discouraging children from smoking until randomized trials in which large numbers of teenagers are assigned to smoking or nonsmoking groups showing that those who smoke experience greater long-term mortality rates. To extend this train of thought even further, we should presumably not ban drunk driving until randomized trials have demonstrated that it is dangerous.

Many of the preventive medical manoeuvres currently in use will never be supported by data from randomized trials. In the 3 examples outlined above, randomized trials would be unethical even if they were possible. The accumulated evidence from nonrandomized studies for the benefits of seat belts, the harmful effects of smoking and the dangers of drunk driving is so vast that further study would be in no one's best interest (except perhaps the tobacco industry).

The wearing of seat belts and the avoidance of smoking and drunk driving are measures that cannot conceivably be harmful. The nature of the evidence we require before advocating a preventive medical intervention depends on the nature of the intervention. A pharmacological intervention is vastly different from a lifestyle intervention, and the quality of the evidence we require may also be vastly different.

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[The author responds:]

To the extent that Gabe Slowey and David Rapoport hold globally negative views on personal preventive measures and because Nicholas Forbath assigns me a nihilistic view of them, I disagree with all 3 of these correspondents.

During my clinical and health policy years I advocated and applied a wide array of personal preventive manoeuvres because I was dedicated, not to global conclusions about the value of preventive medicine, but to methods for generating level 1 evidence[1] as to whether its individual elements did more good than harm (by level 1 evidence I mean either systematic reviews of randomized trials or "all-or-none" evidence by which, for a universally fatal condition, an intervention was followed by survival or a less frequent adverse outcome was completely eliminated by the intervention). On that basis I advocated and practised the vigorous detection and treatment of certain levels of symptomless elevated blood pressure,[2] never ordered testing of prostate-specific antigen in a symptomless man, and changed my practice and teaching about treating hypercholesterolemia from a negative to a positive stance when the accumulating evidence from randomized trials of statin drugs showed that they did more good than harm.

In response to Mark Taylor, because the absence of proof is not the proof of absence, folks like me don't advocate abandoning established practices just because they haven't been tested in randomized controlled trials. Moreover, seat belt use satisfied the second criterion for level 1 evidence as soon as users began to survive auto crashes that were previously uniformly fatal. Importantly, however, when this same criterion is applied to another auto safety tradition, school-based drivers' education, the level 1 evidence shows that this intervention doesn't create better drivers, only younger ones, and its net effect appears to be harmful.[3]

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Weighing the risks and benefits of tamoxifen

I read with interest Eric Wooltorton's article on tamoxifen for breast cancer prevention,[1] which directly followed a summary of the Women's Health Initiative (WHI) study on hormone replacement therapy (HRT).[2] The latter study was stopped early because the prespecified upper boundary for risk of breast cancer in the HRT group had been exceeded. To paraphrase Table 1 in Wooltorton's article,[1] it appears that, per 10 000 woman-years, tamoxifen was associated with 15 more cases of endometrial adenocarcinoma, 2 more cases of uterine sarcoma, 4 more cases of stroke and 5 more cases of pulmonary embolism (relative to placebo), for a total of 26 additional events or a 1.3% absolute risk increase over the 5-year period of the National Surgical Adjuvant Breast and Bowel Project (NSABBP). Tamoxifen was associated with fewer cardiovascular problems than reported for HRT in the WHI study,[2] but HRT did not cause any increase in endometrial cancer.

In the NSABP, the relative risk reduction for breast cancer among high-risk women who received tamoxifen was 49%.[3] Perhaps the Gail model for identifying women at high risk of breast cancer[4] could be modified to incorporate the known risks associated with tamoxifen, adjusted according to the patient's clinical characteristics, such as age, ethnic background and smoking status, to arrive at a net risk-to-benefit ratio. Without such a tool, it is difficult to get an accurate estimate of risk in clinical practice. A workshop has been held to quantify those risks,[4] and the next step would be to incorporate the findings into a tool for hand-held or personal computers. A woman's decision to take tamoxifen would still depend on the values she places on different outcomes, such as stroke or breast