Also, Weiss and coworkers dismiss the observed association between the estimated dietary intake of iron and AMI risk, which could not possibly have been caused by covariation with inflammatory states, unless iron intake is affected by inflammatory diseases. There was no evidence of that in our data. We think that our findings warrant further studies concerning the role of both dietary iron intake and serum ferritin concentration as risk factors for CHD. When there is a more comprehensive picture about both the strength and the impact of these associations in other populations, recommendations concerning the dietary intake of iron will have to be discussed.

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**Infective Endocarditis After Transesophageal Echocardiography**

Transesophageal echocardiography (TOE) generally is indicated in patients with significant valvular lesions, and therefore the question of antimicrobial prophylaxis for prevention of infective endocarditis associated with this procedure is an important one. In their article in *Circulation*, Steckelberg et al. suggested that many patients may not require prophylaxis. They described their study in which 30 mL venous blood was cultured from 47 patients at 5, 10, and 20 minutes after the start of TOE. Significant bacteremia did not occur. Our recent experience with a patient undergoing TOE demonstrates that bacteremia and infective endocarditis can complicate this procedure and emphasizes that the decision regarding prophylaxis should be individualized to each patient, as Steckelberg et al. indicated.

The case concerned a 25-year-old man with complex congenital heart disease who presented in 1989 with gradually worsening dyspnea and cyanosis. Cardiac catheterization in 1991 showed a double-outlet right ventricle, severe infundibular pulmonic stenosis, atrial septal defect, subaortic septal defect, a normally functioning Glenn anastomosis to the right pulmonary artery (performed in 1967), and a patent Blalock shunt (performed in 1981). As part of the assessment for radical repair, because there was concern about atroventricular valve morphology and attachments, the patient had TOE in January 1992. He was distressed by the procedure and tugged out the probe. Transesophageal echocardiography did not resolve the problem. A second TOE also was unsuccessful. Antibiotic prophylaxis was not given on either occasion. Between the first and second attempted TOE, he developed episodic sweating and shivering. On examination 3 weeks later, there were splinter hemorrhages and microscopic hematuria. *Streptococcus sanguis* was isolated from two sets of blood cultures, and appropriate antibiotic therapy was started. Three weeks later, transthoracic echocardiography demonstrated a vegetation on the wall of the ventricular septal defect. To date, the patient has made a satisfactory recovery. His teeth were examined radiologically and showed no abscess.

The history and timing of this patient’s symptoms suggest a causal relation to the endoscopy. The incidence of bacteremia after various forms of instrumentation will vary with the endoscopist, the institution, the procedure, and the state of the patient’s teeth. It is possible that the encouraging data from Steckelberg et al simply reflected the skill of the operator. In another study, Gorge et al. prospectively examined 24 patients with two blood cultures obtained simultaneously 6–12 minutes after the start of the TOE; 17% had positive blood cultures (albeit with organisms insensitive to standard prophylaxis). This rate is higher than those generally reported for upper gastrointestinal fiberoptic endoscopy. We emphasize that the risk of infective endocarditis after TOE will vary with individual cases and institutions. At this stage, it is difficult to generalize about the need for antibiotic prophylaxis for TOE, and further experience is necessary. For the time being, we plan to use antibiotic prophylaxis for this procedure. The issue of whether it is helpful at all in preventing native valve disease is another matter.

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References

**Transesophageal Echocardiography and Bacterial Endocarditis**

Read and colleagues raise an important point about chemoprophylaxis before transesophageal echocardiography. However, this represents only the second case that is about to be reported in the literature showing a temporal relation between the procedure of transesophageal echocardiography and bacterial endocarditis. More details about the present case would have been helpful, such as the actual time between the first transesophageal echocardiography and the reported episode of sweating, whether the patient had fever along with the sweating, and history of previous sweating, fever, or chills. These points would have served to document more fully the temporal association of the onset of symptoms with transesophageal echocardiography. It would have been useful to have information about the gingival and periodontal status at the time of the introduction of the probe in addition to the information about the absence of dental abscess on radiography. The patients in our study did not have any overt dental, periodontal, or...
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 Read 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.
Transesophageal echocardiography and bacterial endocarditis.
B K Khandheria, J M Steckelberg, J P Anhalt, D J Ballard, J B Seward, R L Click and W R Wilson

Circulation. 1993;87:1426-1427
doi: 10.1161/01.CIR.87.4.1426.b

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