sequitur for your editorial commentary on these trials convolutedly to conclude that many patients receiving long-term ACE inhibitors should be denied the proven benefits of long-term aspirin therapy in exchange for the less clearly proven benefits of other antiplatelet agents.

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REFERENCES

REPLY
Thank you for your comments. At issue is not primarily the long-term effects of aspirin in coronary artery disease but its combination with an angiotensin-converting enzyme (ACE) inhibitor in patients with heart failure (1). Any real benefits of long-term aspirin therapy, however, have been regarded, at best, as questionable. The meta-analysis (1) on which your argument is based is characterized by important weaknesses and shortcomings which Dr. Cleland has done a good job of pointing out (2).

I am sure that no responsible physician wants to deny patients drugs of proven benefit. However, the negation of an interaction (3) based on results of the use of otherwise effective heart failure drugs which, in 90,000 patients taking the combination of an ACE inhibitor with aspirin, did conspicuously little or nothing is a benefit with which some physicians are not content. There was not even prevention of heart failure. Moreover, in the most recent meta-analysis cited (4) in your letter in which, similar to that of Latini et al. (3), the patient groups are dissimilar, there was a consistently more favorable risk reduction in patients without aspirin (0.85 vs. 0.75 and 0.76 vs. 0.68 for death and combined death, heart failure and myocardial infarction in the aspirin vs. no aspirin groups, respectively). Consequently, in consideration of the comparative yield of the combination of an ACE inhibitor and aspirin and an ACE inhibitor without aspirin (Table 1 of reference [5]), it appears, rather, that with the combination, we are denying many patients an effective treatment for heart failure.

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Papillary Muscle Hypothesis of Idiopathic Left Ventricular Tachycardia

Nogami et al. (1) recently demonstrated that diastolic (P1) and presystolic (P2) Purkinje potentials are critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. The authors posit that P1 represents the activation potential in the distal portion of the specialized Purkinje tissue and P2 represents the activation potential of the left posterior fascicle. There was no mentioning of the papillary muscle as a possible source of these potentials.

In most parts of the ventricular endocardium, Purkinje potentials and myocardial potentials are nonseparable. This is not true at the papillary muscle, where Purkinje potentials and ventricular muscle potentials are widely separated (2,3). Joyner et al. (2) reported that pacing from a Purkinje strand inserting into the apex of the papillary muscle results in apex to base Purkinje activation. The activation then excites the ventricular muscle via the Purkinje ventricular muscle junction at the base of the papillary muscle, and propagates from base of the papillary muscle to the apex of the papillary muscle. The resulting activation sequence shown in Figure 1B of that article is identical to the sequence of activation shown in Figure 2 of the study of Nogami et al. (1). The Purkinje strands (fibromuscular band or false tendon), which are often seen in dogs, are also found commonly in humans, especially among patients with idiopathic left ventricular tachycardia (4).

The safety factor of propagation from Purkinje to ventricular muscle is lower than that from the ventricular muscle to the Purkinje fibers (2,5). This asymmetrical safety factor of propagation may contribute to the occurrence of unidirectional block and reentry. The papillary muscle may serve as an anchor to reentrant
wavefronts (3), resulting in sustained ventricular tachycardia. These two mechanisms may be important in the generation and maintenance of sustained ventricular tachycardia near the papillary muscle.

Clearly separated Purkinje potentials are characteristic findings for endocardial recordings near the papillary muscle (2,3). Successful radiofrequency ablation at these sites (1) suggests that the reentrant wavefronts responsible for idiopathic left ventricular tachycardia are adjacent to or are located within the papillary muscle.

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REPLY

We are grateful for the opportunity to respond to the comments by Dr. Chen et al. concerning our recent article in the Journal (1). I was very impressed by the study of Joyner et al. (2), and was impressed by the longitudinal dissociation of trabecular muscle and Purkinje signals in isolated papillary muscle of a dog. According to Figure 1B in their article, pacing from a Purkinje strand inserting into the apex of the papillary muscle resulted in apex to base Purkinje activation, which then excited the trabecular muscle via the Purkinje-muscle junction at the base of the papillary muscle with propagation of the activation from the base to apex of the papillary muscle. Their Figure 1B is similar to our Figure 2A, as Dr. Chen et al. have suggested. According to the results of Joyner et al. (2), our diastolic potential (P1) appeared to be the result of a signal from the local ventricular muscle of the papillary muscle, and our presystolic potential (P2) that from the local Purkinje activation of the papillary muscle. However, there are several differences between our study and the study by Joyner et al. First, while the Purkinje-muscle junction in their study was located at the base of the papillary muscle, the Purkinje-muscle transmission occurred at the infero-apical septum in our study. The position of the octopolar electrode catheter shown in our Figure 1 differed from the site of the posterior papillary muscle. The activation sequences of the Purkinje potential during sinus rhythm may also differ. Because Purkinje fibers enter the anterior and posterior papillary muscles through the trabecular carneae, the activation sequence of the Purkinje potential in the papillary muscle during sinus rhythm must be from the apex to the base. However, our P2 was recorded earlier from the proximal than the distal electrodes during sinus rhythm. We think that P1 is the potential from the trabecular carneae. Trabecular carneae form ridges, bridges and small papillary muscles. Lai et al. (3) proposed a false tendon or interlacing Purkinje fiber as a link between the slow conduction tissue and left posterior fascicle. Gallagher et al. (4) and Thakur et al. (5) have also implied that the left ventricular muscular band may be the anatomic substrate for idiopathic left ventricular tachycardia. However, Lin et al. (6) have shown that the left ventricular muscular band is not the specific arrhythmogenic substrate for this tachycardia. However, trabecular carneae or small papillary muscles cannot be detected by echocardiography. Nevertheless, longitudinal dissociation of the ventricular muscle and Purkinje signals from the papillary muscle and trabecular carneae, and the presence of the Purkinje-muscle junction, are important in considering the reentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia.

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The Enigma of Primary Pulmonary Hypertension

Riley et al. (1) observed that exercise increased heart rates and decreased oxygen saturations of arterial hemoglobin abnormally in patients who had primary pulmonary hypertension. They stated that this occurs in subjects with circulatory disease or deconditioning. They did not refer to the studies showing that fast heart rates and desaturation of arterial hemoglobin also occur in well-trained athletes (2,3). They did not measure cardiac outputs in their patients to allow them to calculate stroke volumes, but they concluded that low stroke volume, resulting from pulmonary...