

Balancing “No Blame” with Accountability in Patient Safety

TO THE EDITOR: Wachter and Pronovost (Oct. 1 issue)¹ question the “no-blame” paradigm in patient-safety improvement and suggest the adoption of explicit punitive approaches to poorly performing physicians. We counsel caution. In a longitudinal study over a 2-year period in a large facility,² we found that penalties did not deter undesirable behavior. Rather, penalties drove evidence of non-compliance underground, encouraging people to conceal it and thus perversely reducing accountability.

Drawing the line between blameworthy and blameless acts was difficult and involved subjective judgments of observers about the foreseeability of harm, reasonable care, and prudence. The question was: Who was permitted to draw that line?³ And who reported “violations”? In the example of hand hygiene described in the article, those difficulties are compounded by uncertainties in the evidence base about when and how hands should be washed.⁴

In our study, peer intervention was more effective in generating accountability and desired change than punitive administrative action; less blame led to more accountability. Our research clearly suggests that by demanding penalties, we might stifle accountability rather than enhance it.

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No potential conflict of interest relevant to this letter was reported.

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TO THE EDITOR: With regard to the criteria listed in Table 1 of the article by Wachter and Pronovost, who gets to decide what is a critical “patient-safety practice”? The Institute for Healthcare Improvement and others in the patient-safety indus-

try made a very big point of maintaining blood glucose target ranges of 80 to 110 mg per deciliter (4.4 to 6.1 mmol per liter) in patients in critical care units. Many clinicians who opposed this recommendation were told that they were simply not keeping up with evidence-based medicine. The Normoglycemia in Intensive Care Evaluation–Survival Using Glucose Algorithm Regulation study (ClinicalTrials.gov number, NCT00220987) proved quite definitively that this goal not only did not help, but actually caused excess deaths as compared with looser glucose control. The flip-flop by the Centers for Medicare and Medicaid Services on beta-blocker use immediately after myocardial infarction is another example in which what was advertised as beneficial was actually harmful. A very clear definition of what is truly a patient-safety practice, scientific criteria, and certainty of the evidence are needed to mandate a clinical practice. If not, we will continue to violate the ancient creed of “do no harm” in misguided safety efforts.

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THE AUTHORS REPLY: We agree that we need to proceed cautiously and err on the side of parsimony in choosing practices that are suitable for an accountability approach. Candidate practices should be relatively easy to follow, have a strong and enduring evidence base, and be ones in which other approaches have been tried and not succeeded. We believe that infection-control practices such as hand hygiene easily meet these criteria and are ideal practices to start with. However, at this point, clinical care standards such as tight glucose control and the use of certain medications at appropriate times seem more suitable for approaches that use education, traditional audit and feedback, and computerized decision support.

Our auditing methods will also need to mature. Although some auditing can and should be done by colleagues (promoted by team training and a shared ethic of patient protection), it is human nature for colleagues to avoid “ratting out” each other, particularly when there are penalties at

hand. The solution is not to abandon accountability, but rather to develop stronger auditing strategies with the use of methods such as video surveillance, computerized triggers, and unannounced, secret monitoring of compliance by hospital personnel. Clearly, we have much to learn here, and we agree that we must be careful to preserve the collegial exchange and openness that are so essential to organizational learning.

We recognize that finding the balance between accountability and “no blame” will be difficult. But, a decade into the safety movement, we now know that our present strategy guarantees lackluster adherence to a number of low-risk, universally accepted, and evidence-based safety prac-

tices such as hand hygiene. Without minimizing the challenges we face and fully recognizing the need to proceed slowly, it borders on magical thinking to believe that a strategy of “more of the same” will achieve the levels of safety and reliability that our patients deserve.

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Since publication of their article, the authors report no further potential conflict of interest.

Lovastatin in X-Linked Adrenoleukodystrophy

TO THE EDITOR: As reported previously in the *Journal*, lovastatin lowers levels of very-long-chain fatty acids in plasma in patients with X-linked adrenoleukodystrophy (X-ALD).¹ Further studies did not reproduce this finding with the use of simvastatin in patients or with the use of lovastatin in X-ALD–knockout mice.^{2,3} Still, many patients with X-ALD worldwide receive lovastatin.

We conducted a randomized, double-blind, placebo-controlled, crossover trial comparing lovastatin at a dose of 40 mg once daily with placebo (Current Controlled Trials number, ISRCTN31565393). Outcome measures were levels of hexacosanoic acid (C26:0) in plasma, low-density lipoprotein (LDL) particles, lymphocytes and erythrocytes, and plasma LDL cholesterol after 22 weeks of treatment. For some outcome measures, an intermediary analysis at 8 weeks was performed. A total of 14 men with X-ALD (adrenomyeloneuropathy phenotype) were enrolled in the study. Merck provided lovastatin for this study but did not participate in the trial design, data analysis, or drafting of the letter.

No patients dropped out of the study, and neither myopathy nor rhabdomyolysis or other adverse events were observed. Data on all major outcomes are summarized in Table 1. There were significant decreases of 1.44 mmol per liter in the level of LDL cholesterol in plasma at 8 weeks and 1.35 mmol per liter at 22 weeks. At 8 weeks, the levels of plasma tetracosanoic acid (C24:0) and C26:0 had decreased by 14.2 μ mol per liter and

0.39 μ mol per liter, respectively. However, even with this decrease, C26:0 levels remained above the control level (mean \pm SE) of 0.67 ± 0.13 μ mol per liter.⁴ Furthermore, the reduction in C26:0 was no longer significant at 22 weeks. There was a decrease of 0.38 mmol per liter in the level of oleic acid (C18:1) at 8 weeks and a decrease of 0.44 mmol per liter at 22 weeks. There was no change in levels of C26:0 in erythrocytes or lymphocytes at either measurement. Finally, the levels of C18:1, C24:0, and C26:0 in LDL lipoprotein particles remained unchanged.

This trial was designed to investigate whether lovastatin has a biochemical effect in vivo in patients with X-ALD and to provide pilot data for a possible large-scale trial with clinical outcome variables. We conclude that lovastatin leads to a small decrease in levels of C24:0 and C26:0 in plasma; this must be considered a nonspecific result of the decrease in the level of LDL cholesterol. Since very-long-chain fatty acids are virtually water insoluble, and only a small fraction binds to albumin,⁵ most of the very-long-chain fatty acids in plasma are transported as cholesterol esters in lipoprotein particles such as LDL. This finding is corroborated by the finding that the level of C18:1 was also reduced, and it is further supported by the lack of an effect on C26:0 levels in peripheral-blood lymphocytes and erythrocytes and in the content of very-long-chain fatty acids in the LDL lipoprotein fraction. Our data indicate that investment of substantial