arteriovenous oxygen content difference \((C[a - v]O_2)\) equals 3; and the fractional concentration of oxygen in the inspired gas \((FIO_2)\) equals 0.40. From the oxyhemoglobin dissociation curve, the arterial oxygen saturation equals 0.90. Since \(C(a - v)O_2\) equals 3, the venous oxygen saturation \((SVO_2)\) equals 0.75 approximately. Using these figures, the \(Qs/Qt\) equals 0.502. The predicted value from the nomogram is a \(Qs/Qt\) of 0.34.

**Example 2**

The hemoglobin level equals 11 gm/100 ml, the \(PaO_2\) equals 50 mm Hg, the \(C(a - v)O_2\) equals 3, and the \(FiO_2\) equals 0.40. From the oxyhemoglobin dissociation curve, the \(SaO_2\) equals 0.83. The \(SVO_2\) equals 0.68 approximately for a \(C(a - v)O_2\) of 3. Again, using these figures, the \(Qs/Qt\) equals 0.604. The predicted value from the nomogram is a \(Qs/Qt\) of 0.48.

The nomogram would appear to be predicting significantly lower values for \(Qs/Qt\) than the calculated values. I would appreciate a comment from Shapiro and Peters explaining this discrepancy. If the discrepancy is real, the nomogram might still be useful for two purposes. One would be in following relative changes in \(Qs/Qt\) for a given patient, and the other would be in predicting the effect on \(PaO_2\) of changing \(FiO_2\).

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To the Editor:

When located on the nomogram, values for shunt fraction \(Qs/Qt\) are obtainable to within \(\pm 0.005\) of calculated values. This can be confirmed by comparison of the results from the nomogram with values found in published tables or on the less comprehensive charts or graphs for \(Qs/Qt\). Another way of checking this is as follows: the shunt equation can be expressed as a ratio composed of the alveolar-arterial oxygen content difference \((C[A-a]O_2)\) in the numerator and the sum of the \(C(A-a)O_2\) plus the arteriovenous oxygen content difference \((C[a - v]O_2)\) in the denominator. For the first example, the \(C(A-a)O_2\) is found by calculation to be 1.55. Since the \(C(a - v)O_2\) equals 3, the \(Qs/Qt\) equals 1.55/(1.55 + 3) or 0.34. In fact, the nomogram can be used to determine the \(C(A-a)O_2\) directly, without the need for calculation; under the assumptions stated in our article, the intersection of a horizontal line with the axes on the extreme left and right of the nomogram gives the \(C(A-a)O_2\) for any combination of hemoglobin level, fractional concentration of oxygen in the inspired gas, and arterial oxygen pressure \((PaO_2)\).

The apparently separate terms such as the \(PaO_2\), the oxygen pressure of mixed venous blood \((PvO_2)\), the venous oxygen saturation \((SVO_2)\), and the \(C(a - v)O_2\) that are found in expanded forms of the shunt equation are highly interdependent. Skold's specification of values for \(PvO_2\) and \(SVO_2\) which, given the \(C(a - v)O_2\), are not essential for calculating \(Qs/Qt\) suggests that such terms may have been used in ways that were inconsistent with the stated \(C(a - v)O_2\) of 3. For instance, in example 1, to be consistent with a numerator of 1.55, a \(Qs/Qt\) of 0.502 would also imply a \(C(a - v)O_2\) of approximately 1.55, compared with the value of 3 specified in the example.

The discrepancy raised by Skold appears to be artifactual, so that the nomogram can be used in following the absolute as well as relative changes in \(Qs/Qt\) of an individual patient.

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REFERENCE


Comment on Book Review

To the Editor:

Graham O. Solley, M.D., reviewed my book, Pathogenesis and Therapy of Bronchial Asthma with Special Reference to Organ Vagotonia, in the June 1977 issue of Chest (71:28, 1977). I feel that he has wrongly interpreted my book. I never do charge that American research on allergic diseases studied by immunologic angles is misdirected. Indeed, because I highly esteem the great advances of American research, I did emphasize that this has overlooked the important role of the parasympathetic system, notably organ vagotonia, in the occurrence of the ailments.

Indeed, I recognize that it is difficult as yet to demonstrate this vagotonia in all organs, but so far as the bronchial walls are concerned, it is easy to demonstrate by the inhalation method and the carotid-sinus-dynamometer that bronchial hypersensitivity is closely related to hyperexcitability of the bronchial vagus nerve, clearly revealing a state of pulmonary vagotonia. The bronchial walls are hypersensitive to parasympathomimetics, histamine, serotonin, and bradypin, both in asthmatic subjects and in the sensitive line of guinea pigs we obtained by pedigree breeding. This hypersensitivity is, therefore, nonspecific, but its background is nothing but the hyperexcitability of that nerve. As long as this hyperexcitability is not acquired by antigenic sensitization, it is natural to regard the bronchial hypersensitivity as being inborn, although it might be heightened by antigens; however, the problem of the bronchial hypersensitivity cannot be solved completely by the study of the bronchial walls only, because the bronchial hypersensitivity is much influenced by the sensitivity of other organs through the mutual relationship between pulmonary vagotonia and other organ vagotonia.

The results we obtained by examining asthmatic subjects and those with other allergic or related diseases may seem incredible, yet the success of our treatment over the years is the warrant for writing the book. Organ vagotonia is an important step for those who...
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 when 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.1

In the report by Tubbs and colleagues,2 their criteria for desquamative interstitial pneumonitis are at great variance with those of Liebow et al2 and of Harrison, and the changes described by Tubbs et al3 are more in keeping with usual interstitial pneumonitis. Hence, I believe that the conclusions of Tubbs et al3 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 colleagues4 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 al4 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.1 We are in complete agreement that adherence to the original criteria proposed by Liebow et al2 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,2 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 al2 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.3 The only major criterion at variance with the original description by Liebow et al2 was, as stated in our report,4 a lack of uniform desquamation in the large specimens from open biopsy; however, a careful review of the original description by Liebow et al2 reveals that consolidation varied from 10 to 98 percent of the distal air space, and in four of their2 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.2

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-

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