Pleurectomy for Recurrent Pneumothorax

To the Editor:

Recurrent pneumothorax remains a problem for the clinician. The ideal type of management is not absolute, and inevitably the chest physician takes a much less aggressive approach than does the surgeon. Until there is close cooperation between the physician and the surgeon so that a randomized controlled trial can be organized, the optimum regimen of management may never be realized.

Viewing the problem from the surgeons’ standpoint, which may well be skewed, many patients undergo prolonged intubation on medical wards. Physicians, especially in the United Kingdom, seem reluctant to accept the remarkable safety and success of pleurectomy as the definitive treatment of this usually benign but most distressing condition.

By briefly presenting our results collected over a five-year period, we hope to add weight to the already published work1-3 emphasizing that this surgical procedure is a very safe and and satisfactory form of treatment, and that it should be considered much earlier in the overall program of management.

CLINICAL MATERIAL

Between March, 1978 to June, 1983, 150 patients were referred to us for more extensive treatment of their persistent or recurrent spontaneous pneumothoraces. Only 36 were women, and the lesion predominated on the right side in the ratio of 2:1. Twenty-seven patients had previous pneumothoraces on the same side, and 74 patients were transferred to us because tube thoracostomy had failed. A total of 90 patients were referred with an intercostal tube in situ, and in two patients the tube had penetrated the lung.

We did not subject any patient to a simultaneous bilateral operation because we prefer to use a lateral thoracotomy approach to achieve an extensive and safe parietal pleurectomy, as well as clipping off the leaking emphysematous bulla.

RESULTS

There was no hospital mortality and the only complication to occur was a persistent hemorrhage which required re-exploration in one patient. All patients made a good recovery by the time of discharge on the average of the 11th postoperative day. There has been no recurrence. This compares favorably with the apparently unsatisfactory conservative management with tube thoracostomy where the average admission duration was eight days and the recurrence rate was very high.

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REFERENCES


PIM Subtypes in Healthy Subjects and COPD Patients and Hardy-Weinberg Equilibrium

To the Editor:

In 1980 (Chest 77:761-63) a paper was published in which doubt was cast upon the equal valuation of PIM-subtypes. Our group consisted of 280 healthy subjects and 76 suffering from COPD; the results showed a high frequency of PIM. The hesitance of many experts with regard to the classification of M-Z heterozygotes led us to publish our work in order to provide a more complete, statistically representative study of this question.

In the opinion of Mittman and associates,1 the possibility cannot be ignored that this frequency of PIM types among patient populations tested could be due to the small total number of subjects. We have found that great importance should be attached to the selection criteria, and therefore we made the distinctions between "sick" and "healthy" more definite:

a) in the group classified as sick, airway resistance over the course of several years must have been in the range r<0.35 kPa/L/s; b) the diagnosis must be verified by x-ray examination; c) the criteria for healthy subjects must be just as carefully controlled.

The introduction of these specifications caused the number of subjects to fall to 130 healthy and 66 sick (COPD). Interestingly enough, the prevalence of PIM in the COPD group became even higher4 (Fig 1, 2).

Our observation of this high frequency does not fit expectations, which explains the responses of Kueppers5 and Massi.4 Separating the group PIM,M, offers considerable difficulty, and errors can easily occur in the phenotyping. In order to prevent such errors, we introduced the following measures:

1) after isoelectrofocusing, the minimum distance of the protein fractions M2-M8 should be more than 20 mm;
2) after every record test, a standard serum of phenotype PIM,M, was run.

The standard serum (point 2) was applied to one third of the plate area, allowing a controlled test of the development of the ampholyte species and the sensitivity of the plate simultaneously. When these specifications are observed, it is possible, for example, to distinguish the protein lines S4 from PIS and M6 from PIM. Such high sensitivity is not needed in order to separate the phenotypes PIM, PIM,M,
Fig 3. Isoelektrofokussierung according to Mittman and Taylor on PAG. PiM (Fig 3). When this special method is used, it is no longer possible to test up to 50 samples per plate, as with former methods.

Horne and Cockcroft recommended the use of the Hardy-Weinberg equilibrium in qualitative control of phenotyping. To illustrate, they selected two works (ours and that of Massi et al) in order to show that deviations from expected values when the Hardy-Weinberg equation is applied.

It is interesting to note that the greatest deviations from expected values found in other published studies, such as work reported by Charlionet et al, also occur in the difficult group cited above, but these practically disappear when the new phenotype PiM is introduced.

In the articles of Charlionet et al and Weidinger et al, subjects from one ethnic group who were determined to be free of tuberculosis, syphilis, hepatitis A, and hepatitis B were studied. It can be assumed that blood relationship among the subjects was also ruled out. Thus, all important requirements for the application of the Hardy-Weinberg equation were fulfilled.

In applying the Hardy-Weinberg equilibrium to the cases cited by us and by Massi et al, the following must be considered: when a homozygote carrier of PiZZ is found to suffer from COPD, this fact can be explained by a reasonable hypothesis, but when he does not, then no one can explain why. Equally, when carriers of other phenotypes, which include the greater portion of humanity, are found to suffer from COPD, no explanation can be offered.

The fact that the healthy subjects who were to be compared to the COPD group were selected according to the most rigid criteria still cannot obviate the possibility that in this group classified as healthy, there likely are some individuals whose gene patterns predispose them to the later development of COPD. The presence of a complex and hidden latent genetic disposition for the development of COPD acts as a limiting factor in the valid application of the Hardy-Weinberg equation.

Massi et al classify blood donors as healthy subjects and compare them with persons suffering from COPD. We feel, however, that although blood donors can in some sense be seen as healthy, it is not admissible to compare them as such with patients with COPD, because it cannot be ruled out that these persons might be suffering from light subclinical cases of COPD. In the selection of subjects, it is important that it be definitely determined whether or not COPD is present.

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REFERENCES
3 Kueppers F. PiM Subtypes in COPD. Chest 1981; 80:247

Respiratory Illness and Hypophosphatemia

To the Editor:
We would like to comment on some aspects of the article by Fisher et al entitled "Respiratory Illness and Hypophosphatemia," recently published in Chest (1985; 83:304-09). In which considerable comment is made on the relationship between hypophosphatemia and the worsening of ventilatory function. Two cases of mechanically ventilated patients are summarized in that article. One could not be weaned until normal serum values of phosphorus were reached, while the other died with a severe degree of hypophosphatemia in spite of the intensive efforts made in weaning the patient from the mechanical ventilator. We would like to report a woman who was given phosphate binding antacids, in which hypophosphatemia played an important role in the maintenance of her ventilatory failure. Furthermore, a clear relationship between the patient's maximal inspiratory pressure (Plmax) and her inorganic phosphate level was observed (r = 0.97; p < 0.01). In fact, the patient could not be weaned until the serum phosphate level was within normal ranges.

Muscular pressure is one of the major determinants of maximal flow, a parameter currently used in evaluating the success possibilities of the weaning. It can be measured clinically by determining the negative pressure generated during inspiration from functional residual capacity (Plmax). Moreover, alterations in Plmax reflect well enough the effectiveness of the muscular ventilatory pump of the patient; this is the reason why we think such a relationship (Plmax-inorganic serum phosphate level) is of so great an importance in the management of mechanically ventilated patients. Although hypophosphatemia had been implicated occasionally as the cause of respiratory failure, its role in that management has not been previously stated, at least not strongly enough. We would like to complete the clinical observations made by Fisher et al and emphasize the extreme necessity for obtaining and maintaining normal serum inorganic phosphate values in order not only to avoid the complications derived from the failure of the muscular ventilatory pump, but to achieve successful weaning.

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REFERENCES