any terms to define clinical groups that are useful to them. I have no quarrel with Bailey's proposed classification of diseases in terms of difficulty of treatment. This classification of disease reflects the resistance of members of different species, and looks reasonable and useful to me. However, it should not in any way lead to abandonment of identification of individual strains with species names in clinical reports. That information is needed for diagnosis and for epidemiologic purposes. For example, if several specimens from a patient yield more than one species, as can occur, the interpretation would be different from that of repeated isolation of a single species. The clinical microbiologist must continue to use the well established (albeit still developing) principles to classify and identify mycobacteria. The clinician can then classify disease on the basis of such useful tables as proposed by Bailey.

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To the Editor:

As stated in Dr. Bailey's article, the Runyon Group Classification which is based on the chromogenicity and rate of growth of organisms has been an "invaluable" aid in the identification of atypical mycobacteria, a point with which we fully agree.

Our intent was not to negate the work and importance of the contributions of the "microbiologists who have labored diligently and effectively to bring order to the genus mycobacteria," and certainly it was not our wish to be placed in the unenviable position of causing one of the giants of mycobacterial taxonomy, depression.

Instead, we were pointing out that the current system of classification of mycobacteria is a complex one which has "limited relevance, especially to the clinician caring for patients." Without question, some form of mycobacterial taxonomy is needed to clearly identify these organisms for laboratory as well as epidemiologic purposes, as Dr. Wayne points out.

In a similar fashion, we support Dr. Bailey's classification as being a clear and concise method of clinically classifying mycobacterial infections according to their anticipated response to therapy. This approach, in our opinion, should enhance the management of these unusual infections that increasingly is being provided by nonspecialists in disease caused by these organisms.

For clinicians, there is a need to develop a clear, generally accepted nomenclature for disease caused by mycobacteria other than tuberculosis. Whether it is the responsibility of the "scientist" to aid in this endeavor is, in Dr. Wayne's opinion, an unequivocal no. We don't agree!

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Mitrail Anulus Calcification in Patients with Chronic Renal Failure

To the Editor:

The excellent paper by Forman et al. (Chest 1984; 85:367) called attention to the prevalence of mitral annulus calcification (MAC) in patients with chronic renal failure (CRF) on long-term hemodialysis. Certain aspects of MAC in such patients are worth emphasizing. Whereas MAC commonly affects the posterior mitral anular region, extensive calcification of the anterior and posterior mitral leaflets themselves can also occur. This type of mitral involvement, though rare, has a distinctive anatomic morphology and is associated with unusual and striking echocardiographic appearance.

Unlike MAC of the well-known senile degenerative type, MAC of CRF patients presents as highly characteristic rounded or nodular excrescences and may be associated with calcification in mitral chordae tendineae, papillary muscles and even trabecular areas. Thus, MAC in CRF patients differs from the senile type not only in its occurrence in the third to sixth decades, but also in its morphologic details.

Soft tissue calcification, especially in arteries and around joints, is common in CRF patients with extensive MAC. Parathyroid hormone levels tend to be higher and the duration of hemodialysis longer in CRF patients with MAC, than in CRF patients without MAC, on echocardiography.

The literature on these calcific and other noncalcific mitral valve changes in CRF patients is very scant, so that most physicians and even many cardiologists are not aware of them. Yet, increasing numbers of CRF patients are now surviving for longer periods of time; we are therefore likely to encounter such cardiac complications with increasing frequency.

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To the Editor:

Thank you for calling attention to important aspects of our paper. Since patients with chronic renal failure are at increased risk for the development of mitral anular calcification (MAC), we are in agreement that MAC and chronic renal failure are worthy of emphasis since more and more of these patients are coming to attention because of increased longevity. MAC is associated with soft tissue calcification especially of the lungs, stomach, and joints. Pathogenesis of MAC in chronic renal failure appears to be related to abnormal calcium phosphorus homeostasis in the setting of secondary hyperparathyroidism. Parathyroid hormone levels were not measured in our study, but other workers have found high levels in patients with chronic renal failure who developed mitral anular calcification. Indirect evidence that parathormone contributed to MAC in our study was suggested by decreased incidence of MAC in patients who had undergone parathyroidectomy.

However, we do not agree that senile MAC on morphologic grounds can be separated from MAC of chronic renal failure. Calcific deposits are usually restricted to the mitral anulus or areas just below the mitral valve usually involving first the posterior mitral leaflet. Calcification of the anterior mitral leaflet and the aortic valve may also occur. However, calcification of the mitral chordae tendineae, papillary muscles, and trabeculae are rare in both chronic renal
failure and senile mitral annular calcification. Calcification in these areas is strongly suggestive of chronic rheumatic mitral valvulitis. We recently reported pathologic findings in 28 patients who died of chronic renal failure. Eight of these patients had MAC and only one patient had calcific deposits involving the anterior mitral valve leaflets, chordae tendineae and papillary muscle. No patient had calcification of the trabeculae except in the areas adjacent to the mitral valve attachments. Dr. Virmani has examined over 30 patients with senile mitral annular calcification and has not observed any differences as to the character of calcific deposits or their location (personal observation). Dr. Virmani has also published pathologic findings in 543 autopsy patients with rheumatic heart disease and found that the mitral valve leaflets are fibroed and often calcified with extension into chordae tendineae and papillary muscles. Therefore, we feel that both senile MAC and mitral calcification secondary to chronic renal failure are indistinguishable morphologically whereas rheumatic valvular disease is quite different.

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Candida tropicalis Endocarditis in Idiopathic Hypertrophic Subaortic Stenosis

To the Editor:

Infective bacterial endocarditis is a recognized complication of idiopathic hypertrophic subaortic stenosis (IHSS). Herein we report the first known case of fungal endocarditis complicating IHSS.

CASE REPORT

A 52-year-old man with IHSS has severe outflow obstruction. He was admitted for investigation of a febrile illness of one month duration for which he had received a three-week course of multiple antibiotics. There was no history of intravenous drug abuse. Six blood cultures grew Candida tropicalis.

Within a few days following admission, the patient developed progressive congestive heart failure with bouts of pulmonary edema. Echocardiogram revealed a large vegetation attached to the atrial surface of the anterior mitral leaflet (AML) which appeared flail. Twenty-five mg of amphotericin B was given intravenously followed by emergency mitral valve replacement. At operation, a large exophytic mass occupied the left atrium which was attached to the AML by a short stalk. In close proximity to the base of this mass two chordae tendineae were ruptured (Fig 1). Microscopically, at the periphery of the vegetation, mycelial and yeast-like forms were present. A total dose of 3.0 g of amphotericin B was subsequently administered intravenously over a two-month period. The patient, however, succumbed to Pseudomonas septicaemia.

Candida endocarditis is a known complication in patients receiving prolonged antibiotic therapy. Recent reports emphasize the importance of early valve replacement in combination with antifungal agents in patients with fungal endocarditis. In the present case, the consistent tag on the AML by the pedunculated vegetation (Fig 1) was probably directly responsible for the chordal rupture and consequent flail leaflet detected by echocardiography and subsequently confirmed at surgery.

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