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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REFERENCES

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 strand. The papillary muscle results in apex to base Purkinje activation. 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