Many people want to change the rules when they look at the right heart. My perception is that the very same rules should be applied perhaps more stringently when considering any method of assessment of right heart function. The variable shape of the human right ventricle makes it more difficult by every method. 3

The concept advanced by Albert is a myth about gated blood pool ventriculography which has been perpetuated for over a decade. The myth is summarized in a figure from Strauss and Pitt’s textbook Cardiovascular Nuclear Medicine. What that figure purports to show is that in the conventional left anterior oblique gated blood images, the contamination of left ventricular counts by left atrial counts is trivial but the contamination of right ventricular counts by right atrial counts is insurmountable! Accordingly, the dogma has been uncritically disseminated that gated blood pool data regarding the right ventricle is inherently inaccurate. To my knowledge, the numbers in this figure are not measurements. What is clear is that the anatomy is unusual; the right atrium is usually not four times larger than the left atrium; neither is the right ventricle smaller than the left ventricle. By observing the first passage of a tracer in first pass studies, an RAO view can be used to separate right atrium from right ventricle spatially while the left and right hearts are separated temporally. The point which has never been emphasized is that first pass techniques also have potential inaccuracies! Count rates can be one major problem with first pass studies. Therefore, given the difficulty of the right ventricle, one should validate whatever method one uses.

We have compared single and biplane contrast studies with first pass and blood pool radionuclide and echocardiographic images in hundreds of patients in multiple laboratories over a ten year period. We have shown that one can obtain reasonable gated blood pool measures in a majority of patients just as in individual patients, each method can come up short. We specifically and mathematically have dealt with the issue of chamber overlap by subjecting our gated blood pool images to phase analysis, where by atrial and ventricular pixels are 180° out of phase. Most groups using first pass methods of right ventricular assessment have not validated their methods against anything!

The simple fact is that much progress has been made in overcoming the greatest myth about the right heart, namely that it is dispensable. In accord with that myth, there was no reason to study the right heart at all! Having got past that myth, we next need to defeat the myth that it cannot be studied because it is too complicated. It is likely that the greatest progress will be made when we recognize that multiple different approaches each applied with the same consistency and rigor that is expected on the left side, will finally yield the truth.

Douglas A. Morrison, M.D., F.C.C.P.
University of Colorado Health Sciences Center, Denver

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2 Albert PK. Assessment of right ventricular function. Chest 1980; 94:1123-24
7 Morrison DA, Turregion J, Ovitt T. Right ventricular ejection fraction measurement: contrast ventriculography versus gated blood pool and gated first-pass radionuclide methods. Am J Cardiol 1984; 54:651-53
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To the Editor:

In the editorial to which Dr. Morrison objects, I summarized the literature addressing the accuracy of the equilibrium blood pool method for measuring right ventricular ejection fraction (RVEF). In this editorial I noted that two groups have reported different accuracies in patients with low RVEFs. Morrison et al found that equilibrium RVEFs correlated well with measurements made by other techniques, regardless of the RVEF, while Xue et al found that the accuracy decreased at lower RVEFs. Despite considering each of the references cited by Dr. Morrison in the above letter, I encountered no information suggesting that my summary was in error. However, I concur with his suggestion for the need to validate whatever method one uses, and agree that he has made the most thorough attempt to do so to date.

The comments pertaining to the paper of Biermann et al are quite correct. I overlooked the problem of tricuspid regurgitation and appreciate Dr. Morrison noting this as an additional and important limitation.

Richard K. Albert, M.D., F.C.C.P.
Veterans Administration Medical Center
Seattle

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CF and Emphysema

To the Editor:

I enjoyed the symposium on alpha-antitrypsin deficiency (Chest 1988; 95:151-208), and found it concise, informative, and interesting. I must, however, take exception to Dr. Wewers’ implications in using cystic fibrosis (CF) as a model for his theory of protease/antiprotease imbalance in emphysema. He states that emphysema develops relatively rapidly in CF patients, and implies that the small amount of actual emphysema noted in autopsies is because of their short lifespan. However, this does not go along with the facts. In all series of autopsies—even those with relatively older patients—emphysema plays a very minor role. Indeed, this has been remarkable in the face of clinically evident air-trapping and the very imbalance to which Dr. Wewers refers. This relative absence of emphysema is reported even in patients with both CF and alpha-antitrypsin deficiency. Dr. Wewers quotes Bruce et al to show that the imbalance of protease/antiprotease activity exists. That study
was from expectorated sputum which poorly (at best) reflects what goes on at the alveoli, where destruction in emphysematous patients must take place. Furthermore, the reported autopsy material—while showing some abnormal elastin fibers in the airways and alveoli—did not report an increase in parenchymal destruction. Thus, I feel Dr. Wewers would be better advised to try to explain why CF patients are an exception to his theory instead of using them as support of his contentions. Indeed, if there is a connective tissue imbalance in the lungs of CF patients, it appears to be peribronchial fibrosis, the implications of which deserve further investigations.

Clarke McIntosh, M.D.,
Pediatric Pulmonary Fellow,
Pulmonary Division,
The Children's Hospital of Alabama,
Birmingham

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To the Editor:

I appreciate the interest Dr. McIntosh has demonstrated in emphasizing the potential that exists in studying cystic fibrosis as a model of protease-induced lung destruction. His contention is that parenchymal destruction that occurs in cystic fibrosis is relatively mild when compared to the measured elastase excess in samples from the airways of these patients. I cannot agree more. The large amount of elastase present in the airways of cystic fibrosis patients is indeed striking. How this elastase activity is prevented from reaching and damaging the alveolar spaces is certainly intriguing and worthy of future study.

However, I think that Dr. McIntosh has missed the point of the discussion. The review states that cystic fibrosis is a "disorder characterized by bronchiolitis that progresses to bronchiectasis" and that there is evidence for proteolytic destruction of lung connective tissue in cystic fibrosis . . . at sites of infarction (predominantly the bronchial ules and abscesses) where "there were fragmented and exfoliated elastin fibers." The point I attempted to make is that, in cystic fibrosis, one sees destruction of lung connective tissue predominantly at the sites of elastase excess. The presence of fragmented elastin fibers at these sites is evidence for a role of protease excess in the pathogenesis of the bronchial wall damage that occurs in cystic fibrosis. It was not my intention to suggest that the lung destruction in cystic fibrosis is primarily emphysema. Dr. McIntosh evidently equated all lung destruction with emphysema. Unfortunately, he misinterpreted and misquoted the review. It was never stated nor implied that "emphysema develops relatively rapidly in CF patients" or that "the small amount of actual emphysema noted in autopsies is because of their short life span".

Again, I submit that the protease excess in the airways of patients with cystic fibrosis is relevant to the pathogenesis of the destructive airway disease. There may also be some associated emphysema but the bulk of the destruction is in the airways. In this context, an additional comment may be appropriate. Since alpha,-antitrypsin is now available for treatment of alpha,-antitrypsin deficient individuals, studies to evaluate the potential benefit of inhibiting elastase activity in the airways of patients with cystic fibrosis are now conceivable and warranted.

Mark D. Wewers, M.D., F.C.C.P.
Pulmonary and Critical Care Division,
Ohio State University,
Columbus

Drug Abuse and Aneurysm

To the Editor:

I read with interest Dr. Bush's case report of intracranial hemorrhage following thrombolytic therapy in an intravenous drug abuser and feel that additional comments are warranted.

Notwithstanding the absence of a mycotic aneurysm at autopsy, the patient may have had a previously asymptomatic mycotic aneurysm. Creation of septic bacterial emboli in an animal model has produced unruptured mycotic aneurysms. In addition, incomplete treatment with antibiotics in these animals resulted in chronic mycotic aneurysms with associated deep abscesses. Hemorrhage can destroy previous arterial pathology of mycotic aneurysms; this may have accounted for the autopsy finding. However, in Dr. Bush's presentation, there is no mention at all of a prior history of bacterial endocarditis (treated, incompletely treated, or untreated). Without this history, I am skeptical about Dr. Bush's presumptive diagnosis of ruptured mycotic aneurysm.

Secondly, there is no known correlation between the presence of pulmonary talc emboli and that of cerebral mycotic aneurysm, as suggested.

The author does not mention the time of last intravenous drug exposure. This is important, as I would have less trepidation administering thrombolytic therapy to someone whose last exposure was 20 years ago than to one who is actively engaged in IV drug use.

Finally, the dose of tPA used (120 mg) is above that which is currently recommended (100 mg). There seems to be a dose-related incidence of intracerebral hemorrhage with tPA, demonstrated by the TIMI trial data.1 In addition, rates of intracerebral bleeding are reported to be lower with streptokinase than with tPA.4

In summary, I agree that a history of intravenous drug abuse should be a factor when considering a patient for thrombolytic therapy. However, it should be individualized (ie, last drug use, prior history of endocarditis), and streptokinase may be considered instead of tPA as the intracerebral bleeding rate may be lower.

David Lee Scher, M.D.,
Philadelphia Heart Institute,
Presbyterian-University of PA Medical Center,
Philadelphia

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1 Bush HS. Cocaine-associated myocardial infarction. Chest 1988; 94:878

Erratum

The Editorial Department of CHEST apologizes for having inadvertently omitted the name of Lawrence E. Kellett, Visalia, California, as a co-author of the article, "Bilateral carotid body resection for the relief of dyspnea in severe chronic obstructive pulmonary disease" (Chest 1989; 95:1129-30).

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