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 rupture remains elusive, particularly because it need not be an advanced atherosclerotic lesion that severely compromises the infarct vessel lumen. In the accompanying letter, Femino 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?"

Eric J Topol, M.D.,
Ann Arbor, Michigan

REFERENCES

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 duc tus 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.

Reiko Tsukamoto, M.D.,
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Chigasaki Tokushukai Medical Center,
Kanagawa, Japan

REFERENCES

Lost in the Translation

To the Editor:

In Dr. David Cugellis 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 "estertores" proposed to translate "crackles" may confuse the subject. At least in some of the Spanish speaking countries the term "estertores" 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 rochus (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 "estertores", a widespread discussion involving Spanish speaking countries is necessary before a useful universal Spanish nomenclature is proposed.

Edgardo Cruz, M.D.,
Lung Sounds Committee,
Chilean Society of Respiratory Diseases and Tuberculosis,
Santiago, Chile

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."
Such distinctions, on the basis of what one hears are really made from a subconscious diagnosis that is made by observing the patients. I am confident of this because when recorded crackles are played back to an audience of trained pulmonary physicians who then listen with acoustic stethoscopes, they agree that he hear crackles, but totally disagree as to wet vs dry, alveolar vs bronchial, etc. The only terms that make any sense are those that define the sounds as either coarse, medium or fine crackles, and to specify when they occur within the respiratory cycle—early, middle or late, either inspiratory or expiratory. Furthermore, the characteristics of crackles have been carefully defined in physical terms by Murphy. An adventitious sound that qualifies as a crackle has specific physical properties—it is no longer a matter of your opinion or of mine. The same certainly cannot be said for "wet" or "dry" or "airways" as opposed to "alveoli."

The International Lung Sounds Association welcomes recommendations from reputable organizations representing the mainstream of Spanish speaking physicians. What we have published is only what was recommended to us. Achieving a consensus was very difficult because I understand neither Spanish nor Portuguese. Even the several knowledgeable pulmonologists who advised us had difficulty deciding on the proper terms. This was due in part to some differences between the preferred nomenclature in Spanish and Portuguese that is used in South America and in Europe. Incidentally, the original article in Chest contained several errors in the Table. They were largely corrected before the reprints were published.

David W Cugell, M.D.
Chicago

**Bronchial Non-adenoma**

To the Editor:

The review entitled "Bronchial Adenoma" by Rozenman and associates (Chest 1987; 92:145-47) provokes a reaction. The authors are commenting about symptoms, signs, and age and distribution, and other features of bronchial adenoma as if it were one entity. This is contrary to their own statement in the beginning of the review, that "bronchial adenomas are a heterogenous group of tumors." Indeed, this group of tumors consists of several vastly different neoplasms. They are different pathologically, physiologically and clinically.

Bronchial carcinoid accounting for 85 percent of all neoplasms in this group has a considerable malignant potential. In the experience of my colleagues and myself,1 lymphatic metastases were present in seven of 69 patients, in seven the tumor invaded extrapulmonary structures, and hematogenous dissemination occurred in four.

I have no appreciable experience with mucoepidermoid tumor, but it too has a marked malignant potential, and is called, appropriately, carcinoma. The term mucoepidermoid adenoma used by Rozenman and colleagues is incorrect. Adenoid cystic carcinoma is always malignant.1 Local invasion is invariably present, as the tumor spreads along the submucosal plane and perineural lymphatic spaces. Penetration of adjacent organs and hematogenous metastases, particularly to the lungs, are common. Adjuvant radiotherapy in addition to resection is mandatory, if recurrences (very common!) are to be avoided.

Bronchial adenoma is a misnomer applied all too often to various bronchial neoplasms implying a specific pathologic entity, while in fact these are biologically different entities. There is no pathologic relation whatever between the neoplasms under discussion. In addition, I object to the use of the term "adenoma" with reference to a tumor with any malignant potential, because it implies benignity. Adenoma, by definition, is a benign neoplasm. If, as stated by the authors "bronchial adenomas are all relatively low-grade malignant neoplasms", then they are not adenomas.

I suggest discontinuing the term "bronchial adenoma" with reference to this group of neoplasms. The term bronchial adenoma should be reserved for bronchial mucous gland adenoma, the only true adenoma of the bronchus.

**REFERENCES**


To the Editor:

Regarding the name bronchial adenoma, we agree with Dr. Weissberg that the term is actually a misnomer, as all these tumors are malignant as stated in our review.1 This name is however, still in use in the main textbooks such as Fraser and Pare.2

We commented on the heterogeneity of this tumor, although 90 percent belong to one group, the carcinoid tumor, and can therefore be discussed together in our opinion.

We regret that we omitted the valuable article of Dr. Weissberg et al from our list of references.

Judith Rozenman, M.D.
Chaim Sheba Medical Center,
Tel Hashomer, Israel

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2 Fraser RG, Pare JAP. Diagnosis of diseases of the chest, 2nd ed.

**Predicting CO Diffusing Capacity**

To the Editor:

In their recent paper, Harber and colleagues point out an important problem using the Crapo prediction equations for single breath carbon monoxide diffusing capacity.2 These provide predicted values considerably higher than other equations in wide use, with the result that "many more persons are considered to have an abnormal diffusing capacity." Harber and co-workers reported 164 of 643 subjects with an isolated diffusing abnormality using the Crapo equation, vs only 34 using Cotes.2 Indeed, "the striking disparity demonstrated [26 vs 5 percent] probably underestimates the true situation because persons with abnormal spirometry were [not analyzed for abnormal diffusion and] many more persons will be considered 'abnormal' if the Crapo equations are used."

In publishing predicted values for DCO\textsubscript{a} based on 582 subjects from a representative sample of a large population,4 we reported an increased frequency of "abnormal" results using Crapo values of the same magnitude as Harber's: 46 percent of the men were abnormal