domized controlled trials of the exercise component (intensity, upper versus lower limb, inspiratory muscle training, breathing exercises) as well as education and psychosocial support. We used a level-of-evidence scale that included not only statistical significance but also homogeneity of the results and their relationship to the “minimally clinically important difference” (MCID), which is the smallest difference perceived by the average patient.

However, there are some differences in the levels of evidence in support of the findings related to exercise and to quality of life (Table 1) which deserve comment. The differences likely reflect our preference for interpreting the meta-analysis with reference to the magnitude of the treatment effect and the MCID. Although most randomized trials have resulted in statistically significant improvements in exercise capacity, the magnitude of the differences between the treatment and control groups has been small and of unknown clinical relevance. The results of measures of functional exercise capacity have been heterogeneous. Thus, the MCID for the 6-min walk test is approximately 50 m and the best estimate of the effect of respiratory rehabilitation from the meta-analysis was 56 m (95% confidence interval, 28-93). Therefore, the limits of the confidence intervals around the effect size leave some doubt in our minds as to whether the treatment effect of rehabilitation on functional exercise capacity is significant. Consequently, we would conservatively assign grade B rather than grade A to the level of evidence supporting the use of respiratory rehabilitation to improve exercise capacity in patients with COPD.

For quality of life, we believe that stronger than grade-B evidence exists for at least some of its domains. This point is especially important as the choice of clinical outcome frequently determines the judgment of intervention effectiveness. Indeed, the treatment of COPD is primarily aimed at the relief of symptoms and improvement in the quality of life of affected individuals. Accordingly, we anticipate that health professionals who prescribe or deliver rehabilitation and those who allocate financial resources will be very interested in the effect of respiratory rehabilitation on quality of life. Randomized trials in which generic questionnaires (with little potential to detect changes in health status over time) are used might provide misleading conclusions as to the effectiveness of rehabilitation. Thus, in limiting the analysis to trials in which investigators used disease-specific questionnaires, we found that for dyspnea and mastery (two important domains of quality of life in patients with COPD), the treatment effect of rehabilitation exceeded the MCID. Therefore, we would assign a rating of A rather than B to such measures of quality of life.

For optimum resource allocation, any differences in support of the effectiveness of rehabilitation must be understood within their respective methodologic contexts. Guidelines should be evidence-based as well as being modulated by issues such as access, program availability, patient preferences, and resource constraints.

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REFERENCES

To the Editor

On behalf of the American College of Chest Physicians/American Association of Cardiovascular and Pulmonary Rehabilitation (ACCP/AACVPR) Pulmonary Rehabilitation Guidelines Panel, I appreciate the thoughtful and insightful comments of Drs. Lacasse and Goldstein in response to our Panel’s recently published review of scientific evidence in the field of pulmonary rehabilitation. It is gratifying that the results of our review coincide, in large part, with their excellent meta-analysis. The fact that both of these systematic reviews, performed independently and by different methodologies, came to similar conclusions provides further validation and weight to the evidence in support of the effectiveness of pulmonary rehabilitation in patients with COPD. It is striking that the similarities between the publications are greater than the differences.

The differences cited relate to the subjective interpretation of the strength of the published evidence in support of the primary documented benefits of pulmonary rehabilitation—namely, improvement in lower extremity exercise function, dyspnea, and quality of life. Drs. Lacasse and Goldstein correctly point out that the statistically significant published

| Table 1—Evidence Scores Attached to Respiratory Rehabilitation Outcomes |
|-----------------------------|-----------------------------|-----------------------------|
| Outcomes       | Results from Meta-analysis | Conclusions of ACCP/AACVPR Consensus Panel |
| Dyspnea        | A                          | A                          |
| Quality of life| A-B                        | B                          |
| Exercise capacity | B                      | A                          |

Communications to the Editor

Diagnostic Tests on Pericardial Fluid

To the Editor

We wish to comment on the article by Meyers and colleagues (May 1997) about the usefulness of diagnostic tests on pericardial fluid. While we acknowledge the authors' attempt to address this important issue with a rather large amount of data, we are concerned about the validity of the approach and conclusions.

Since the description of the diagnostic separation of exudates and transudates in pleural effusions, attempts have been made to apply the same concept to other effusions, without any evidence to support this practice. Indeed, the anatomy and physiology of the pericardium suggests that differences in the pathogenesis and pathophysiology of effusions may exist.

We feel that Meyers and colleagues failed to demonstrate in their study any usefulness in the cut points they used to identify an effusion as an exudate. As delineated in their Figure 2, the ability to use their cut points to distinguish transudates and exudates is basically nil, with most data points above their cut points for pericardial total protein, ratio of pericardial total protein to serum total protein, pericardial lactate dehydrogenase, and ratio of pericardial lactate dehydrogenase to serum lactate dehydrogenase. This is reflected by high sensitivities but specificities ranging from 13 to 40%.

Meyers and colleagues may have inadvertently contributed to their difficulty in demonstrating any utility by their initial assignment of various disease states as transudate. If the response of the pericardium is presumed to be similar to that of the pleura, the assignment of radiation, uremia, and trauma cases as transudates may be incorrect. Pleural effusions associated with these three diagnostic categories are generally exudates.

Meyers and colleagues have a large amount of data that may be useful in the diagnosis of pericardial diseases. However, in our opinion, analysis should be restricted to cases that meet presudy criteria for diagnostic categories, excluding cases based on presumptive clinical diagnoses. Similarly, the data should be presented in categories based on diagnosis rather than on preassignment as presumed exudates or transudates. This may allow clinicians to determine if there is any usefulness to the measurement of protein, lactate dehydrogenase, and the various ratios in delineating the underlying etiology for pericardial effusions.

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REFERENCES


To the Editor:

I thank Drs. Effren and Obaji for their interest in our article. I wish to respond to their three concerns.

We first analyzed our data stratified by exudate/transudate. For each disease, the classification was based on literature.

We did not require histologic confirmation of the diagnoses. Given the diseases evaluated, this would have been unrealistic. Indeed, pre-established criteria were used to confirm the clinical impression in each case.

I am unsure that I understand their third concern about our failure to analyze by disease. In Tables 2 to 4, 17 characteristics of pericardial fluid are evaluated individually for each of the 10 diseases. No useful differences were found.

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REFERENCE


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