tory — all known risk factors for rupture. The risk of rupture in our series of persons without a clinical indication for undergoing brain imaging is likely to be even lower. Therefore, we decided not to refer these persons. However, should new evidence become available in support of a different approach, we intend to adjust our protocol accordingly.

With regard to the incidental findings in a Chinese population in Taiwan, presented by Lee and colleagues, we agree with them that the lower prevalence of aneurysms and asymptomatic infarcts in their population as compared with ours might be explained by a combination of factors — most notably, the younger mean age of their study participants, but possibly also the population's different ethnic composition and differences in the scanning protocol. We strongly support the performance of similar studies of prevalence rates of incidental brain findings, to further augment knowledge in this area.

Finucane raises an interesting question about the left–right hemispheric distribution of asymptomatic strokes, since it has been hypothesized that these strokes may predominantly be right-sided.[4] Among the 145 persons with asymptomatic stroke in our population, 35 had only cerebellar infarcts (30) or brain-stem infarcts (5). There were 19 persons who had bilateral hemispheric infarcts. Among the 91 persons who had unilateral asymptomatic stroke, 39 (43%) had right-sided lesions, and 52 (57%) had left-sided lesions. This difference was not significant and furthermore does not seem to support the aforementioned hypothesis.

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Visualizing Out-of-Body Experience in the Brain

TO THE EDITOR: The single-subject study design used by De Ridder et al. (Nov. 1 issue)[1] makes it difficult to conclude whether the changes seen on positron-emission tomography (PET) were due to out-of-body experiences or simply to the differential effects of stimulation at 3.7 V in 40-Hz burst mode as compared with other modes, a confounder that has not been controlled for. A more robust approach would be to compare this patient with a group of patients with tinnitus, but without the out-of-body experiences, receiving the same stimulation. Furthermore, the short duration of the out-of-body experiences in this patient (average duration, 17 seconds, starting within 1 second after stimulation) means that the experiences had almost disappeared by the time the scans started (10 seconds after stimulation started). Therefore, it is possible that most of the PET changes reported in this study, despite being consistent with the authors' hypothesis, were due to the effects of stimulation alone.

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1. De Ridder D, Van Laere K, Dupont P, et al. Visualizing out-of-body experience in the brain. N Engl J Med 2007;357:1829-33.

TO THE EDITOR: The report by De Ridder and colleagues describing a sense of disembodiment elicited by temporoparietal-junction stimulation in a patient with tinnitus extends similar findings in patients with epilepsy.[1] We should be cautious, however, about drawing analogies between an induced sense of disembodiment and spontaneous out-of-body experiences. That they have similar neuroanatomical loci is a plausible hypothesis but an untested one.

The sense of disembodiment induced by electrical stimulation is limited to a fixed location; those in whom this experience is induced by stimulation perceive the environment from the

visual perspective of the physical body, and they perceive the event as illusory. Spontaneous out-of-body experiences often involve accurate perception of the environment (including the physical body) from an extracorporeal visual perspective; the disembodied center of consciousness may seem to move about independently of the physical body, and those who have such a spontaneous experience usually perceive the event as profoundly real.[2,3] Given the differences in phenomenology and in psychological aftereffects for those who have the experience, it is premature to assume that the mechanism of an induced sense of disembodiment also applies to spontaneous experiences.[4]

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THE AUTHORS REPLY: In response to Tai's comments, comparison of the patient with a group of patients receiving the same stimulation without out-of-body experiences is practically impossible because of the interindividual variability of the anatomy of this area, both topographically (1.5 to 2.0 cm)[1] and morphologically, which precludes delivery of an identical stimulus[2] at the exact same functional area and thus comparable data.

Regarding PET signal interpretation, we argue that it is unlikely that the PET changes reported simply reflect the effects of stimulation alone and not the out-of-body experience. Stimulation started about 35 seconds after the start of the tracer injection (10 seconds before the start of the scan) and lasted until the end of the scan. The start of the scan was initiated by the sharp increase in the intracranial radioactivity count rate on the acquisition monitor, thereby capturing the full out-of-body experience. Nevertheless, we agree that even with this optimal acquisition, the out-of-body experience was reported by the subject only in the first 15 to 20 seconds after stimulation; thus, only a portion of the PET signal reflected the perfusion changes during the out-of-body experience. On the other hand, this also means that the peak maximum of the temporoparietal cluster was strongly underestimated because of temporal averaging out.

We fully agree with Greyson et al. that one should be cautious about drawing analogies between an induced sense of disembodiment and spontaneous out-of-body experiences. However, because of the unpredictable and infrequent occurrence of spontaneous out-of-body experiences, it seems impossible to image them functionally with current technology in a scientific way. Our opinion is that it is highly likely that both induced and spontaneous experiences of disembodiment have common neuroanatomical circuits.

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Learning from Failure in Health Care Reform

TO THE EDITOR: In Oberlander's Perspective article on failure in health care reform (Oct. 25 issue),[1] the author's assessment of the current prospects for major reform is too bleak. Many changes since the failure of the Clinton plan make health care reform much more likely. Most important