	73.3	91.3	68.7	83.3	
post-intervention persisted (%)	19.7	26.7	8.7	31.3	16.7	.32
X5: On metformin and indications of CHF						
pre-intervention errors N	118	33	27	30	28	
post-intervention resolved (%)	38.1	30.3	44.4	43.3	35.7	
post-intervention persisted (%)	61.9	69.7	55.6	56.7	64.3	.59
X6: On TZD and indications of CHF						
pre-intervention errors N	57	17	14	12	14	
post-intervention resolved (%)	45.6	52.9	57.1	41.7	28.6	
post-intervention persisted (%)	54.4	47.1	42.9	58.3	71.4	.42
X7: High CK and on fibrate. No pre-intervention errors.						
X8: On statin and high CK						
pre-intervention errors N	10	4	4	1	1	
post-intervention resolved (%)	40.0	75.0	0	100	0	
post-intervention persisted (%)	60.0	25.0	100	0	100	.09

[a] Omnibus test of significance for differences across intervention groups.

X9: On metformin and indications of COPD						
pre-intervention errors N	102	30	25	24	23	
post-intervention resolved (%)	30.4	36.7	28	29.2	26.1	
post-intervention persisted (%)	69.6	63.3	72.0	70.8	73.9	.90
X10: age 80+ with low creatinine clearance and on metformin. No pre-intervention errors.						
X11: On metformin and high serum creatinine						
pre-intervention errors N	12	5	4	1	2	
post-intervention resolved (%)	75.0	80.0	50.0	100	100	
post-intervention persisted (%)	25.0	20.0	50.0	0	0	.64
metformin error: T3 or X4 or X10 or X11						
pre-intervention errors N	325	68	92	74	91	
post-intervention resolved (%)	87.7	82.4	89.1	86.5	91.2	
post-intervention persisted (%)	12.3	17.6	10.9	15.5	8.8	.50
		significant covariate: age				
CHF error: On metformin or TZD and indications of CHF						
pre-intervention errors N	158	46	35	39	38	
post-intervention resolved (%)	38.6	39.1	40.0	41.0	34.2	
post-intervention persisted (%)	61.4	60.9	60	59.0	65.8	.91

Any error						
pre-intervention errors N	698	173	174	158	193	
post-intervention resolved (%)	59.3	57.2	59.8	61.4	59.1	
post-intervention persisted (%)	40.7	42.8	40.2	38.6	40.9	.89

[a] Omnibus test of significance for differences across intervention groups.

Table 5. The association of pre-intervention glucose and lipid status with post-intervention healthcare utilization. Each glucose and lipid error group is compared to goal status (i.e., A1c<7%, LDL<100 mg/dl), with blank cells indicating no significant differences in utilization.

	Professional Care	Hospitalized	Outpatient Care	Medication Use
Pre-Intervention Glucose Error Group				
At goal, A1c <7% (reference)				
A1c 7 - 7.9%; not on insulin, no medication adjustment				
A1c 7 - 7.9%; on insulin or medication adjustment				+ 19%
A1c 8 - 10.9%; not on insulin and no oral medication adjustment				
A1c 8 - 10.9%; on insulin or oral medication but insufficient		+ 32%	+ 27%	+ 16%
A1c ≥ 11%			+ 77%	
No A1c Test				- 32%
Pre-Intervention Lipid Error Group				
At goal, LDL <100 mg/dl (reference)				
LDL 100 - 129, no medication adjustment				
LDL 100 - 129, with medication adjustment				+ 43%
LDL ≥ 130, no medication adjustment				
LDL ≥ 130, with insufficient medication adjustment				
LDL not determined, high TGY				
No LDL test				- 28%