Although we support the need for regulatory guidelines in India and other medical tourism destinations, we believe that such guidelines must be coupled with guidelines in source countries as part of a comprehensive and global approach. If these guidelines are developed piecemeal they risk being less effective or not implemented owing to worries that less regulated countries will develop pricing advantages.

We declare that we have no conflicts of interest.

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Why are drug trials in Alzheimer's disease failing?

You suggest several reasons why trials in Alzheimer's disease are failing (Aug 28, p 658),[1] but you do not consider an obvious one: that the hypothesis on which most Alzheimer's trials are based might not be valid.

If a scientist does several experiments on the basis of a hypothesis and they all fail, he will abandon the hypothesis. Why are we so reluctant to do this in medicine? The dominant hypothesis in the field is the amyloid hypothesis and almost all trials of potential disease-modifying drugs are based on manipulation of β amyloid. In the recently abandoned semagacestat trial,[2] some patients on the drug got worse—ie, the drug, which was designed to inhibit formation of β amyloid, seemed to speed up cognitive decline. One interpretation is that the formation of β amyloid might be the brain's protective mechanism against the disease process. But this view is regarded as pure heresy. Is that because so much research funding and such large drug development budgets are at stake?

The review by Mangialasche and colleagues[3] concluded that "the one protein, one drug, one disease hypothesis used as a basis of most Alzheimer's disease therapy studies needs to be revised." It is time that we stopped looking for a "cure" but directed our research effort to the prevention of Alzheimer's disease. This complex disease will yield not to a single drug but to multiple approaches to modify the disease process, starting in mid-life. Two examples of hopeful avenues are the treatment of hypertension in mid-life[4] and the lowering of homocysteine early in the disease process.[5]

I declare that I have no conflicts of interest.

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1 The Lancet. Why are drug trials in Alzheimer's disease failing? *Lancet* 2010; **376:** 658.
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Department of Error

Ismail-Beigi F, Craven T, Banerji MA, et al, for the ACCORD trial group. Effect of intensive treatment of hyperglycaemia on microvascular outcomes in type 2 diabetes: an analysis of the ACCORD randomised trial. Lancet *2010;* **376:** *419–30*—In this Article (Aug 7), there were some errors in figures 4 and 5. Additionally, on page 419 (Summary), the last line of the Findings should have read "Six secondary measures at study end favoured intensive therapy (p<0·05)." On page 423, paragraph 2 of column 1 should have read "For diabetes-related eye events, cataract extraction was significantly reduced (by 21%) in the intensive group compared with the standard group at study end (figure 5). Other diabetes-related eye outcomes did not differ significantly between the two groups." On page 423, paragraph 3 of the right column should have read "Analysis of secondary renal endpoints shows that the risk of development of macroalbuminuria was 31% lower with intensive therapy at transition and 28% lower at study end than with standard therapy." These corrections have been made to the online version as of Oct 29, 2010. The webappendix has also been updated as of this date.