because of an inadequate data base (the point of my column).
I am sorry that the word "pressure" was inadvertently substituted for the word "resistance," but what has that to do with the relationship between pulmonary vasodilator treatment and patient outcome? To state that the level of PVR correlates in untreated patients with survival and, hence, to imply that reducing PVR by drugs improves survival, is a variant of a famous logical error.

Aristotle is an animal.
A horse is an animal.
Aristotle is a horse.

Proof of decreased mortality requires a demonstration that the length of survival is increased.
I am, of course, delighted that after a decade of widespread use of pulmonary vasodilators, their safety and efficacy will at last be tested. If you have any influence on these clinical trials, could you suggest that: 1) workup deaths (deaths resulting from catheterization, recatheterization, open lung biopsy and drug titration, etc.) be included in the final statistics; 2) patients be informed that premature death is one of the possible outcomes; 3) sequential analysis be performed so that as few patients as possible are exposed to the hazards of the trial; and 4) an estimate of quality of life in the treated vs untreated group be included so that if there is a significant subgroup which is improved, this will not be overlooked. The New York Heart Association classification could be used for this purpose.

Details concerning patient protection during clinical trials can be found in Silverman's book,7 a complex treatment by Feinstein,8 and a simplified account by me.15

I did not claim personal rigorous honesty. The words in my column say, "the candidates are provided rigorously honest informed choices about the procedure (heart-lung transplantation)." The honesty is provided by my surgical and medical colleagues who frequently serve as my medical conscience. Judging by the lack of understanding manifested by some patients with PPH during treatment with pulmonary vasodilators, perhaps the more general use of surrogate consciences might not be a bad idea.3

The data which I used from the PPH Registry were provided in the form of periodic printed reports from the Registry, by my direct communication with Drs. S. Rich and P. Levy (who were very forthcoming as was I), and by a definitive publication in the Annals of Internal Medicine.4

I do plan to discuss the PPH Registry report in a future column. I believe the term "vilify" to be doubly inaccurate. It is inaccurate from the semantic standpoint and it does not describe my state of mind. However, as my mindset has become an issue, let me describe it as accurately as I can. For this purpose, perhaps the word "appalled" would be the most accurate term. It is difficult for me to accept the fact that patients died during the course of being worked up for a registry. I have been informed that there were deaths clearly related to drug titration and probably to open lung biopsy. The latter issue became sharp enough so that open lung biopsy was explicitly removed as a requirement for inclusion in the Registry. The final report equivocates with respect to this issue as follows: ... ten reported adverse reactions from the catheterization (not including drug testing).15 Does this mean that there were no adverse reactions during drug testing (I know that there were), or does this mean that there were deaths during drug testing but, if the patient died at that stage, he/she no longer qualified for the Registry and was excluded from the data? If the latter is true, as I have been led to believe, this is surely one of the most ironic literary treatments of human death since Gogol's "Dead Souls." 40 And were there no deaths during Registry workups from (surrounding) open lung biopsy? If my information is wrong and no patient died during the peri-workup period, then I will issue a public apology in my column on the Registry report. I will predict that no apology will be required.

I do not agree that past therapeutic efforts in PPH are the process by which "more pieces will be yielded until the mystery of PPH is ultimately solved." As noted above, there are well defined processes for establishing the safety and efficacy of therapeutic modalities in medicine. These have not been used. As a result, what has taken place does not even resemble jigsaw puzzle solving. It has been more like using a Ouija board to come up with answers.

I realize that in an exchange which is this sharp, the original letter writer does not have the chance for a final rebuttal. In consideration of this, I invite Dr. Rubin to submit a column for Chest on "the risks benefits of pulmonary vasodilators in pulmonary hypertension" so that you can present your ideas in greater detail and without a rebuttal by me.

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REFERENCES

Detection of Right Pulmonary Artery Thrombosis by Two-Dimensional Echocardiography

To the Editor:

Two-dimensional echocardiography is frequently used in evaluation of right heart structures in patients with suspected or proven pulmonary artery thromboembolism,1,2 and pulmonary artery thromboses have been imaged using suprasternal views.2,3 We have recently imaged a right pulmonary artery thromboembolus in a patient using standard parasternal short axis views.

The patient was a 75-year-old man admitted for substernal chest pain, dyspnea, and transient cyanosis. He had a history of polymartosis nodosa treated with prednisone, 20 mg bid, and had recently fallen, sustaining injury to his right leg. His heart rate was 100 bpm, blood pressure 148/80 mm Hg, and respiratory rate 22 cycles/min. His neck veins were distended and he had a right ventricular gallop. There was 2+ pitting edema of the right lower extremity.

Two-dimensional echocardiographic examination on the second hospital day (Fig 1) showed a right pulmonary artery thromboembolus. Repeat-echocardiography after three weeks of anticoagulation showed complete resolution of the thromboembolus.

In our experience, two-dimensional echocardiography has been helpful in the diagnosis of pulmonary embolism in several ways. First, this technique allows detection of entrapped right heart...
thromboembolism,\textsuperscript{1} which may allow the diagnosis to be made noninvasively and avoid potentially hazardous invasive procedures. Second, the finding of right ventricular volume and/or pressure overload heightens the clinical suspicion for pulmonary embolism. Finally, two-dimensional echocardiography may actually demonstrate pulmonary artery thromboembolism. DiCarlo and Kasper have detected pulmonary emboli with two-dimensional echocardiography using suprasternal views.\textsuperscript{4,5} As the current case demonstrates, the standard short axis view may also be used to detect pulmonary embolus.

The sensitivity and specificity of echocardiographic techniques for diagnosing pulmonary embolus is unknown, but they may have a role in screening patients for suspected pulmonary embolism.

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Criteria for Steroid Dependence

To the Editor:

Steroid dependence in bronchial asthma is a clinical phenomenon with no diagnostic biochemical parameters.\textsuperscript{1} Its grave implications have led us to categorize steroid-dependent asthmatic patients as a clinically identifiable subset of asthmatic patients. Several trials\textsuperscript{4} have been undertaken to replace oral or parenteral steroid therapy with aerosol steroid therapy in this group. Published results\textsuperscript{5} so far have been conflicting, mainly because the criteria for selection of cases have been ill-defined. A clear definition of the term steroid dependent has thus become necessary. We have observed that steroid dependence generally occurs following uninterrupted steroid intake for more than a year at a dosage of 0.3 mg/kg/day. Occasionally, it may occur earlier with a higher dosage. We have formulated a set of norms to label an asthmatic patient as steroid dependent (Table 1). These have been followed in our institution for over a decade now. It is hoped that, with uniform guidelines to follow, results of trials on steroid-dependent asthmatic patients will be more comparable.

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REFERENCES


2 Brompton Hospital/Medical Research Council Collaborative Trial. Double-blind trial comparing two dosage schedules of beclomethasone dipropionate aerosol with a placebo in chronic bronchial asthma. Br J Dis Chest 1979; 73:121-32


5 Brompton Hospital/Medical Research Council Collaborative Trial. Double-blind trial comparing two dosage schedules of beclomethasone dipropionate aerosol in the treatment of chronic bronchial asthma. Lancet 1974; 2:303-07


Table 1—Criteria for Steroid Dependence

1) Sign and symptom score\textsuperscript{1} of asthma worsens on temporary stoppage of steroid therapy while on the usual schedule of bronchodilator treatment.

2) FEV\textsubscript{1}/VC ratio, PEFR and other parameters of pulmonary function decrease on steroid withdrawal. Evidence of reversible airways obstruction may even be absent.

3) Pulmonary function parameters and sign/symptom score do not significantly (<15%) improve after increasing the usual daily dose of bronchodilator medication (oral or parenteral).

4) Reasonable improvement (≥15%) in these parameters is evident only after the administration of two to three times the previous daily steroid dosage. (Improvement is shown in the reduction of the asthma sign and symptom score, improvement of parameters of pulmonary function, and an increase in airway obstruction reversibility).

5) Symptoms of steroid withdrawal (eg, lethargy, headache, weakness, pseudorheumatism, emotional disturbances, etc) are precipitated after stoppage of steroid therapy. This is a feature of physiologic dependence.