 gingival infection, and our observation of low risk of bacteremia applies only to a similar population. We think that there would be consensus that patients with overt oral infection and high-risk valve lesions should receive chemoprophylaxis before transesophageal echocardiography.

We would like to draw the authors' attention to other published studies on this subject with results similar to ours, that is, the low risk of bacteremia with transesophageal echocardiography.3-5 The authors also need to look at the isolated organism from the study by Gorge et al,6 because the organisms that were isolated would not have been susceptible to the recommended antimicrobial prophylaxis regimen.

The issue of who should perform transesophageal studies, training requirements, and number of examinations one must perform to maintain competency needs to be revisited. Clearly, this low-risk procedure must not be performed by unskilled operators, and adequate training in the art of intubation is mandatory. We believe that this procedure must be performed only by physician echocardiographers who have level II or equivalent training and have learned the art of intubation under the tutelage of a skilled gastroenterologist endoscopist and that the individual performs at least 50–75 examinations per year for maintenance of competency. Similar recommendations have been suggested by the American Society of Echocardiography.7,8 This serves to underscore the point raised by Drs. Pearlman and colleagues about the skills of the physician echocardiographer performing the study. We assume that the individuals who performed the study in this case met the criteria recommended by the American College of Cardiology and the American Society of Echocardiography.

Perhaps the most important comment concerning this report is that the question is not whether viridans streptococcal infection ever occurs after transesophageal echocardiography, but rather what is the magnitude of the risk relative to the well-known risks of widespread prophylactic antibiotic use, both to the individual patient and in populations. As transesophageal echocardiography becomes increasingly common, no doubt there will be additional reports of patients who have had both transesophageal echocardiography and subsequent endocarditis, whether the association is chance or causative. The observation that one case occurred in temporal association of unknown proximity in a patient with unknown dental status after repeated and difficult intubation by a physician with unknown training and background seems insufficient to justify the risks inherent in a blanket recommendation for antibiotic prophylaxis for all patients undergoing the procedure of transesophageal echocardiography. Likewise, a case report of a patient having anaphylaxis or anaphylaxis and death following antimicrobial prophylaxis for transesophageal echocardiography would be inadequate to support a blanket condemnation of chemoprophylaxis. Ultimately, the question will need to be answered based on better quantitative estimates of the risks with and without chemoprophylaxis.

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Smoking and Acute Myocardial Infarction

Drs. Ockene1 summarize the studies that establish that quitting smoking prevents both occurrence and recurrence of myocardial infarction. They also comment that the name for “current smokers” in our series was perhaps not the best choice. Indeed, all of our patients probably stopped smoking during the index hospitalization (since smoking is not allowed), and in all likelihood, a high proportion quit smoking altogether.

Several issues were not discussed. One is that survival in our nonsmokers remained significantly lower even after the effect of their worse baseline characteristics was accounted for by multivariate analysis. The explanation that Drs. Ockene apparently favor is that, associated with the better baseline risk factor profile, survival was further enhanced when the smoking risk factor was removed. However, this factor can explain at best only part of the phenomenon, since the results of the multivariate analysis clearly show that even when this subgroup of smokers who quit smoking is matched to nonsmokers by their baseline risk factors, the in-hospital as well as 6-month mortality in the nonsmokers will still be 1.4-fold higher.

We believe that, at least in part, the better survival of the smokers is related to the fact that the pathogenesis of myocardial infarction in smokers is different, involving more thrombogenic and fewer atherosclerotic coronary artery lesions. Atherosclerosis in smokers is milder, as demonstrated by the negative correlation of degree of coronary occlusion with smoking in the 15,000 participants of the CASS study.2 There is also evidence of a higher proportion of smokers with myocardial infarction and normal coronary arteries,3 suggesting a higher rate of acute spasmatic occlusion, perhaps related to stimulation of the sympathetic nervous system by smoking.4,5 Thus, after thrombolysis (endogenous or pharmacological), smokers may be left with less severe underlying coronary artery disease, contributing to their better outcome. In summary, the reason for better prognosis in those who smoked up to the development of myocardial infarction is probably multifactorial, the most interesting aspect being the implication regarding the pathogenesis of the infarction. The results in no way refute the beneficial effect of quitting smoking after myocardial infarction.

One last comment: Drs. Ockene in their editorial comment cite our article6 and say that our smoking patients had lower levels of cholesterol. This is not exactly what we wrote. Our smoking patients had a lower prevalence of history of hypercholesterolemia as elicited by the admitting physicians who took their anamnesis.
Blood levels of cholesterol were not measured as part of the trial protocol.

References

Stability of Plasma Atrial Natriuretic Peptide
As contributors to the extensive literature on atrial natriuretic peptides (ANP),1–3 we have read with great attention the article by Nelesen et al4 entitled “Plasma atrial natriuretic peptide is unstable under most storage conditions.” Although the authors have presented a well-designed study to examine the stability of ANP, we have not observed the rates of degradation that they are reporting under similar storage conditions. In our laboratory, we routinely assay for ANP from human, dog, and rat plasma samples. Despite a concerted effort to assay samples soon after they are taken, it is often the case that samples may be stored at −80°C for periods of 1–3 months before we are able to perform an assay. We have not observed significant alterations in the values obtained when compared with the expected normal range for a sample. This is also the case for samples extracted and stored for long periods in lyophilized form, which should in theory provide an added measure of protection against degradation.

In our laboratory, the stability of ANP that we have observed is also evident in culture media samples from ANP-secreting cell types. We do not observe significant degradation of ANP in time periods of up to 3 months at −20°C, despite the fact that the media samples contain up to 15% calf serum. In addition, we routinely prepare standards for ANP radioimmunoassay in large quantities (20–30 samples for each standard) and freeze them for extended periods (up to 3 months). We have not observed significant degradation as a result of peptide loss even in standards stored at −20°C.

Given the potential magnitude of this issue and its implications, we are interested in the further views not only of the authors but also of other members of the research community who are routinely assaying for ANP.

References

Reply
We share the concerns of Flynn et al concerning the implications of our findings. In our laboratory, we previously found a puzzling, wide discrepancy in the atrial natriuretic peptide (ANP) levels obtained from renal failure patients on hemodialysis (unpublished data). These inconsistencies could not be attributed to patient conditions or to errors caused by technique or assay. The only factor that possibly could account for the discrepancy was storage degradation or breakdown products. For this reason, we decided to explicitly test for this effect in our study.1

In surveying the literature for expected values of ANP levels and ANP stability, we found that reported ANP levels in individuals other than patients with renal failure tended to be low, near the end of the linear range of the standard curve.2–4 This makes determination of significant breakdown during storage difficult to determine. We used the plasma of renal failure patients (obtained before hemodialysis) and control plasma (spiked to high levels) so that the ANP levels obtained would fall well into the linear range of the standard curve. Therefore, we reasoned that we might better be able to perceive the posited degradation of ANP during storage.

Data published by Flynn et al cover a wide range of ANP measurements. Their plasma ANP levels in volume-expanded individuals during supraventricular tachycardia and in rat plasma are at the same high levels that we worked with.7,8 Their reports, however, do not specify storage time, and it is at least a possibility that their actual levels were even higher, given the phenomenon that we have observed. The fact that they “have not observed significant alterations in values obtained when compared with the expected normal range for a sample” does not negate our findings. The best way of observing is to explicitly test the hypothesis, as we did. Unless and until other investigators specifically examine degradation of ANP, our findings remain troubling to the field.

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