Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

The Serum-Effusion Albumin Gradient in the Evaluation of Pleural Effusions

To the Editor:

In the September 1990 issue of Chest, Roth et al.3 proposed that a serum-effusion albumin gradient (serum albumin minus effusion albumin) of 1.2 g/dl was effective in differentiating between exudate and transudate. They found 100 percent sensitivity and 72 percent specificity by using the Light criteria, and 95 percent sensitivity and 100 percent specificity by using the serum-effusion albumin gradient. They proposed that the serum-effusion albumin gradient was superior to the Light criteria in specificity.

We also compared those two methods in 101 patients with pleural effusions. They were followed up at Ataturk Chest Diseases Hospital between November 1990 and May 1991. Of these patients with effusions, 74 had exudates and 27 had transudates. The diagnoses in the patients with exudates were malignant neoplasm (n = 23), tuberculosis (n = 37), parapneumonic effusion (n = 11), pulmonary embolism (n = 2), and Behçet's disease (n = 1). The diagnoses in the patients with transudates were heart failure (n = 24) and renal diseases (n = 3). The sensitivity and specificity of the tests were 100 percent and 81 percent, respectively, when the Light criteria were used, compared with 91.9 percent and 100 percent when the serum-effusion albumin gradient was used (Table 1).

In our study we found that a gradient of 1.4 g/dl was effective in differentiating between exudate and transudate. Pleural effusions with a serum-effusion albumin gradient greater than 1.4 g/dl were regarded as transudative. There was no statistically significant difference between these two methods.

When the Light criteria were used, pleural effusions were misclassified as exudate in five patients, four with congestive heart failure and one with renal failure. The serum-effusion albumin gradient was most accurate when values less than 1.4 g/dl were accepted as identifying exudate. In six patients (three with mesothelioma, one with metastatic adenocarcinoma, and two with tuberculosis), pleural effusion was misclassified as transudate when the serum-effusion albumin gradient was used. Many investigators believe that although the sensitivity of the Light criteria is high in identifying exudative effusions, it is not as sensitive in evaluation of pleural effusions secondary to congestive heart failure.4,5

We conclude that the serum-effusion albumin gradient can be used as a helpful method in patients with chronic transudative pleural effusions.

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REFERENCES
1 Roth BJ, O'Meara TF, Cragun WH. The serum-effusion albumin gradient in the evaluation of pleural effusions. Chest 1990; 98:546-49

To the Editor:

We enjoyed reviewing the data presented by Dr. Akkurt and his colleagues, as it confirms the efficacy of the serum-effusion albumin gradient in differentiating exudative from transudative pleural effusions. A number of important points should be made in reviewing both their data and ours.

First, Light's criteria are very sensitive for the identification of exudative pleural effusions (100 percent sensitive in the combined 160 patients). In our experience, we have seen only two patients with diagnostic criteria for an exudative pleural effusion in whom the effusions were classified as transudative by Light's criteria (both had malignant cells in the fluid with coexistent congestive heart failure).

Second, the actual cutoff to separate transudates from exudates when using the albumin gradient is debatable. Assuming that Akkurt et al meant to classify those effusions with a gradient equal to 1.4 as transudative, then applying their cutoff to our data yields 98 percent sensitivity and 83 percent specificity for identifying exudates. In any test that has a range of values for "disease" and "no disease," those values tend to fall in two overlapping bell-shaped curves. The sensitivity can be improved at the expense of specificity, or vice versa, by adjusting the cutoff point. Values close to the cutoff point are always going to be questionable.

Why, then, did their data show a relatively low sensitivity for the albumin gradient when increasing the cutoff should have improved

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<th>Table 1—Results in 101 Patients According to Serum-Effusion Albumin Gradient and Light Criteria*</th>
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<tr>
<td>Patients With Exudates (n = 74)</td>
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<tr>
<td>S-E albumin gradient, g/dl</td>
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*Values are expressed as mean ± SD. Differences between groups were significant at the level of p < 0.0001. S = serum; E = effusion; LDH = lactate dehydrogenase.
the sensitivity? The two patients in our series with diagnostic criteria for exudates yet high albumin gradients had coexistent criteria for a transudative etiology (collapsed lung and congestive heart failure). We wonder if any of their misclassified exudates could have also had a transudative etiology. The three patients with mesothelioma could have had trapped lung leading to transudative effusions. Also, their patients included a number with tuberculosis (two of whom were misclassified as having transudates), which we did not see in our group of patients.

The major criticism of both our sets of data is that the diagnoses are based on clinical criteria. This problem would best be solved by examining long-term outcome data on patients classified as having transudative effusions to rule out the possibility of a clinically silent exudative cause that was missed. More work should also be done to elucidate the pathophysiology of pleural effusions, especially those with potentially more than one cause.

How then, can the serum-effusion albumin gradient be used in the evaluation of pleural effusions? We feel it can be helpful in two circumstances. First, in a patient with clinical congestive heart failure, an effusion with Light's criteria suggesting an exudate, negative cytology, and a high albumin gradient, a period of observation with further diuretic therapy is warranted. Second, in a patient with a known malignant effusion but a high albumin gradient, a coexistent transudative cause for the effusion is suggested, and perhaps a trial of diuretic therapy for palliation is indicated.

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The opinions and assertions contained herein are the private views of the authors and do not necessarily reflect the views of the Department of Defense.

Churg-Strauss Angitis

To the Editor:

We read with interest in the November 1991 issue of CHEST the article by Guillevin et al.¹ in which the authors forcefully suggest a possible etiologic role for inhaled actinomycetes in systemic vasculitis with respiratory manifestations.

In our case, the diagnosis of Churg-Strauss angitis (CSA) was supported by pathologic specimens from lung biopsy showing vasculitis of medium-sized vessels associated with infiltration of lymphocytes and plasmocytes in the absence of fibrinoid necrosis. However, this conclusion seems questionable, as the classical histologic criteria for CSA (which incidentally are thought to be nonspecific) include the finding of vasculitis of small vessels and necrotizing extravascular granulomas, usually with eosinophilic infiltrates.² Moreover, the patient in our case cannot be said to have CSA on clinical grounds, since according to the traditional format classification of the American College of Rheumatology³ at least four of six criteria have to be fulfilled for a clinical diagnosis of CSA to be made. Therefore, the observation in our case of only three criteria, namely, asthma, neuropathy, and pulmonary infiltrates (with the notable absence of eosinophilia of more than 10 percent, paranasal sinus abnormality, and extravascular eosinophilia), makes the suggested diagnosis untenable.

It is important to recall that, unlike American authors, Guillevin and other French investigators do not consider CSA and polyarteritis nodosa as two separate clinical entities and propose the application of common diagnostic criteria.⁴ Recognizing that this patient fulfilled the "French criteria" for polyarteritis nodosa, it is unclear why, accordingly, a diagnosis of polyarteritis nodosa was not established, instead of the less consistent conclusion of CSA.

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REFERENCES


To the Editor:

We agree with Riccio and coworkers that the case we reported¹ should be attributed to polyarteritis nodosa (PN), not CSA. Our case satisfied only three of the classification criteria established by the American College of Rheumatology (ACR),² despite the fact that the clinical history and symptoms obviously adhered more closely to CSA than PN. We would like to mention that the ACR criteria for CSA have a sensitivity of 85 percent and a specificity of 99.7 percent and that some patients do not fit with the published criteria. In our opinion, it is often difficult to sharply separate CSA (now called Churg-Strauss syndrome [CSS]) from PN. The Faucci classification³ was helpful in identifying three subsets in the PN group: (1) PN, (2) CSA/CSS, and (3) overlap syndrome. In CSS the pathologic manifestations vary somewhat, with only some of the patients presenting a granuloma, which is conversely present in a broad spectrum of vasculitides, including typical Küstnmaul-Maier disease. For all these reasons, we are not so peremptory in separating PN and CSS patients, as the line of demarcation is not clear-cut.

On the basis of our experience with large groups of patients,⁴ we think that CSS and PN belong to the same group, expressing some common symptoms (eg, fever, neuropathy) and others that are more specific to each entity (eg, asthma in CSS, presence of hepatitis B virus markers and orchitis in PN). Hyperesoinophilia is usually present in CSS but can also be present in PN without symptoms of allergic or respiratory signs. Differences between CSS and classic PN could be due to the etiologic factors observed in each disease (eg, inhaled antigens responsible for asthma and sinusitis in CSS as opposed to a nonrespiratory mode of entry for antigens in classic PN). In PN, hepatitis B virus has been considered responsible for 36 percent of cases,⁵ and the characteristics of the disease exclude pulmonary symptoms. Laboratory data also show that in patients with CSS and microscopic PN,⁶ anti-neutrophil cytoplasmic antigens (ANCAs) are often, but not exclusively, present and that in classic PN, ANCAs are rare.

For all these reasons we believe that, without denying the great