COMMENTS AND RESPONSES

Cost-Effectiveness of Clopidogrel plus Aspirin versus Aspirin Alone

TO THE EDITOR: As Schleinitz and Heidenreich pointed out (1), their finding that combination antiplatelet therapy with clopidogrel and aspirin in the first year following an acute coronary syndrome "represents good value according to traditional limits of cost-effectiveness" has serious economic implications if adopted into practice. Although decision analytic models offer valuable insight into treatment decisions, such as the observation that the first month of treatment is much more cost-effective than subsequent months, the simplifying assumptions required for modeling can lead to biased conclusions when comparing the cost-effectiveness of an intervention with an external benchmark. Before making treatment or policy decisions on the basis of a decision model, it is important to examine the key assumptions. In this analysis, the authors apply the 20% reduction in overall vascular events seen in the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial (2) equally to nonfatal myocardial infarctions, strokes, and death from cardiovascular causes. Although patients randomly assigned to clopidogrel did experience a 23% reduction in nonfatal myocardial infarction, the reductions in stroke (14%) and cardiac deaths (7%) did not reach statistical significance. This finding parallels observations of warfarin therapy in acute coronary syndromes (3, 4). In CURE, the 95% CI for the relative risk for cardiac death extended from 0.79 to 1.08, making a 20% reduction unlikely. The authors did perform sensitivity analysis, but the minimum risk reduction considered was 10%, greater than the 7% reduction in cardiovascular mortality seen in the trial.

Because there was no long-term quality adjustment for myocardial infarction, any gain in quality-adjusted life-years had to come from decreasing rates of stroke and cardiac death. As neither of these outcomes alone nor the combination of the 2 showed statistically significant decreases with clopidogrel, more data are needed to know the true cost-effectiveness of the treatment. In the meantime, a Monte Carlo analysis using a different relative risk reduction for each outcome based on its 95% CI would offer a truer estimate of the cost-effectiveness of clopidogrel than what has been presented.

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Potential Financial Conflicts of Interest: None disclosed.

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TO THE EDITOR: Although I read Schleinitz and Heidenreich's article (1) with interest, I am concerned about their conclusion. They stated, "In patients with high-risk acute coronary syndromes, 1 year of therapy with clopidogrel plus aspirin results in greater life expectancy than aspirin alone, at a cost within the traditional limits of cost-effectiveness." There are evidently better ways to be cost-effective.

In the CURE trial (2), patients with an acute coronary syndrome without ST-segment elevation received clopidogrel or placebo, in addition to aspirin, for a mean of 9 months. Nonetheless, most ischemic events (cardiovascular death, myocardial infarction, or stroke) occurred early, and there was basically no difference in the rates of events between the clopidogrel and placebo groups after the first 3 months. Approximately 85% of the total benefit achieved after 1 year with clopidogrel was observed already by 3 months (2, 3). The excess risk for bleeding with dual antiplatelet therapy is constant, however, which unfavorably changes the risk–benefit ratio when clopidogrel is given over the long term (4). If clopidogrel is used for 3 months rather than 1 year, most of the benefit of the drug would be achieved and bleeding hazards as well as costs of therapy would be reduced by approximately 75%. This would be a judicious compromise among clinical efficiency, side effects, and economy.

There is an even more provocative interpretation of the CURE study. The investigators showed that there was no advantage of clopidogrel over placebo in 3109 patients when the dose of aspirin was 101 to 199 mg (relative risk, 0.97 [95% CI, 0.77 to 1.22]) (5). Obviously, this observation must be confirmed in a prospective study.

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IN RESPONSE: Dr. Rothberg suggests that our analysis would have been stronger if we had used outcome-specific estimates of the efficacy of clopidogrel when added to aspirin. We agree in theory and

have used this approach in an analysis of clopidogrel monotherapy (1). The CURE trial (2), however, did not report the numbers of each type of event that made up the composite outcomes. The cardiovascular death rate cited by Rothberg, for example, is itself a composite of deaths due to stroke, myocardial infarction, and other cardiovascular causes. We felt that the potential to introduce bias by estimating event rates for aspirin monotherapy as well as combination therapy and using these calculations to approximate outcome-specific efficacy was too great to justify this method.

Dr. Eriksson emphasizes our point that balancing protection from thrombotic events with the risk for hemorrhage is critical in determining the benefit of adding clopidogrel to aspirin, and thereby its cost-effectiveness. Assuming constant relative efficacy of combination therapy, we found that the diminishing absolute benefit in thrombotic event rates is negated by the constant risk for hemorrhage after 5 years.

Cost may help specify the "ideal" duration of therapy in 1 of 2 ways: at the average individual level, as reflected by the incremental cost-effectiveness ratio, or at the societal budgetary level, a function of both the incremental cost-effectiveness ratio and the size of the affected population. Variation in both societal preference and available resources means that this "ideal" duration may differ among countries.

Eriksson also notes that in a post hoc evaluation, the efficacy estimate of adding clopidogrel to aspirin varied with the dose of aspirin (3). Aspirin dose was determined at the physician's discretion. We have shown that efficacy estimates of antiplatelet therapies derived from nonrandomized studies may be statistically distinct from results of randomized comparisons (4), and we agree that a randomized comparison is warranted to clarify the optimal dose of aspirin for combination therapy.

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CLINICAL OBSERVATIONS

Leukopenia and Thrombocytopenia Caused by Thiazolidinediones

TO THE EDITOR: *Background:* Thiazolidinediones are orally administered drugs used to control hyperglycemia in patients with type 2 diabetes mellitus (1). Although mild dilutional anemia is often seen with thiazolidinedione therapy, no adverse effects of thiazolidinedione on leukocytes or platelets have been described.

Objective: To report the first case, to our knowledge, of a patient who developed leukopenia and thrombocytopenia while taking currently approved thiazolidinediones, rosiglitazone, and pioglitazone, given sequentially.

Case Report: A 50-year-old woman with a long-standing history of type 2 diabetes mellitus presented with fatigue in the summer of 2003. Her diabetes had been diagnosed in 1988 and was initially managed with lifestyle modification and glipizide, a sulfonylurea compound. Previous routine laboratory evaluation had been unremarkable, with the exception of transient mild asymptomatic thrombocytopenia (platelet count, 145×10^9 cells/L [normal range, 150 to 400×10^9 cells/L]). In February 2002, rosiglitazone, 4 mg once daily, was added to glipizide, and the dose was increased 6 months later to 8 mg once daily. In July 2003, the patient reported a 2-month history of fatigue and leg cramping. Findings on physical examination were normal. Laboratory evaluation was notable for a hemoglobin A_{1c} level of 7.8%, a leukocyte count of 2.5×10^9 cells/L (normal range, 4 to 11×10^9 cells/L), an absolute neutrophil count of 1.4×10^9 cells/L (normal range, 1.5 to 7.5×10^9 cells/L), an absolute lymphocyte count of 0.7×10^9 cells/L (normal range, 1 to 4×10^9 cells/L), a hematocrit of 0.34 (normal range, 0.32 to 0.45), and a platelet count of 105×10^9 cells/L. Although the patient had been treated with a sulfonylurea compound for several years, glipizide therapy was discontinued because of reports of cytopenia associated with sulfonylurea use. However, the patient's cytopenia persisted and she was referred to the hematology department for further evaluation.

Results of peripheral blood smear analysis and serum protein electrophoresis were both normal. A bone marrow biopsy showed hypocellular bone marrow with normal tri-lineage maturation; metaphase cytogenetics were normal. Rosiglitazone was withdrawn, and glipizide therapy was restarted. Approximately 1 month later, both leukocyte count and platelet count had improved, and by December 2003, the patient's leukocyte count (4.4×10^9 cells/L) and platelet count (151×10^9 cells/L) had normalized.

In March 2004, the patient's glycemic control remained suboptimal, and pioglitazone, 30 mg once daily, was added. Three months later, in June 2004, a complete blood count showed a leukocyte count of 2.7×10^9 cells/L, a hematocrit of 0.36, and a platelet count of 119×10^9 cells/L. Pioglitazone was immediately withdrawn. Within 2 months, the patient's leukocyte count normalized (4.4×10^9 cells/L) and her platelet count had improved (142×10^9 cells/L). Five months later, her leukocyte count (4.1×10^9 cells/L) and platelet count (153×10^9 cells/L) remained normal.

Discussion: This case satisfies many of the factors that suggest a drug-related adverse reaction (2): low likelihood of the event in a patient with diabetes, absence of prodromal symptoms or signs of the

adverse event before drug exposure, abatement of the adverse reaction with withdrawal of the drug, and recurrence of the event on rechallenge with a different agent from the same class. The adverse reaction probability scale by Naranjo and colleagues (3) also indicated a probable adverse reaction due to thiazolidinedione. The mechanism for the observed effect of thiazolidinedione on leukocyte and platelet counts is unclear. Thiazolidinedione activation of peroxisome proliferator–activated receptor-γ receptors in hematopoietic cells may explain the adverse effect we observed (4). Our patient previously had a normal leukocyte count and hematocrit, but her platelet count had been subnormal on 1 occasion before beginning thiazolidinedione therapy. It is possible, therefore, that the observed effect of thiazolidinedione on platelets may be of significance only in individuals who have or are at risk for cytopenia. Our patient may also have a genetic polymorphism in peroxisome proliferator–activated receptor-γ receptors, making her more susceptible to a suppressive effect of thiazolidinedione on bone marrow than the general population.

Conclusion: Clinicians should be aware that thiazolidinedione-associated leukopenia and thrombocytopenia can occur, especially in patients with or at risk for cytopenia.

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New Onset of Myelofibrosis in Association with Pulmonary Arterial Hypertension

TO THE EDITOR: *Background:* Marrow fibrosis (myelofibrosis) occurs in patients with idiopathic myelofibrosis and other myeloproliferative disorders, which are characterized by a clonal hematopoiesis (1), and in patients with lupus erythematosus and cancer involving marrow. However, to our knowledge, myelofibrosis has not been previously reported in patients presenting with pulmonary arterial hypertension. The observation of severe myelofibrosis in a patient with this disorder prompted us to systematically evaluate patients with pulmonary arterial hypertension and some evidence of hematologic abnormality (for example, thrombocytopenia or anemia). Clonal proliferation of endothelial cells has been shown to occur in idiopathic

Table. **Outcomes of Study Patients**

Outcome	Pulmonary Hypertension Group (n = 17), n/n	Controls (n = 27), n/n
Fibrosis*		
Present	17/17	5/27
Absent	0/17	22/27
Degree of fibrosis		
Severe	11/17	0/27
Moderate	3/17	0/27
Mild	3/17	5/27
Clonality†		
Polyclonal	14/15	127/128‡
Clonal	1/15	1/128‡

* $P < 0.001$ (Fisher exact 2-tailed test).
† 2 patients were not informative for clonality studies.
‡ Expected probability of "pseudoclonality" was based on published data using the analyses used here (5).

pulmonary arterial hypertension (2). Therefore, we performed clonality studies to differentiate a clonal hematopoiesis (favoring a diagnosis of idiopathic myelofibrosis) (3) from polyclonal hematopoiesis (indicating a secondary reactive process).

Objective: To determine whether patients with pulmonary arterial hypertension demonstrate myelofibrosis and to differentiate primary clonal proliferation in the marrow from a secondary reactive process by determination of clonal hematopoiesis.

Methods and Findings: We enrolled 17 consecutive women with pulmonary arterial hypertension in a protocol approved by our institution's institutional review board. Twenty-seven consecutive patients, who had marrow biopsies performed for anemia or staging of solid tumors and who did not have conditions known to be associated with marrow fibrosis, served as controls for the histologic evaluation of the marrow for fibrosis. Three hematologists and hematopathologists blindly and independently analyzed all of the marrow biopsies and graded the presence and severity of fibrosis on sections stained with reticulin stain. Clonality studies were done on granulocytes and platelets from the peripheral blood by X-chromosome transcriptional polymorphism analyses (4).

Seventeen women, 10 with primary pulmonary arterial hypertension and 7 with pulmonary arterial hypertension secondary to a connective tissue disease, were enrolled in the study. Of the 7 women with connective tissue disease, 3 had scleroderma, 2 had mixed connective tissue disease, 1 had polymyositis, and 1 had lupus erythematosus. The median age was 46 years (range, 27 to 61 years). All patients had severe pulmonary vascular disease (New York Heart Association functional class III or IV). The median systolic pulmonary artery pressure was 80 mm Hg (range, 51 to 141 mm Hg), and median duration of pulmonary arterial hypertension was 49 months (range, 1 to 155 months). Patients were anemic (range of hemoglobin level, 86 to 120 g/L), had a low platelet count (range, 14 to 149×10^9 cells/L), or both.

Myelofibrosis was seen in all patients with pulmonary arterial hypertension (**Table**). Eleven had severe fibrosis, 3 had moderate fibrosis, and 3 had mild fibrosis. In contrast, none of the 27 controls had severe or moderate fibrosis and 5 had mild fibrosis ($P < 0.001$ [2-tailed Fisher exact test]). Of 17 studied patients, 15 were heterozygous for at least 1 of the studied X-chromosome transcriptional

polymorphisms (4) and were thus suitable for clonality studies. Fourteen of 15 women expressed both X-chromosome allelic transcripts in both granulocytes and platelets (polyclonal hematopoiesis); only 1 expressed single transcript.

Discussion: To our knowledge, this study is the first to show that myelofibrosis is common and may contribute to impaired hematopoiesis in both primary and secondary pulmonary arterial hypertension. Fourteen of 17 patients showed moderate or severe fibrosis, which was not seen in the bone marrow of any control. The uncommon occurrence of both idiopathic myelofibrosis and pulmonary arterial hypertension makes it unlikely that all 17 patients had both diseases simultaneously. More conclusively, platelets and granulocytes were polyclonal in 14 of 15 patients with pulmonary arterial hypertension. Using the same assays, we have shown that platelets and granulocytes are clonal in patients with the related myeloproliferative disorders polycythemia vera and essential thrombocythemia. It is plausible but not certain that epoprostenol therapy may cause myelofibrosis. All but 2 of our patients received this agent. However, it is also possible that the processes that initiate and propagate pulmonary vascular injury resulting in pulmonary arterial hypertension may also result in myelofibrosis.

Conclusion: Myelofibrosis is present and often unrecognized and unreported in patients with pulmonary arterial hypertension. It is not a primary neoplastic hematopoietic disorder. Its mechanism, management, and prognostic and pathologic significance remain to be elucidated.

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Potential Financial Conflicts of Interest: None disclosed.

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Improving the Introductions of Manuscripts

TO THE EDITOR: *Background:* Introductions of manuscripts are crucial because they place clinical problems into context. The importance of a clinical problem is influenced by its incidence, morbidity, mortality, and costs. Quantifying these factors requires attention to denominators. For example, stating that some number of hospital admissions results from heart failure does less to quantify this issue compared with other illnesses than does also including the total number of annual hospital admissions.

Objective: To examine the use of quantitative information in the introductions of published clinical studies.

Methods and Findings: We reviewed clinical reports in 4 clinical journals. Two were general medical journals (the *Journal of the American Medical Association* [JAMA] and the *New England Journal of Medicine*), and 2 were emergency medicine journals (*Academic Emergency Medicine* and *Annals of Emergency Medicine*). Publications were included if they reported the original results of a clinical study. Although the CONSORT (Consolidated Standards of Reporting Trials) statement is specifically directed at randomized clinical trials, we included other study types to broaden our scope.

Statements in introductions were categorized as general, denominator, or numerator statements, depending on whether they were only descriptive and nonquantitative, included an explicit or implicit denominator, or included only a numerator or raw number, respectively.

Typical denominator statements included the following: "Anemia is common in the elderly, occurring in 24 to 40 percent of hospitalized patients" (1) and "Neuroblastoma . . . affects 1 in 7,000 children" (2). In both examples, the authors provide denominator information, either implicitly (in the form of a percentage) or explicitly. In contrast, numerator statements provide only a single specific number, for example, the number of people who experience sudden cardiac arrest each year. A typical numerator statement would be, "The Office of National Drug Control Policy estimates that in 1998 there were 3.3 million chronic cocaine users" (3). We categorized each article according to the highest level of information provided, with denominator taking precedence over numerator and numerator taking precedence over general statements.

Between 1 August 2001 and 31 July 2002, 600 clinical studies were published in the 4 journals: 240 in *JAMA*, 199 in the *New England Journal of Medicine*, 86 in *Academic Emergency Medicine*, and 75 in *Annals of Emergency Medicine*. Overall, 32% of introductions included denominator information, 13% included only numerator information, and 55% included only nonquantitative general statements (**Table**).

Discussion: While the importance of denominator information is well established for many aspects of the reporting of clinical trials (4),

Table. **Introductions of Manuscripts in 4 Medical Journals***

Journal	Denominator Statements, *n* (%)	Numerator Statements, *n* (%)	General Statements, *n* (%)	Total, *n* (%)†
Academic Emergency Medicine	23 (27)	14 (16)	49 (57)	86 (100)
Annals of Emergency Medicine	23 (31)	9 (12)	43 (57)	75 (100)
JAMA	78 (33)	36 (15)	126 (53)	240 (100)
New England Journal of Medicine	69 (35)	18 (9)	112 (56)	199 (100)
Total	193 (32)	77 (13)	330 (55)	600 (100)

* Journals are listed in alphabetical order. *JAMA = Journal of the American Medical Association.*
† These values are used as the denominator for each row.

the same standard is not applied to the information used to put these clinical trials into context. Only 32% of published clinical studies included implicit or explicit denominator information in the introduction, and over half of the publications did not include any quantitative information at all. Our findings demonstrate an opportunity to standardize information included in the introductions of published reports of clinical research.

Conclusion: We believe explicit denominator information, meaning the inclusion of a specific number, is inherently more meaningful than the implicit inclusion of a denominator in a proportion or percentage. For example, a statement that a certain percentage of patients has a disease is of unclear significance without knowing the total number of patients from which the percentage was derived.

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CORRECTION

Correction: Meta-Analysis: Surgical Treatment of Obesity

In the clinical guideline by Maggard and colleagues on the surgical treatment of obesity (1), an author's name was misspelled. The fifth author's name is Harvey J. *Sugerman*, not "Sugarman."

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