Serveur Académique Lausannois SERVAL serval.unil.ch

Author Manuscript

Faculty of Biology and Medicine Publication

This paper has been peer-reviewed but dos not include the final publisher proof-corrections or journal pagination.

Published in final edited form as:

Title: Treatment intensification and risk factor control: toward more

clinically relevant quality measures.

Authors: Selby JV, Uratsu CS, Fireman B, Schmittdiel JA, Peng T,

Rodondi N, Karter AJ, Kerr EA

Journal: Medical care

Year: 2009 Apr

Volume: 47

Issue: 4

Pages: 395-402

In the absence of a copyright statement, users should assume that standard copyright protection applies, unless the article contains an explicit statement to the contrary. In case of doubt, contact the journal publisher to verify the copyright status of an article.



UNIL | Université de Lausanne Faculté de biologie et de médecine



Med Care. Author manuscript; available in PMC 2014 August 01.

Published in final edited form as: Med Care. 2009 April; 47(4): 395-402.

Treatment Intensification and Risk Factor Control: A Quality Improvement Perspective

Joe V. Selby, MD¹, Connie S Uratsu, BA¹, Bruce Fireman, MA¹, Julie A Schmittdiel, PhD¹, Tiffany Peng, MA¹, Nicolas Rodondi, MD², Andrew J Karter, PhD¹, and Eve A. Kerr, MD³

¹Division of Research, Kaiser Permanente Medical Care Program, Northern California, Oakland, CA ²Department of Ambulatory Care and Community Medicine, University of Lausanne, Lausanne Switzerland ³Center for Clinical Management Research, VA Ann Arbor Healthcare System, and the Division of General Medicine, University of Michigan Medical School, Ann Arbor, MΙ

Abstract

Background—Intensification of pharmacotherapy in persons with poorly controlled chronic conditions has been proposed as a clinically meaningful process measure of quality.

Objective—To validate measures of treatment intensification by evaluating their associations with subsequent control in hypertension, hyperlipidemia, and diabetes mellitus across 35 medical facility populations in Kaiser Permanente, Northern California.

Design—Two-level, hierarchical analyses of associations of improvements in facility-level treatment intensification rates from 2001 to 2003 with patient-level risk factor levels at the end of 2003.

Patients—515,072 and 626,130 members (age > 20 years) with hypertension, hyperlipidemia, and/or diabetes mellitus in 2001 and 2003, respectively.

Measurements—Treatment intensification for each risk factor defined as an increase in number of drug classes prescribed, of dosage for at least one drug, or switching to a drug from another class within 3 months of observed poor risk factor control.

Results—Facility-level improvements in treatment intensification rates between 2001 and 2003 were strongly associated with greater likelihood of being in control at the end of 2003 (p 0.01 for each risk factor) after adjustment for patient- and facility-level covariates. Compared to facility rankings based solely on control, addition of percentages of poorly controlled patients who received treatment intensification changed 2003 rankings substantially: 14%, 51%, and 29%) of the facilities changed ranks by 5 or more positions for hypertension, hyperlipidemia, and diabetes, respectively.

Conclusions—Treatment intensification is tightly linked to improved control. Thus, it should be an effective process measure for motivating quality improvement and for measuring clinical performance.

Keywords

diabetes mellitus; hypertension; hyperlipidemia; quality of health care; cardiovascular disease

Introduction

Morbidity and mortality from cardiovascular diseases are largely attributable to poor control of hypertension, hyperlipidemia, and diabetes mellitus, along with cigarette smoking (1-5). Despite the availability of evidence-based treatments (6-10), recent reports (11-14) find persistent suboptimal control of blood pressure, LDL-cholesterol and hemoglobin A1c. Moreover, physicians often fail to intensify pharmacologic treatment appropriately in the face of poor risk factor control (15-21). "Clinical inertia" has been suggested as an explanation for poor control and a target for quality improvement (15,22-23).

Kerr (24,25) and others (15) have suggested that measurement and feedback of the frequency with which pharmacotherapy is intensified in patients with poorly controlled risk factors may represent a useful approach to clinical quality measurement and improvement. Voorham et al (26) found that simple measures of proportions of patients in control for hypertension, hyperlipidemia and diabetes underestimate the proportions receiving high quality clinical care and concluded that more sophisticated measures including measures of treatment intensification could improve quality assessment. Considering a process measure such as treatment intensification along with measures of risk factor control addresses concerns that case-mix differences rather than variation in clinical quality could explain observed differences in control and may reduce provider reluctance to include difficult-to-treat patients in their panels. It may also reduce risks of overtreatment and adverse consequences from an overzealous focus on tight risk factor targets (27,28).

Because numerous clinical trials demonstrate that treatment intensification leads to improved hypertension, hyperlipidemia and diabetes control, intensification measures have been called "tightly linked" (24,25) or "evidence-based," in contrast to simpler non-evidence-based processes such as rates of testing. However, linkage of treatment intensification rates in populations, as would be used in quality measurement, with better rates of risk factor control has not yet been demonstrated.

We reported treatment intensification rates (29) in patients with poorly controlled hypertension, hyperlipidemia, or diabetes mellitus for Kaiser Permanente Northern California during 2003. Intensification rates were somewhat higher than in earlier reports (15, 18-19), but room for further improvement remained and significant variation in rates was observed across the 39 medical facilities within this system. In this paper, we test hypotheses that variation among these facilities in treatment intensification rates for hypertension, hyperlipidemia, and diabetes mellitus are associated with differences in levels of risk factor control.

Methods

Study Population

The study population has been described previously (29,30). We identified all adult members (age 20 years) of Kaiser Permanente Northern California who were recognized by clinicians as having hypertension, diabetes mellitus, and/or hyperlipidemia during two 18-month periods (June 1, 2000 - December 31, 2001 and June 1, 2002 - December 31, 2003), and were continuously enrolled for at least the final 4 months of either period. Kaiser Permanente is an integrated health care system providing comprehensive care to a diverse population (31) of approximately 3.2 million members in Northern California.

Identifying criteria for each condition are given in the Appendix. Patients with elevated risk factor values were not included if no evidence was found of physician awareness (i.e., recorded diagnoses, prescriptions for disease-specific medications, or in the case of diabetes a follow-up hemoglobin A1c test). Cutpoints for identifying hyperlipidemia varied depending on patients' cardiovascular disease risk status, using ATP-III guidelines (7). Assignment of risk required identification of previous coronary heart disease from inpatient and outpatient diagnoses and calculation of 10-year coronary heart disease risk for each patient. Totals of 515,072 and 626,130 members met criteria for hypertension, hyperlipidemia, and/or diabetes in 2001 and 2003, respectively.

Enrollment and demographic data (age, sex) were obtained from membership databases. Burden of co-morbid illness was assessed using the DxCG Diagnostic Cost Group risk adjustment methodology, (32,33) which is based on 1-year hospital discharge and ambulatory diagnoses (for 2003). Self-reported race/ethnicity was found for approximately 88% of all members using information from birth, maternity and hospitalization records and responses to member satisfaction surveys.

Members were assigned to a facility using the location of their primary care provider, as recorded in system databases. We excluded 20,134 (3%) otherwise eligible patients from 4 small facilities that opened after the end of 2001, because change scores could not be calculated for these facilities, and an additional 2.5% of eligible patients who could not be linked with a primary care provider.

Definitions of Control

Risk factor control was assessed for all patients with each condition, based on last recorded values in 2001 and 2003 from laboratory (LDL-cholesterol and hemoglobin A1c) and encounter (blood pressure) databases. We have previously shown that last readings are equivalent to means of all readings within a year for aggregate measures.(34) Values were available for at least 95 percent of those with hypertension and for 94% of those with hyperlipidemia or diabetes in each year. Persons with no values for a condition were excluded only from analyses of that condition.

For each condition, we defined two levels of control (Table 1). "Stringent control" was defined using the most rigorous guideline targets in place in 2003. "Near control" was then defined using somewhat higher cutpoints that corresponded with the action levels used in

calculating treatment intensification (see below). "Poor control" designates values above the cutpoints for near control. Definitions of control also varied depending on risk for both LDL-cholesterol and blood pressure control (Table 1). In December 2002, a switch to the IFCC reference standard promoted by the National Glycohemoglobin Standardization Group (35) at Kaiser Permanente's regional laboratory effecting an immediate decrease of 0.3 percentage points in Hb A1c values. This affected all facilities simultaneously, but led to a prompt 10 percentage-point improvement in the percentage of patients with A1c values <7%.

Treatment intensification

Treatment intensification was identified from pharmacy databases and calculated for all patients who met criteria for poor control at any point during either year. We did not measure intensification for those in near control out of concern that an intensification measure in these patients could encourage over-treatment and adverse effects. Intensification was defined as an increase in number of drug classes prescribed, increased dosage of at least 1 medication, or a switch to another medication in a different therapeutic class within 3 months (and 6 months) following the initial observation of poor control. Follow-up was extended into 2004 to detect intensification when poor control was first identified in the last months of 2003.

Seven medication classes were tabulated for hypertension, 5 for hyperlipidemia, and 5 for diabetes (29). Daily dosages were categorized as low, medium, or high based on package insert recommendations and inspection of actual dosage distributions. Dosages at or below recommended initial starting doses were categorized as low; those within the recommended maintenance range as intermediate; and dosages at or above the high end of the recommended range as high. To identify intensification, comparison was made to medications dispensed during the four-month period before poor control was noted. We included switches to a medication in a different class as treatment intensifications because they represent physician responses intended to render therapy more "intense" either by changing to a more effective or better tolerated medication. Only 11%, 2%, and 4% of "intensifications" for hypertension, hyperlipidemia and diabetes were solely switches among classes. We excluded all patients with diabetes who were using insulin prior to the observation of poor control because day-to-day adjustments in insulin can not be identified in prescription databases.

Data Analyses

We examined whether longitudinal improvements in treatment intensification were associated with improvements in control for each condition. Primary analyses treated both stringent and near control as being in control, because these were the cutpoints used in calculating treatment intensification. However, we repeated all analyses to see whether treatment intensification as calculated was also associated with rates of stringent control.

We examined inter-facility differences in proportions of patients in stringent and stringent/ near control and proportions of poorly controlled patients receiving treatment intensification for each time period and condition. Systolic rather than diastolic blood pressure control was

examined because of its greater importance in predicting coronary heart disease risk.(36,37) Only 16% of all hypertensive patients had diastolic pressure 90 mmHg in 2003 and of these, 72% of those also had elevated systolic pressure.

Unadjusted associations of facility-level improvements in treatment intensification from 2001 to 2003 with improvements in control rates were assessed using Spearman rank correlations. To adjust for case-mix differences and baseline differences in control, we then constructed two-level (38-40) models with improvement in facility-level treatment intensification rates from 2001 to 2003 as the main independent variables and patient-level control (stringent/near control vs. poor control) at the end of 2003 as dependent variables. We used Generalized Linear Models (SAS PROC MIXED with GLIMMIX Macro (Version 9.1)) and included random effects for facility and for provider nested within facility. Two facility-level covariates were included as fixed effects: the percent of patients in control in 2001 and the percent of poorly controlled patients receiving treatment intensification in 2001. Patient-level fixed effects included age, sex, and race-ethnicity, and the DxCG score. Race/ethnicity was included because African-American patients are consistently found to have higher levels and more difficulty controlling each of these risk factors (41-43). Race/ ethnicity distributions differed greatly by facility and treatment intensification has been previously shown to vary by race/ethnicity (29). Hyperlipidemia models were further adjusted for patients' prior history of coronary heart disease and hypertension models for prior history of target organ disease as defined by JNC VI (44), because these characteristics altered target LDL-cholesterol (7) and blood pressure levels (43) in place during this period, may have differed between facilities, and therefore could confound facility comparisons of treatment intensification and control.

Model results are presented as adjusted odds ratios for control associated with a 1 percentage point change in the facility treatment intensification rates from 2001 to 2003. To facilitate interpretation of these results, we used all model coefficients to calculate predicted probabilities of being in control at two different facilities: one with the average observed improvement in treatment intensification and the other with 5 percentage points greater improvement. This 5% difference represents one third to one half of the observed ranges across facilities for increases in treatment intensification for the three risk factors. The predicted differences in control (and 95% confidence intervals) are presented.

In sensitivity analyses, we examined associations of treatment intensification with likelihood of being in stringent control (rather than in stringent/near control). We also re-fit models after deleting small percentages whose only treatment intensification was switching to a medication of a different class; and after lengthening the period in which treatment intensification could occur to 6 months.

To study the impact of measuring treatment intensification on performance ranking, we compared crude rankings of the 35 facilities by proportions in control (stringent/near control) at the end of 2003 with rankings based on combined measures of proportions in control or receiving treatment intensification. Patients who received treatment intensification during the year and were in control by their last reading were counted as in control, not as having received treatment intensification. We also calculated the mean change in rankings

and the proportion of facilities that changed by 5 or more rank positions when the combined measure was substituted. This study was reviewed and approved by Kaiser Permanente's Institutional Review Board.

Results

Numbers of persons meeting criteria for each condition increased between 2001 and 2003 (Table 2), especially for hyperlipidemia, but there were no important changes in age, gender or race distributions, nor in disease severity for hypertension or hyperlipidemia. Sample sizes per facility were large, the smallest cell being 240 diabetic patients at one facility in 2001. Proportions of patients who were African-American varied widely across facilities as did mean age and levels of comorbidities.

Levels of Control and Treatment Intensification

Control improved for each condition from 2001 to 2003 (Table 2) and varied substantially across facilities in both years. For hypertensive patients, 48% and 78% met criteria for stringent control and stringent/near control of systolic blood pressure, respectively, in 2003; 45% and 77% of those with hyperlipidemia met these criteria for LDL-cholesterol; and 51% and 77% of diabetic patients did so for hemoglobin A1c. Improvements in control appeared greatest for hemoglobin A1c, but as much as half of this was due to the change in laboratory standardization.

On average, treatment intensification for hypertension increased by 7 percentage points from 2001 to 2003; increases were smaller for hyperlipidemia and diabetes. These changes varied from less than 1% to a 15.2% increase for hypertension; from a 9% decrease to a 9.4% increase for hyperlipidemia, and from a 4.9% decrease to 7.4% increase for diabetes.

Association of treatment intensification with control

Facility-level increases in treatment intensification from 2001 to 2003 were associated with concurrent improvements in control rates (stringent/near control) for each condition. Correlations with improvements in stringent control were of similar size for hypertension and hyperlipidemia, but somewhat weaker for diabetes (Spearman rank correlations: 0.53, p < 0.01 for hypertension; 0.42, p=0.01 for hyperlipidemia; 0.20, p=0.24 for diabetes)

In two-level models, improvement in a facility's treatment intensification rate from 2001 to 2003 was positively associated with a patient's likelihood of being in control (stringent/near control) at the end of 2003 (p<0.01 for each condition) after adjustment for facility- and patient-level covariates (Table 3). Compared with the average improvement, a 5% greater increase in the facility's treatment intensification rate predicted a 1.1% greater likelihood of systolic blood pressure control; a 1.1% greater likelihood of LDL-cholesterol control; and a 1.8% greater likelihood of hemoglobin A1c control. These associations are remarkably strong considering that only 23-25% of patients were eligible for treatment intensification, while control was measured in the entire affected population. Higher facility-level control and treatment intensification rates in 2001 were also strong, independent predictors of patient control in 2003.

Associations were of similar size when the dependent variables were changed to being in stringent control (adjusted OR's: systolic blood pressure: 1.012, p=0.01; LDL-cholesterol: 1.013, p=0.018; and hemoglobin A1c, 1.020, p=0.0005); when switching to a medication in a different class was not counted as treatment intensification; and when the period for identifying treatment intensification was lengthened to 6 months.

Among patient-level predictors, greater age was associated with poorer control of systolic blood pressure but better control for LDL-cholesterol and hemoglobin A1c. Female gender was associated with slightly lower odds of blood pressure control, but not with LDL-cholesterol or hemoglobin A1 control. Compared with white patients, all non-white race/ethnicity groups were substantially less likely to be in control for A1c values. African- and Native-Americans were also less likely to be in control for hypertension and hyperlipidemia, but Asian patients were more likely to be in control for each. Greater levels of comorbidity were associated with better blood pressure control, but not with better LDL-cholesterol or hemoglobin A1c control.

Comparison of rankings Show facility rankings by proportions of patients in stringent/near control. The non-monotonic pattern of heights for the full vertical bars indicates that including the additional proportions of poorly controlled patients receiving treatment intensification changes facility rankings. Spearman rank correlations between these two ranking approaches were lower and average changes in ranking greater for hyperlipidemia and diabetes than for hypertension (Table 4).

Discussion

Clinical inertia remains an important cause of poor risk factor control in chronic conditions (20,21). Clinical uncertainty and competing demands have been identified as contributors to inertia (45). Nevertheless, the present findings confirm that clinical populations experiencing greater increases in treatment intensification rates for hypertension, hyperlipidemia and diabetes mellitus had better control of these conditions, strengthening the case that greater emphasis on treatment intensification would be useful for quality improvement (24-26). The strength of the associations we observed was large for each condition. Five percent improvements in treatment intensification, calculated in the 32 to 38% of patients eligible for intensification, led to 1.1 to 1.8% improvements in control for the entire population. Associations were equally strong whether we used less stringent or stricter definitions of control.

Although many clinical trials demonstrate that treatment intensification improves risk factor control in individual patients, it could still be argued that when treatment intensification is aggregated to the population level, higher rates may not necessarily be equivalent to better control. Conceivably, the wrong patients could be intensified or the wrong medications used. Non-adherence to medications could undermine the effects of intensification. The present analyses reduce these concerns and suggest that, on average, physicians groups or health plans with higher intensification rates for these conditions are achieving better control.

We used two-level analyses, relating facility-level measures of treatment intensification to patient-level outcomes, rather than individual-level analyses for two reasons. It was the population measure of treatment intensification that we wished to evaluate. Secondly, two-level analyses are less biased than individual-level observational studies by confounding by indication (38-40). Thus, in a recent individual-level study, (46) patients with poorly controlled hypertension who received treatment intensification appeared *more* likely to remain poorly controlled than those who did not. In a second study (21), only a weak, nonsignificant association of treatment intensification with better hypertension control was found, even after controlling for baseline blood pressure levels. Decisions to intensify treatment in individual patients involve consideration of factors that strongly confound observational analyses. By focusing on practice variation between facilities rather than between patients, two-level analyses reduce such confounding.

More generally, our findings support the concept that linked process measures offer an attractive, additional approach for quality improvement. Focusing on and improving simpler process measures is often unaccompanied by improvements in meaningful outcomes (47-51). Clinicians and health care systems can be more confident that a focus on improving linked process measures will translate to benefit for patients.[1] Such measures have additional advantages in that the action required of physicians or systems to improve quality (in this case, intensifying treatment) is implicit in the measure. They can be tailored to focus on optimal medication or clinical strategies for improving long-term outcomes.

Despite these attractive features, treatment intensification measures as constructed here would have serious limitations as external performance measures for health plans, physician groups, or individual physicians. Their measurement requires both risk factor and prescribing data, which are not readily available in less integrated delivery settings without electronic health records (EHRs). Conceivably, chart reviews could be adapted to obtain this added information, but standardized measurement would also require complex categorization of multiple drugs and combination medications into treatment classes, standard quantification of dosage increases, and measurement of risk-specific target levels. Each of these would require updating over time.

A second limitation is that the treatment intensification measures we constructed include only the minority of patients who are in poor control. As control improves, these denominators shrink. Using stricter definitions of control could enlarge denominators (i.e, by including the larger numbers in "near control"), but we would be concerned that in a context of performance reporting and incentivization, more stringent cutpoints could lead to adverse effects of overmedication in patients who would stand to gain very little (27,28). However, in the context of internal quality improvement efforts, where there is less risk of perverse incentives, broader definitions of poor control could be examined. For example, patients who are in near control on two consecutive measures could be included, reasoning that non-pharmacologic efforts do not appear to be helping.

An additional limitation of these measures is that systems or groups with higher proportions already in control would not be recognized and could even be penalized if their smaller numbers of remaining poorly controlled patients were more resistant to intensification or

more likely to have valid reasons not to intensify, such as intolerance to some medication classes or known non-adherence to current medications. Measures that combine proportions in control with proportions of remaining patients who received treatment intensification (25,26) capture both aspects of clinical quality and were shown in this study to change performance rankings compared with simpler measures of percent in control.

Although the associations we observed were robust, the measures could be refined in several ways in settings where data are available. Credit could be given for the small percentages of patients (3% of persons with diabetes or hyperlipidemia; 11% of those with hypertension) who returned to control without changes in pharmacotherapy by their next reading. (29) Patients who appeared to be nonadherent to present medications could be excluded before calculating intensification rates, as treatment intensification may be inappropriate for this group (52). Credit could be given for small percentages (well below 10%) of hypertensive or hyperlipidemic patients already on maximal medical therapy (20,29) and for persons with contraindications to some medication classes or with comorbidities that may make intensification inappropriate. (53) Measures could ultimately be refined to distinguish among treatment intensification choices, possibly giving more credit for using recommended next steps in treatment intensification and not crediting intensifications that have been shown to be harmful. Lastly, prescriptions written rather than prescriptions filled may be a more direct measure of clinical performance, although data from Kaiser Permanente's EHR indicate that more than 95% of physician-ordered prescriptions for new medications are filled at least once (54) and thus captured in prescription database. Each of these refinements could enhance the credibility of intensification measure with providers. A persistent limitation without obvious solution is the inability, in either EHR or prescription databases, to capture day-do-day intensifications of insulin therapy.

In conclusion, improvements in rates of treatment intensification for patients with poorly controlled hypertension, hyperlipidemia, or diabetes mellitus are directly related to improved population risk factor control. These measures deserve further consideration for use in improving clinical quality of care.

Acknowledgments

Funded in part by a research grant from Pfizer Pharmaceuticals, Inc. By contract, first-author (JVS) retained right to publish findings without approval from Pfizer. Dr. Rodondi supported by a grant from the Swiss National Foundation (PBLAB-102353). Dr. Kerr's time was funded in part by the VA HSR&D Quality Enhancement Research Initiative for Diabetes Mellitus (DIB 98-001) and by the Measurement Core of the Michigan Diabetes Research and Training Center (P60DK-20572).

Appendix 1. Diagnostic Criteria for Diabetes Mellitus, Hypertension, and Hyperlipidemia

Diabetes Mellitus (one of the following)

- 1. at least one prescription of insulin or an oral hypoglycemic agent; or
- 2. at least two outpatient diagnoses of diabetes mellitus; or
- 3. one outpatient diagnosis of diabetes mellitus plus 1 Hemoglobin A1c 7%; or

4. at least one hospital discharge with a primary diabetes-related diagnosis (ICD-9 code 250.X).

Hypertension (one of the following)

- 1. at least one prescription for an anti-hypertensive medication plus an outpatient diagnosis of hypertension; or
- 2. at least two outpatient diagnoses of hypertension; or
- 3. at least one prescription for an anti-hypertensive medication plus one or more elevated outpatient blood pressure readings (140 mm Hg systolic, or 90 mm Hg diastolic); or
- 4. at least one outpatient diagnosis of hypertension plus at least one blood pressure reading of 140 mm Hg systolic or 90 mm Hg diastolic;

Hyperlipidemia (one of the following)

- 1. at least one prescription for an anti-lipemic agent; or
- 2. Outpatient diagnosis of hyperlipidemia/hypercholesterolemia with an LDL-cholesterol value risk-appropriate cutpoint value*; or
- **3.** Outpatient diagnosis of hyperlipidemia/hypercholesterolemia with a <u>prior LDL</u>-cholesterol value risk-appropriate cutpoint value (within 2 years prior to 7/01/00).

References

- 1. Anderson KM, Wilson PW, Odell PM, et al. An updated coronary risk profile. A statement for health professionals. Circulation. 1991; 83(1):356–362. [PubMed: 1984895]
- 2. Poulter N. Coronary heart disease is a multi-factorial disease. Am J Hypertens. 1999; 12(10 Pt 2): 92S–95S. [PubMed: 10555607]
- 3. Vaccaro O, Stamler J, Neaton JD. Sixteen-year coronary mortality in black and white men with diabetes screened for the Multiple Risk Factor Intervention Trial (MRFIT). Int J Epidemiol. 1998; 27:636–641. [PubMed: 9758118]
- Greenland P, Knoll MD, Stamler J, et al. Major risk factors as antecedents of fatal and nonfatal coronary heart disease events. JAMA. 2003; 290:891–7. [PubMed: 12928465]
- Khot UB, Khot MB, Bajzer CT, et al. Prevalence of conventional risk factors in patients with coronary heart disease. JAMA. 2003; 290:898–904. [PubMed: 12928466]
- 6. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003; 42:1206–1252. [PubMed: 14656957]
- Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP).
 Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA. 2001; 285:2486–2497. [PubMed: 11368702]
- 8. American Diabetes Association. Implications of the diabetes control and complications trial. Diabetes Care. 2003; 26(Suppl 1):S25–7. [PubMed: 12502616]

^{*}For persons with coronary heart disease or coronary heart disease risk equivalents: 100 mg/dL; for persons with at least 2 coronary heart disease risk factors, but 10-year predicted risk <20%: 130 mg/dL; for persons with less than two coronary heart disease risk factors, no coronary heart disease risk equivalents: 160 mg/dL.

 Snow V, Aronson MD, Hornbake ER, et al. Clinical Efficacy Assessment Subcommittee of the American College of Physicians. Lipid control in the management of type 2 diabetes mellitus: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2004; 140:644–9. [PubMed: 15096336]

- Snow V, Weiss KB, Mottur-Pilson C. Clinical Efficacy Assessment Subcommittee of the American College of Physicians. The evidence base for tight blood pressure control in the management of type 2 diabetes mellitus. Ann Intern Med. 2003; 138:587–92. [PubMed: 12667031]
- 11. Borzecki AM, Wong AT, Hickey EC, et al. Hypertension control: how well are we doing? Arch Intern Med. 2003; 163:2705–11. [PubMed: 14662624]
- 12. Saaddine JB, Cadwell B, Gregg EW, et al. Improvements in diabetes processes of care and intermediate outcomes: United States, 1988-2002. Ann Intern Med. 2006; 144:465–74. [PubMed: 16585660]
- Wong ND, Lopez V, Tang S, et al. Prevalence, treatment and control of combined hypertension and hypercholesterolemia in the United States. Am J Cardiol. 2006; 98:204–208. [PubMed: 16828593]
- 14. Vittinghoff E, Shlipak MG, Varosy PD, et al. Risk factors and secondary prevention in women with heart disease: the Heart and Estrogen/progestin Replacement Study. Ann Intern Med. 2003; 138:81–9. [PubMed: 12529088]
- 15. Berlowitz DR, Ash AS, Glickman M, et al. Developing a quality measure for clinical inertia in diabetes care. Health Serv Res. 2005; 40:1836–53. [PubMed: 16336551]
- Asch SM, McGlynn EA, Hiatt L, et al. Quality of care for hypertension in the United States. BMC Cardiovasc Disord. 2005; 5:1. [PubMed: 15638933]
- 17. Shrank WH, Asch SM, Adams J, et al. The quality of pharmacologic care for adults in the United States. Med Care. 2006; 44:936–45. [PubMed: 17001265]
- 18. Grant RW, Buse JB, Meigs JB. Quality of diabetes care in U.S. academic medical centers: low rates of medical regimen change. Diabetes Care. 2005; 28:337–442. [PubMed: 15677789]
- Hyman DJ, Pavlik VN, Vallbona C. Physician role in lack of awareness and control of hypertension. J Clin Hypertens (Greenwich). 2000; 2:324–30. [PubMed: 11416669]
- Schmittdiel JA, Uratsu CS, Karter AJ, et al. Why Don't Diabetes Patients Achieve Recommended Risk Factor Targets? Poor Adherence versus Lack of Treatment Intensification. J Gen Intern Med. 2008; 23:588–94. [PubMed: 18317847]
- 21. Rose AJ, Berlowitz DR, Orner MB, et al. Understanding uncontrolled hypertension: is it the patient or the provider? J Clin Hypertens (Greenwich). 2007 Dec; 9(12):937–43. [PubMed: 18046098]
- 22. Phillips LS, Branch WT, Cook CB, et al. Clinical inertia. Ann Intern Med. 2001; 135:825–34. [PubMed: 11694107]
- 23. Asai Y, Heller R, Kajii E. Hypertension control and medication increase in primary care. J Hum Hypertens. 2002; 16:313–18. [PubMed: 12082491]
- Kerr EA, Krein SL, Vijan S, et al. Avoiding pitfalls in chronic disease quality measurement: a case for the next generation of technical quality measures. Am J Manag Care. 2001; 7:1033–43.
 [PubMed: 11725807]
- 25. Kerr EA, Smith DM, Hogan MM, et al. Building a better quality measure: are some patients with 'poor quality' actually getting good care? Med Care. 2003; 41:1173–82. [PubMed: 14515113]
- 26. Voorham J, Denig P, Wolffenbuttel BH, et al. Cross-sectional versus sequential quality indicators of risk factor management in patients with type 2 diabetes. Med Care. 2008 Feb; 46(2):133–41. [PubMed: 18219241]
- 27. Pogach L, Engelgau M, Aron D. Measuring Progress Toward Achieving Hemoglobin A1c Goals in Diabetes Care: Pass/Fail or Partial Credit. JAMA. 2007; 297:520–23. [PubMed: 17284702]
- 28. Hayward R. All-or-nothing treatment targets make bad performance measures. Am J Manag Care. 2007; 13:126–28. [PubMed: 17335355]
- 29. Rodondi N, Peng T, Karter AJ, et al. Treatment intensifications in response to poorly controlled hypertension, dyslipidemia, and diabetes mellitus. Ann Intern Med. 2006; 144:475–84. [PubMed: 16585661]

30. Selby JV, Peng T, Karter AJ, et al. High rates of co-occurrence of hypertension, elevated low-density lipoprotein cholesterol, and diabetes mellitus in a large managed care population. Am J Manag Care. 2004; 10(2 Pt 2):163–170. [PubMed: 15005509]

- 31. Krieger N. Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. Am J Public Health. 1992; 182:703–710. [PubMed: 1566949]
- 32. Ash A, Ellis RP, Pope GC, Ayanian JZ, Bates DW, Burstin H, Iezzoni L, McKay E, Yu W. Using Diagnoses to Describe Populations and Predict Costs. Health Care Financing Review. 2000; 21:7–28. [PubMed: 11481769]
- 33. DxCG website: http://www.dxcg.com/solutions/global.asp.
- 34. Alexander MA, Tekawa I, Hunkeler E, et al. Evaluating hypertension control in a managed care setting. Arch Intern Med. 1999 Dec 13-27; 159(22):2673–7. [PubMed: 10597757]
- 35. Hoelzel W, Weykamp C, Jeppsson JO, et al. IFCC reference system for measurement of hemoglobin A1c in human blood and the national standardization schemes in the United States, Japan, and Sweden: a method-comparison study. Clin Chem. 2004; 50:166–174. [PubMed: 14709644]
- 36. Thomas F, Bean K, Guize L, et al. Combined effects of systolic blood pressure and serum cholesterol on cardiovascular mortality in young (<55 years) men and women. Eur Heart J. 2002; 23:528–535. [PubMed: 11922642]
- 37. Benetos A, Thomas F, Bean K, et al. Prognostic value of systolic and diastolic blood pressure in treated hypertensive men. Arch Intern Med. 2002; 162:577–581. [PubMed: 11871926]
- 38. Morgenstern H. Ecologic studies in epidemiology: concepts, principles, and methods. Annu Rev Public Health. 1995; 16:61–81. [PubMed: 7639884]
- 39. Johnston SC. Combining ecological and individual variables to reduce confounding by indication: case study subarachnoid hemorrhage treatment. J Clin Epidemiol. 2000; 53:1236–41. [PubMed: 11146270]
- Johnston SC, Henneman T, McCulloch CE, van der Laan M. Modeling treatment effects on binary outcomes with grouped-treatment variables and individual covariates. Am J Epidemiol. 2002; 156(8):753–60. [PubMed: 12370164]
- 41. Kirk JK, D'Agostino RB Jr, Bell RA, et al. Disparities in HbA1c levels between African-American and Non-Hispanic white adults with diabetes: a meta-analysis. Ann Pharmacother. 2005; 39:1489–501. [PubMed: 16076917]
- 42. Bosworth HB, Dudley T, Olsen MK, et al. Racial differences in blood pressure control: potential explanatory factors. Am J Med. 2006; 119:70.e9–15. [PubMed: 16431192]
- 43. Yood MU, McCarthy BD, Kempf J, et al. Racial differences in reaching target low-density lipoprotein goal among individuals treated with prescription statin therapy. Am Heart J. 2006; 152:777–84. [PubMed: 16996858]
- 44. The sixth report of the Joint National Committee on prevention, detection evaluation, and treatment of high blood pressure. Arch Intern Med. 1997; 157:2413–46. [PubMed: 9385294]
- 45. Kerr EA, Zikmund-Fisher BJ, Klamerus ML, Subramanian U, Hogan MM, Hofer TP. The role of clinical uncertainty in treatment decisions for diabetic patients with uncontrolled blood pressure. Ann Intern Med. 2008; 148:717–27. [PubMed: 18490685]
- 46. Ho PM, Magid DJ, Shetterly SM, et al. Importance of therapy intensification and medication nonadherence for blood pressure control in patients with coronary disease. Arch Intern Med. 2008; 168:271–6. [PubMed: 18268167]
- 47. Mangione CM, Gerzoff RB, Williamson DF, et al. Association of Diabetes Disease Management Programs' Intensity with Process and Outcomes Measures of Quality of Care: The Translating Research into Action for Diabetes (TRIAD) Study. Ann Intern Med. 2006; 145:107–116. [PubMed: 16847293]
- 48. Landon BE, Hicks LS, O'Malley AJ, et al. Improving the managemet of chronic disease at community health centers. N Engl J Med. 2007; 356:921–34. [PubMed: 17329699]
- Duru OK, Mangione CM, Steers NW, et al. The association between clinical care strategies and the attenuation of racial/ethnic disparities in diabetes care: the Translating Research Into Action for Diabetes (TRIAD) Study. Med Care. 2006; 44:1121–8. [PubMed: 17122717]

50. Ackermann RT, Thompson TJ, Selby JV, et al. Is the number of documented diabetes process-of-care indicators associated with cardio-metabolic risk factor levels, patient satisfaction, or self-rated quality of diabetes care. Diabetes Care. 2006; 29:2108–13. [PubMed: 16936161]

- 51. Fonarow GC, Abraham WT, Albert NM, et al. Association Between Performance Measures and Clinical Outcomes for Patients Hospitalized With Heart Failure. JAMA. 2007; 297:61–70. [PubMed: 17200476]
- 52. Heisler M, Hogan MM, Hofer TP, Schmittdiel JA, Pladevall M, Kerr EA. When more is not better: treatment intensification among hypertensive patients with poor medication adherence. Circulation. 2008; 117:2884–92. [PubMed: 18506011]
- 53. Pogach LM, Tiwari A, Maney M, et al. Should mitigating comorbidities be considered in assessing healthcare plan performance in achieving optimal glycemic control? Am J Manag Care. 2007 Mar; 13(3):133–40. [PubMed: 17335354]
- 54. Karter AJ, Parker MM, Moffet HH, et al. Poor medication adherence for newly prescribed cardiometabolic therapies in diabetes: An under-recognized public health issue. Am J Managed Care. 2008 under review.

Table 1
Risk-specific Definitions of Control for Systolic Hypertension, Hyperlipidemia, and Diabetes Mellitus

Condition and Risk Category	Measure	Stringent control	Near control	Poor control
Systolic Hypertension				
No diabetes and no target organ disease $\dot{\tau}$	2 consecutive systolic blood pressures (mm Hg)	< 140	140-159	160
With diabetes or target organ disease		< 130	130-139	140
Hyperlipidemia				
Low Risk: No diabetes or coronary heart disease, < 2 coronary heart disease risk factors	LDL-cholesterol, (mmol/L (mg/dL))	< 4.2 (160)	4.2-5.0 (160-190)	5.0 (190)
Moderate Risk: 2 coronary heart disease risk factors, but 10-yr Framingham risk < 20%		< 3.4 (130)	3.4-4.1 (130-159)	4.2 (160)
High Risk: With diabetes or coronary heart disease, or 2 coronary heart disease factors and 10-yr Framingham risk 20%		< 2.6 (100)	2.6-3.3 (100-129)	3.4 (130)
Diabetes				
	Hemoglobin A1c (%)	< 7%	7.0–7.9%	8%

^{*}DM: diabetes mellitus;

 $^{^{\}dagger}\text{TOD:}$ target organ disease (cardiovascular or renal disease, 44);

 $^{^{\}ddagger}$ SBP: systolic blood pressure;

[§]CAD: coronary artery disease

Table 2 Characteristics of Patients, Levels of Control, and Rates of Treatment Intensification (with Inter-facility Ranges) for Hypertension, Hyperlipidemia, and/or Diabetes Mellitus, across 35 Medical Facilities Kaiser Permanente Northern California, 2001 and 2003

Hypertension: Total N (inter-facility range)	2001 381,671 (988 – 25,556)	2003 478,320 (1,287 – 29,592)
Mean Age (standard deviation) (inter-facility range)	62.9 (13.7) (57.3 – 69.2)	62.7 (13.9) (58.2 – 68.6)
% Female (inter-facility range)	55.4 (50.2 – 59.6)	54.9 (51.0 – 58.3)
% African American (inter-facility range)	11.0 (0.2 – 45.2)	10.8 (0.4 – 45.3)
% with diabetes or target organ disease (inter-facility range)	39.9 (30.6 – 43.5)	41.2 (32.1 – 44.9)
Mean SBP in mm Hg (standard deviation) (inter-facility range)	144.3 (17.1) (138.7 - 149.5)	139.1 (15.8) (136.1 – 142.8)
% in stringent control for SBP (inter-facility range)	34.0 (25.0 - 47.9)	48.1 (34.9 - 56.2)
% in stringent/near control for SBP (inter-facility range)	66.5 (56.7 – 77.0)	77.5 (68.5 – 84.6)
% of poorly controlled receiving treatment intensification within 3 months (inter-facility range)	47.7 (40.2 - 53.8)	54.4 (47.3 – 61.1)
Hyperlipidemia: Total N (inter-facility range)	211,257 (594 – 13,708)	321,549 (875 – 19,669)
Mean Age (standard deviation) (inter-facility range)	62.7 (11.9) (58.2 – 67.9)	62.5 (12.5) (58.6 – 68.0)
% Female (inter-facility range)	45.7 (40.1 – 51.5)	47.4 (43.2 – 51.4)
% African American (inter-facility range)	7.7 (0.4 - 34.0)	8.2 (0.1 – 35.7)
% High risk (inter-facility range)	60.0 (51.7 – 65.8)	59.9 (53.6 – 65.1)
% Moderate risk (inter-facility range)	21.5 (17.6 – 25.5)	22.6 (19.9 – 25.6)
Mean LDL-c in mg/dL (standard deviation) (inter-facility range)	126.7 (38.7) (120.1 – 132.1)	118.2 (36.2) (113.1 – 122.7)
% in stringent control for LDL-c (inter-facility range)	34.6 (30.4 – 41.1)	44.8 (37.9 – 53.9)
% in stringent/near control for LDL-c (inter-facility range)	67.5 (62.7 – 73.9)	76.5 (69.2 – 82.3)
% of poorly controlled receiving treatment intensification within 3 months (inter-facility range)	45.5 (37.0 – 52.1)	46.9 (40.3 – 56.0)
Diabetes: Total N^* (inter-facility range)	128,101 (240 – 8,124)	151,401 (313 – 9,366)
Mean Age (standard deviation) (inter-facility range)	60.7 (13.2) (56.6 – 68.7)	61.2 (13.3) (57.8 – 67.2)
% Female (inter-facility range)	46.7 (40.6 – 52.1)	47.0 (40.4 – 52.5)
% African American (inter-facility range)	11.9 (0.4 – 49.1)	11.4 (0.4 – 47.9)
Mean Hb A1c in % (standard deviation) (inter-facility range)	7.9 (1.8) (7.4 – 8.4)	7.3 (1.5) (6.9 – 7.5)
% in stringent control for Hb A1c (inter-facility range)	32.6 (25.9-44.6)	51.1 (45.2-62.0)
% in stringent/near control for Hb A1c (inter-facility range)	61.5 (51.0 – 72.1)	77.1 (70.5 – 86.3)
$\%$ of poorly controlled patients receiving treatment intensification within 3 months (inter-facility range) $\!\!^*$	56.3 (49.1-63.7)	56.9 (48.3 – 66.1)

Patients on insulin during 4 months before elevated value are excluded from all analyses

Selby et al.

Odds Ratios and Predicted Differences in Probability of Control at the end of 2003 Associated with 5% Differences in Treatment Intensification from 2001 to 2003 – from Two-Level† Multivariable Logistic Regression Models Table 3

Facility-Level Characteristics	Likelihood Con	Likelihood of Risk Factor Control, 2003	Predicted Probability of Control	Predicted Probability of Control, End of 2003, at Facility with Average Increase and a 5% Greater than Average Increase in Treatment Intensification from 2001-2003	erage Increase and a 5% Greater on from 2001-2003
	\mathbf{OR}^{\dagger}	p-value	Average Increase	5% Greater Increase	Difference (95% CI)
Systolic Blood Pressure $(n = 478,320$ patients with hypertension)	hypertension)				
Change in Treatment Intensification 2001-2003 (1% increase)	1.018	0.001	85.4%	86.5%	1.1% (0.51 – 1.63%)
Treatment Intensification Rate in 2001 (1 % increase)	1.015	0.007			
% in Control in 2001 (1% increase)	1.018	<0.001			
LDL-cholesterol ($n = 321,549$ patients with hyperlipidemia)	ipidemia)				
Change in Treatment Intensification 2001-2003 (1% 1.012 increase)	1.012	0.03	77.2%	78.2%	1.1% (0.15 – 1.94%)
Treatment Intensification Rate in 2001 (1% increase)	1.006	0.225			
% in Control in 2001 (1% increase)	1.031	<0.001			
Hemoglobin A1c $(n=151,401)$ patients with Diabetes mellitus)	s mellitus)				
Change in Treatment Intensification 2001-2003 (1 % increase)	1.025	0.0001	81.1%	82.9%	1.8% (1.0 – 2.6%)
Treatment Intensification Rate in 2001 (1% increase)	1.017	0.003			
% in Control in 2001 (1% increase)	1.024	<0.0001			

†
For each condition, the two-level hierarchical model included random effects for facility and provider, the three fixed facility-level predictors shown in the table, and the following patient-level variables: age, sex, race/ethnicity, DxCG DCG score, presence of either or both of the other two conditions, prior CHD (hyperlipidemia model only), or prior cardiovascular or target organ disease (hypertension model only). Page 16

Condition	Spearman Rank Correlations	Mean Absolute Change in Rank (SD)	% of the 35 Facilitie changing by 5 ran positions
Hypertension	0.96	2.34 (1.83)	14.2%
Hyperlipidemia	0.67	6.34 (5.29)	51.4
Diabetes Mellitus	0.93	3.09 (2.33)	28.6

 $^{^{\}dot{7}} \text{Control defined as stringent/near control.}$