Allow me to ensure you that pharmacy curriculums do not provide background as an insurance broker, nor do pharmacists desire to be in such a position. This environment is one of the principle reasons for the current shortage of retail pharmacists. This is heightened by the standardization of the Doctor of Pharmacy degree and its replacement of the Bachelor of Science curriculums nationwide. Pharmacists today go through 6 to 7 years of education whose foundation prepares students to practice pharmaceutical care in a clinical setting. It is nothing short of degrading to be forced by Managed Care to assume responsibility for poor medical decisions justified by business cases.

So, allow me to assure you as a pharmacist that I am equally frustrated by the constraints of Managed Care on the clinician’s ability to decide what is best for the patient. However, change will only be achieved when Managed Care is forced to change. Perhaps this is an endeavor on which physicians and pharmacists should collaborate since the goal of both professions is to optimize patient care.

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REPLY

We thank J. Mikkelsen for his comments on our article (1). The authors would like to take this opportunity to discuss the effects of patient selection and points of interest in more detail when our results are compared with studies on sudden cardiac death (SCD).

We studied out-of-hospital ventricular fibrillation (VF) in the early phase of acute myocardial infarction (AMI). To compare our study with studies on SCD, two main points deserve attention. First, we focused on the early phase of AMI. In victims of SCD identification of subjects that were in the early phase of AMI is extremely challenging. Standard histological techniques underestimate the true frequency of early AMI. The articles on SCD cited by Mikkelsen confirmed that only 5 to 21% of victims were in the early phase of AMI. Presence of a fresh coronary occlusion or ruptured plaque also varied between 23 and 82%, reflecting heterogeneity of methodology or studied populations. Diagnosis of
early phase of AMI in our study was based on ST segment elevation and angiographically confirmed presence of a fresh coronary occlusion. Identical criteria were applied for the control group. Second, we specifically focused on VF and not on all fatal arrhythmias as a whole. Severe bradyarrhythmias are reported up to 30% in SCD (2). In studies on SCD the fatal arrhythmia is seldom specified. In our study VF was confirmed by rhythm recordings. So, studies on SCD are impossible to compare with our study as long as VF, early phase of AMI and coronary anatomy are not simultaneously specified.

The main finding of our study was that acute occlusion in the left coronary artery is associated with greater risk for out-of-hospital VF compared with the right coronary artery in the early phase of AMI. This finding is not the result of differential selection. We fully agree that the AMI patients in our study do not represent all patients with AMI. To reach the group of “AMI with VF,” patients had to survive VF. To explain our findings by selection bias, as suggested by Mikkelsson, one has to assume that patients with out-of-hospital VF and occlusion of left coronary artery have a higher probability of being admitted than patients with out-of-hospital VF and occlusion of right coronary artery. To the best of our knowledge, there are no data suggestive of this assumption.

The comments of Mikkelsson and our article raise another important field of interest: What is the effect of site of occlusion on life-threatening bradyarrhythmias in the early phase of AMI? Therefore, studies on SCD that document bradyarrhythmias, early phase of AMI and coronary anatomy would be very interesting.

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Underestimation of the Valvulopathy Effect of Fenfluramine

In an effort to evaluate the relationship between the use of fenfluramines as diet drugs and the prevalence of mitral and aortic valve regurgitation, Burger et al. (1) compared measurements from a study conducted for another purpose to those described by Singh (2). Burger observed that the prevalence of mitral valvulopathy in his study was comparable with the Framingham study (1.3% vs. 1.6% from Framingham) and aortic regurgitation (6.6% vs. 4.8% from the Framingham study). Burger surmised that the valvulvar regurgitation seen in his patients may not be due to fenfluramine but to age-related degenerative changes. Schiller (3), in an accompanying editorial, seconds this point of view stating (page 1161), “It would seem then that as studies have become more scientifically rigorous, the role of fen/phen in valve disease appears to be approaching the vanishing point.” However, there are two important additional observations concerning Burger’s methodology that undermine these conclusions.

There were 591 patients in Burger’s study. Of these patients, only 226 (38.2%) returned for an echocardiogram. The remaining 365 patients who were also exposed to the fenfluramines, for unknown reasons, did not undergo echocardiography. Since only three of the 226 patients who returned had mitral regurgitation, and only 15 of the 226 patients had aortic regurgitation, the fate of the remaining 365 patients is critical in a proper assessment. The absence of the echocardiograms in over 60% of the cohort makes this study especially vulnerable to selection bias.

A second concern involves Burger’s simple comparison between regurgitation prevalence in his population and Framingham. The difference in the mean ages between that of Burger’s cohort (mean age 46.9, standard deviation 8.9) and that of the Framingham population (mean age 55, standard deviation 10) suggests that a coarse comparison of the crude prevalences from these two populations is inappropriate and misleading. Fortunately a more appropriate adjustment is available through an examination of Singh’s data (4). Given both the mean age (standard deviation) and the gender distribution provided by Burger one can, assuming the normal distribution, approximate the distribution of age and gender in the Burger study. From Singh (4) the prevalence of each of mitral regurgitation and aortic regurgitation is available (Table 1).

From Table 1 one can compute the expected prevalence in the Burger cohort based on the gender and age-specific mitral and aortic valve prevalence in the Framingham study. If the 10.2% of patients whose ages are outside the 26 to 83 age range (based on a normal distribution with mean age 46.9 and standard deviation 8.9) fall in the upper age range (greater than 83) and these patients have the same prevalence of valvular regurgitation as those in the 70 to 83 age range, the computations reveal that the expected prevalence for mitral regurgitation (Food and Drug Administration criteria) is 1.0% and for aortic regurgitation is 3.3%. This conservative computation provides mitral and aortic prevalences that are less than those observed by Burger. Based on these age- and gender-adjusted prevalences, the prevalences seen by Burger et al. are greater than would be expected from degenerative changes alone.

These two observations substantially weaken the explanation provided by Burger. Since Schiller chose to build his conclusions on Burger’s results, this editorial’s foundation is now effectively removed. The data collected by Burger, while representing an incomplete assessment, support, rather than refute, the association found between fenfluramine and cardiac valvulopathy.

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