wished to solve the complicated problems of clinical allergy. I sincerely hope that American investigators and clinicians will research thoroughly the important role of the parasympathetic system in the study and the treatment of allergic diseases.

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Desquamative Interstitial Pneumonitis vs Usual Interstitial Pneumonitis

To the Editor:

My colleague and collaborator, the late Edgar G. Harrison, Jr., M.D., was of the opinion that “desquamation” was a fundamental phenomenon in the spectrum of usual interstitial pneumonitis. In our report on classic interstitial pneumonitis-fibrosis, we found that almost two-thirds of our biopsies revealed this finding. Harrison believed that the designation, “desquamative interstitial pneumonitis,” should be reserved for those instances where the alveolar cells were rounded, uniform, and monotonous in appearance and were associated with no or a very slight interstitial reaction. This concept adheres to the original description by Liebow and associates.

In the report by Tubbs and colleagues, their criteria for desquamative interstitial pneumonitis are at great variance with those of Liebow et al and of Harrison, and the changes described by Tubbs et al are more in keeping with usual interstitial pneumonitis. Hence, I believe that the conclusions of Tubbs et al about desquamative interstitial pneumonitis and usual interstitial pneumonitis being phases of the same disease are not justified. The photomicrographs appearing in the report of Bedrossian and colleagues are more in keeping with the appearance of desquamative interstitial pneumonitis.

Does all this nit-picking really make any difference? Only in the sense that if terms like desquamative interstitial pneumonitis and usual interstitial pneumonitis are used, care should be taken to follow the criteria as originally proposed. The finding of Bedrossian et al of localized desquamative interstitial pneumonitis suggests that histopathologic findings might not in this instance be a unifying element in describing a pathologic entity. I am of a similar persuasion, and for that reason the concept of classic interstitial pneumonitis-fibrosis was set forth; that is, the pathologic findings make sense only in the context of a consistent clinical and laboratory setting. Until someone discovers specific etiologic agents or pathogenic mechanisms behind the changes designated as desquamative interstitial pneumonitis and usual interstitial pneumonitis, it must be so.

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REFERENCES


To the Editor:

We have carefully reviewed DeRemee’s communication and the original publication of DeRemee et al. We are in complete agreement that adherence to the original criteria proposed by Liebow et al for desquamative interstitial pneumonia is necessary. The principal objections raised by DeRemee appear to relate to a misunderstanding regarding our light-microscopic criteria for desquamative interstitial pneumonia. These criteria closely parallel those promulgated by Liebow et al, including absent or mild interstitial inflammation, as well as absence of necrosis and fibrin hyaline membranes. A careful review of the individual cases of Liebow et al reveals that some specimens demonstrated significant interstitial inflammation, and in the postmortem material studied, significant interstitial fibrosis was present.

We excluded patients from our series who did not fulfill our absolute light-microscopic criteria. These included specimens demonstrating a necrotizing interstitial process associated with fibrin hyaline membranes and one patient with PAS-negative free alveolar cells. Furthermore, neither asbestos bodies nor birefringent dust material was identified. The only major criterion at variance with the original description by Liebow et al was, as stated in our report, a lack of uniform desquamation in the large specimens from open biopsy; however, a careful review of the original description by Liebow et al reveals that consolidation varied from 10 to 98 percent of the distal air space, and in four of their 18 specimens, consolidation involved less than 50 percent of the pulmonary parenchyma available for evaluation. Our observation of lymphoid collections and occasional germinal centers is also in concert with the original description by Liebow et al.

Since the majority of our knowledge relative to desquamative interstitial pneumonia and usual fibrosing interstitial pneumonitis is based upon retrospective studies, the value in distinguishing between a cicatrized and cellular phase of desquamative interstitial pneumonia remains to be elucidated. Most of these studies suggest that a cellular (as opposed to a cicatrized) process implies a more favorable prognosis on a short-term basis. Conversely, the cicatrized phase portends a less favorable prognosis, although individual patients in this category may benefit from therapy with steroids. Until a well-
controlled prospective study is done with close follow-up and standardized therapy with steroids, the relative therapeutic benefit in each group is not certain. What cannot be ignored is the relative duration of symptoms prior to biopsy, the progression of very typical cellular desquamative interstitial pneumonia to cicatrizied desquamative interstitial pneumonia, and the strikingly similar morphologic appearance, excluding fibrosis, in the two phases (Fig 1 and 2).

In Rosan’s4 epigrammatic comments as umpire in the eternal contest between “lumpers” and “splitters,” he appears to have also overlooked the importance of multifocal exfoliation in the original material of Liebow et al.2 We are in agreement that one must see beyond the morphologic appearance to a disease process which provides us with a superb model for the study of cell-mediated immune mechanisms. The migrating macrophage, modified by poorly characterized inhibiting and stimulating agents (such as migration-inhibiting factor, chemotactic factor, or other lymphokines), should be the focus of these future investigations.

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REFERENCES

To the Editor:

DeRemee’s comments pose a very interesting question. What is a new disease? Is it one with a consistent clinical and laboratory setting? Is it one with a distinctive morphologic pattern detectable by histopathologic studies? Or is it a combination of the two?

Desquamative interstitial pneumonia illustrates very vividly the difficulties in answering these questions. The original description relied heavily on the clinical response to therapy with steroids and the characteristic histopathologic pattern. Under ancillary and laboratory findings the radiographic picture of bilateral was emphasized.

Of these three criteria the histologic pattern remains as the only distinctive feature of desquamative interstitial pneumonia because of the innumerable conditions that respond to therapy with steroids and are present as bilateral pulmonary infiltrates on the chest x-ray film. The problems soon started when “biopsy-proven,” histopathologically classic cases of desquamative interstitial pneumonia deviated from the other two criteria, either by not responding to therapy with steroids or by occurring in a localized fashion. Subsequently, numerous conditions associated with the histopathologic pattern of desquamative interstitial pneumonia appeared in the literature, as cited in our report.1

As a result, the acceptance of desquamative interstitial pneumonia as a separate new disease became controversial. It seems advisable, therefore, that until the etiology of “classic” desquamative interstitial pneumonia is elucidated, the diagnosis of desquamative interstitial pneumonia as a clinicopathologic entity should be approached with caution after ruling out other underlying conditions. As a pattern of reaction, the description by Liebow et al1 of desquamative interstitial pneumonia

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**Figure 1:** Cellular phase of desquamative interstitial pneumonia (hematoxylin-eosin, x 104).

**Figure 2:** Cicatrizied phase of desquamative interstitial pneumonia (hematoxylin-eosin, x 104).