The unfortunate fact is that the “new” definition of myocardial infarction was already being widely used before our meeting in Nice, France, took place. Indeed, many hospitals and many clinicians around the world already define myocardial infarction based on an abnormal blood troponin value. It was this fact, and the resulting confusion created between hospitals and physicians who used the new sensitive and specific cardiac markers versus those who did not, that led us to organize the ESC/ACC consensus conference. Thus, the “confusion and chaos” referred to by Dr. Tunstall-Pedoe were already present when we began sending out invitations to the meeting at the European Heart House.

In conclusion, we believe that the process as well as the product that led to the ESC/ACC new definition for myocardial infarction were both fair, reasonable and represented the opinion of most of the participants at the ESC/ACC conference as well as a variety of experts who subsequently examined the manuscript and made useful comments. We look forward to further work in this area that will undoubtedly result in revisions to the currently recommended definition for myocardial infarction.

Joseph S. Alpert, MD, FACC, FESC
Department of Internal Medicine
University of Arizona
1501 North Campbell Avenue
Tucson, Arizona 85724

Kristian Thygesen, MD, FACC, FESC

REFERENCES


Benefit of Aspirin Plus Angiotensin-Converting Enzyme Inhibitor

The conclusions of your recent editorial commentary (1) on a meta-analysis (2) of the trials of angiotensin-converting enzyme (ACE) inhibitors in acute myocardial infarction (MI) go far beyond the randomized evidence and could well be mistaken. As treatments for acute MI, aspirin substantially improves survival (3) and ACE inhibitors moderately improve survival (2,4). The trials of aspirin were done at a time when ACE inhibitors were not routinely used in acute MI, and demonstrated the substantial effectiveness of aspirin. The trials of ACE inhibitors were done more recently, at a time when aspirin was already widely used in acute MI, and the meta-analysis of their results showed that the addition of ACE inhibitors produced a small but significant additional benefit: that is, that aspirin + ACE inhibitors produce slightly better survival than aspirin alone (2). It was concluded that aspirin is of value in the treatment of acute MI, and that the combination of aspirin + ACE inhibitors is slightly but significantly better than aspirin alone. A similar conclusion was suggested by meta-analyses of the trials of long-term aspirin therapy (3) and of the trials of long-term ACE inhibitor therapy (5); aspirin is of value, but the combination of aspirin + ACE inhibitors is somewhat better than aspirin alone.

In the trials of ACE inhibitors both during and after MI, there was no significant interaction between the presence of aspirin and the efficacy of ACE inhibitors (2,5), and it was a bizarre non-
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.

Colin Baigent
Rory Collins
Richard Peto
Radcliffe Infirmary
Clinical Trial Service Unit
Harkness Building
Oxford OX2 6HE
UK

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.

Donald Hall, MD, ACC
Department of Cardiology
German Heart Center
Technical University of Munich
Lazaretstrasse 36
80636 Munich
Germany
E-mail: hall@dhm.mhn.de

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 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