federal establishment.

Changes have been made in the review and accreditation process since the founding of the ACCME in 1980, which incorporated the Liaison Committee on Continuing Medical Education material relating to national and state sponsors. Bylaws were approved by the parent organizations in 1980, as were "Essentials" in 1981 and 1982, along with "Guidelines" developed by the Continuing Medical Education Advisory Committee of the American Medical Association's Council on Medical Education.

Initial applications for accreditation are evaluated by on-site visit teams, at the time of a CME offering. While Mr. Breeling is correct in his observation that some members of the Accreditation and Review Committee (ARC) may have exhibited various biases (sites, management style, categories, etc) in the past, continued refinement of the selection and indoctrination process promises to eliminate most (if not all) questions unrelated to the "Essentials". This will not preclude close questioning to assure the review committee that the applicant is in compliance with all seven essentials. Within the past year, ACCME has instituted procedures to insure that all reviewers are informed, that questions and recommendations are based on the "Essentials", and that representatives of the reviewed organizations leave the reverse site visit feeling helped rather than harassed. Further, the ACCME's certificate of accreditation no longer mentions credits, and a monitoring process of the ARC is now in place to assure that Council deliberations and decisions are based on a just, fair and equitable review process with Council oversight at each step in the accreditation pathway.

As Mr. Breeling points out, all of the materials submitted by an organization seeking accreditation or reaccreditation are available at each Council meeting; you may be assured that these materials are used and closely examined by the Council whenever necessary. The recommendations of the ARC are contained in the agenda book sent to every Council member to study before each meeting; these are presented individually by the Chairman of the ARC for discussion and vote, with abstention by members of the Council who may have a real or perceived conflict of interest in a particular case. The Monitoring Committee, established in 1987, reports to the Council on its findings of the activities of the ARC, with questions and comments from Council members before acceptance of that report.

Other measures which have been instituted in the recent past include: 1) adoption of a standard four-year term of accreditation, with up to two additional years for exceptional excellence. 2) procedures for reconsideration and appeal of adverse accreditation decisions; 3) procedures for handling complaints regarding accredited programs; 4) guidelines for commercial support of CME; and 5) guidelines for producers of enduring educational materials, including sponsors whose sole products are such materials.

As changes in the field of CME occur, they will be addressed by the ACCME and its parent bodies, as necessary.

In a few short years of its existence, having just completed the first full cycle of review of all applicants, ACCME has demonstrated its ability to function as a responsible organization, which is dedicated to abiding by its Bylaws, with particular attention to Article II—Purposes and Functions: "review periodically its role in continuing medical education to insure it remains responsive to public and professional needs." It's apparent that the process is working, not only through the ACCME and its committee structure, but through editorial by Mr. Breeling and this opportunity to respond to his comments.

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Houston

Why Myocardial Infarction?

To the Editors:

We were interested in Dr. Topol's review article on myocardial infarction (MI) where he stated that one in four patients have less than total occlusion of a coronary artery during the event. What, then, is the true cause of MI? It may not occur simply because of mechanical alterations within the coronary artery. It is not known what precipitates the event.

There is current data to support the hypothesis that an immunologic reaction may be associated with this ischemic cardiopathy of MI. There are four additional facts that support this hypothesis. Mastocytes are found in normal cardiac tissue. Increased mastocyte levels are found in the coronary artery wall in patients with coronary spasm. Mastocytes are also located in atherosclerotic plaques. Significant concentrations of histamine and other mediators are released by the myocardium after a specific IgE allergic stimulus. Another investigator has reported a four-fold greater risk of myocardial infarction in subjects with an elevated number of circulating PMN.

In laboratory animals, elevated numbers of T-cells are found in artificial MI. In human subjects with naturally-occurring MI, T-cell levels are significantly diminished compared to age-matched normal subjects. Cutaneous-type IV hypersensitivity is also reduced in MI. The explanation of T-cell changes in MI is not understood. Perhaps the diminished numbers of T-cells in severe MI represent the body's effort to diminish inflammation and this, in turn, diminishes myocardial destruction.

Just as there are immediate and late stages in the classic allergic reaction, the unusual presence of mast cells in the heart and alterations of T-cells in ischemic heart disease suggest that there may be a similar immunologic phenomenon occurring in MI.

In a sample review of 25 randomly selected patients in Florence admitted to the hospital with a diagnosis of MI, five were RAST-positive for common airborne allergens found in Italy and the US. This incidence (approximately 20 percent) is to be expected.

In summary, the presence of specific IgE and alteration of T-cells in MI does not explain the MI phenomenon. It is necessary to find another pathway to explain a possible immunologic pathogenesis of myocardial infarction.

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To the Editor:

On the basis of pathogenic studies by Davies and Thomas, the actual mechanism for myocardial infarction is likely to be plaque rupture or fissure. However, the explanation for why a plaque ruptures remains elusive, particularly because it need not be an advanced atherosclerotic lesion that severely compromises the infarct vessel lumen. In the accompanying letter, Femia et al raise the possibility that the pathogenesis may be immunologic. Although this must be considered speculative, it is possible that white cells or their products could play a role in triggering plaque rupture. The data to support this hypothesis are particularly scant. Nearly 90 percent of patients with acute myocardial infarction have total occlusion of the infarct-related artery at angiography performed less than four hours from symptom onset. The answer to, “Why myocardial infarction?” certainly is likely to be plaque dynamics, with exposure of collagen and attendant platelet aggregation and thrombus formation. Perhaps a more precise question should be, “Why plaque rupture?”

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Medical Bronchial Artery Embolization

To the Editor:

Muthuswamy et al recently reported very successful treatment of massive hemoptysis by bronchial artery embolization. This treatment method is not only invasive and technically quite difficult, but rebleeding is also relatively frequent.

Recently, I had a very interesting experience with a patient who had massive hemoptysis lasting more than a month due to bronchiectasis. The bleeding suddenly decreased the day following administration of indomethacin for his high fever. This phenomenon reminded me of the indomethacin therapy of patent ducus arteriosus in newborns.

Since then, I tried indomethacin therapy in five other massive hemoptysis patients with a 100 percent success rate. The effect seems to be dose-dependent. In my experience, a 50 mg suppository given daily for three days is completely effective. This therapy could be called medical bronchial artery embolization, with a future supporting study employing bronchial arteriography pre- and post-treatment.

The mechanism of action is probably decreased bronchial circulation caused by indomethacin-induced inhibition of cyclo-oxygenase, which accelerates the production of vasodilating prostaglandins from arachidonic acid.

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Lost in the Translation

To the Editor:

In Dr. David Cugelli’s report of the proceedings of the International Symposium on Lung Sounds (Chest 1987;92:342-45), a Spanish translation of the lung sounds nomenclature is offered. I think the initiative deserves warm congratulations considering the vast number of Spanish speaking readers of your journal. However the word “estetores” proposed to translate “crackles” may confuse the subject. At least in some of the Spanish speaking countries the term “estetores” has been traditionally applied to sounds generated in airways as opposed to “crepitos” (crackles), supposedly originated in alveoli. The term, therefore, includes wheezes (sibilancias) and ronchus (roncos).

In April, 1986, the Chilean society of Thoracic Diseases and Tuberculosis issued a statement and recommended a translation of the ATS nomenclature. For the designation of crackles we chose the word “crepitaciones” which has not been used before in this context, but is similar in meaning to “crepitos” (fine crackles) and “crujidos” (coarse crackles) used in the traditional nomenclature. Although we naturally consider this name better than “estetores”, a widespread discussion involving Spanish speaking countries is necessary before a useful universal Spanish nomenclature is proposed.

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Chilean Society of Respiratory Diseases and Tuberculosis,
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To the Editor:

I was very pleased to receive Dr. Cruz’ communication regarding breath sound terminology. It is reassuring to learn that others are interested in this subject. The letter raises several interesting issues, and I welcome the opportunity to discuss them.

Most if not all members of the Lung Sounds Association, including myself, firmly believe that there is no clear cut distinction between adventitious sounds that have been traditionally “wet” or “dry.”