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[Three of the authors respond:]

Michael Klein argues against our conclusion[1] that planned cesarean section was safer and less expensive than planned vaginal birth during the period reported in the Term Breech Trial. He bases his arguments on other studies by us,[2,3] which "showed no difference in outcome for the babies or the mothers" at 2-year follow-up. He also claims that by looking only at the duration of the Term Breech Trial we have "vastly underestimate[d] the real costs of elective cesarean for breech or any birth." While we agree that a longer-term analysis might be useful, we disagree with these arguments.

The argument that our own studies show no difference at 2 years represents a misunderstanding of the results of those trials.[2,3] The appropriate interpretation of those results is that the benefits of planned cesarean section are limited to reducing perinatal and neonatal mortality and serious neonatal morbidity during the first 6 weeks of life. These remain important benefits for the baby, the mother, the family and the health care providers.

Regarding the question of what will happen to the costs of planned cesarean section and planned vaginal birth after, say, 2 years, the answer is "we do not know." Any argument that the costs will be higher is nothing more than speculation. For example, we agree with the assumption that most women will have more than one birth, but we do not know if breech presentation will occur for the first birth, the last birth or a birth in between. Also, our experience from the Term Breech Trial has taught us that until actual resource utilization is measured in a controlled environment, it is not easy to predict what will happen. We thought that planned cesarean section would be more expensive than planned vaginal birth, but found that it was not. Furthermore, there was no single specific factor that explained why the costs of planned cesarean section were lower, which tells us that it is dangerous to try to predict (rather than measure) future costs.

Finally, we do not feel responsible for the headlines and content of what is published in the popular press. The Interpretation section of our paper discusses the study's limitations and the consequent constraints on any conclusions drawn.

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Multitherapy for diabetes

Julie Ménard and associates[1] used pravastatin or bezafibrate (or both) as a component of intensive multitherapy in their study of patients with type 2 diabetes mellitus. Although pravastatin is of questionable benefit (according to the ALLHAT-LLT study,[2] in which no cardiovascular, peripheral vascular, cerebrovascular or mortality benefits were found), my main concern here is with the use of fibrates.

Fibrates, including bezafibrate, are effective in lowering low-density lipoprotein cholesterol and triglycerides while raising high-density lipoprotein cholesterol, but there was no mortality benefit in a large bezafibrate trial.[3] In addition, there have now been 3 trials with different fibrates (gemfibrozil,[4] clofibrate[5] and fenofibrate[6]) that ended with numerically more deaths in the group receiving fibrates than in the placebo group.

Fibrates lull patients and doctors into a false sense of accomplishment by bringing several blood lipid markers closer to guideline targets, thus reducing the urgency of more difficult dietary and lifestyle changes. However, with "over two-thirds of patients with diabetes [dying] of cardiovascular causes,"[7] the established failure of fibrates to lower mortality should lead to an urgent call to stop their use and to examine the clinical efficacy of the lipid guidelines.

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[Two of the authors respond:]

Eddie Vos questions the use of fibrates for patients with type 2 diabetes mellitus. In our trial,[1] 9 patients were given fibrates, in accordance with recommendations of the Canadian Diabetes Association (CDA).

Use of statins or fibrates has been the subject of debate for some time. Statin trials in patients with diabetes have provided convincing evidence of a substantial benefit stemming from this class of drugs. Trials have been conducted with gemfibrozil,[2,3] bezafibrate,[4] clofibrate[5] and fenofibrate.[6] Some of these had positive results in terms of primary prevention (Helsinki Heart Study[2] and a World Health Organization study[5]) and secondary prevention (Veterans Affairs High-Density Lipoprotein Cholesterol Intervention Trial [VA-HIT][3]). However, the results of the Bezafibrate Infarction Prevention (BIP) trial[4] and the FIELD trial[6] were mixed: positive outcomes were observed only in certain subgroups, raising reservations related to an increase in noncardiovascular mortality. Reasons for the differences in outcomes were not immediately apparent.

It emerged in post hoc subgroup analyses of data from the Helsinki Heart Study,[7] the VA-HIT[8] and the BIP study[9] that fibrate-induced reductions in cardiovascular events were especially pronounced (on the order of 30%–50%) in subjects with evidence of insulin resistance or other features of metabolic syndrome, such as dyslipidemia and increased body weight, or in people with both diabetes and preexisting cardiovascular disease. These results were not found in the FIELD study.

The observation that the cardioprotective effects of gemfibrozil were substantially greater than those of other fibrates may be no more than fortuitous or may reflect differences in the populations studied. However, it is also possible that gemfibrozil has either protective properties that are lacking in other fibrates, or that other fibrates have adverse properties not shared by gemfibrozil.

Accordingly, the debate continues, more research is needed, and whether or not diabetes associations will alter their clinical recommendations is an open question. At present, the FIELD study has introduced doubts but not enough evidence to change the current guidelines.

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The "Editor's Take" on the article by Julie Ménard and associates[1] about intensive multitherapy for patients with diabetes was both discouraging and baffling. Russell Rothman and Tom Elasy, in their accompanying commentary,[2] correctly state that "Ménard and colleagues ... demonstrate again that an intensive disease management program can improve glycemic control and cardiovascular risk factors in patients with poorly controlled diabetes." The fact that patients were unable to sustain these improvements when intensive therapy was stopped is hardly surprising. This small trial is best seen as a proof-of-concept study that adds to the literature showing that intensive multifactorial and interdisciplinary treatment improves patient outcomes.[3–5] Rather than pointing to the need to establish health care teams and systems of care that are sustainable over the long term, the Editor advises that, "physicians should expect few of their patients to attain CDA goals and even fewer to maintain the goals over extended periods."

The Editor also questions whether CDA guideline targets are realistic. It is important to note that the CDA metabolic and blood pressure targets are based on evidence and reflect thresholds for improved patient outcomes. These are clinical goals for best practice. Even if targets are not achieved, the evidence also points to the benefits of incremental improvements in blood glucose levels, blood pressure and lipids.

The defeatist attitude reflected in the Editor's comments does little to motivate physicians to advance diabetes care in this country. Physicians should continue to strive to achieve evidence-based targets, as the literature has clearly demonstrated that the serious complications of diabetes can be delayed or averted by this clinical approach. Our patients deserve nothing less.

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