shown that patients with OSA often have cranio-facial abnormalities that play a role in the development of their disease. Very obese patients often have few of these abnormalities, but it is highly recommended that patients be reassessed when weight loss has been sufficient to eliminate residual apneas related to upper airway anatomic abnormalities that persist despite weight loss. Considering the new weight-reduction programs now available with regular long-term follow-up by multi-specialists, one may have to decide which program (surgical or medico-behavioral) will be able to provide the most appropriate and significant weight reduction with maintenance of weight loss and with the least amount of morbidity risk for each individual case. However, nasal CPAP (or tracheostomy), with or without supplemental oxygen, will initially be required if any of these weight loss-related approaches is selected for immediate control of OSA and related clinical symptoms.

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REFERENCE

To the Editor:

In two articles published in the December issue of Chest, the authors fail to refer to the effectiveness of gastric surgery-induced weight loss for the treatment of morbidly obese patients with obstructive sleep apnea syndrome (OSA). The article by Nahmias and Karetzky offer follow-up studies on the use of a nasopharyngeal tube for the treatment of patients with OSA when they are unable to be treated with nasal CPAP. This would certainly be an ideal temporizing measure prior to gastric bypass surgery for obesity. The article by Partiten, Jamieson and Guilleninault noted a significantly greater mortality rate in patients "conservatively treated for weight loss", as compared to those who underwent tracheostomy. Dietary treatment of morbidly obese patients is notorious for an extremely high failure rate or incidence of recidivism. Gastric bypass surgery is associated with loss of approximately two-thirds excess weight within one year with correction of OSA syndrome in over 90 percent of patients. Physicians who manage these patients should be aware of this definitive modality of therapy.

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To the Editor:

As mentioned in our publication, the nasopharyngeal catheter is meant to be used during the weight loss period, which we feel is the definitive treatment for OSA. Although only 20 to 25 percent of our patients are able to lose weight and maintain their weight loss, we feel dietary treatment is the primary modality which should be attempted. To this end we work closely with the metabolic clinic at our sleep center which utilizes a 450 calorie, protein sparing (Optifast) diet, and have achieved good results.

If, however, the morbidly obese patient cannot lose weight in a reasonable period of time, we recommend gastric stapling or bypass surgery. In the few patients who have opted for gastric surgery, their disordered breathing events have been eliminated. The problem remains that the majority of patients offered surgery refuse it despite their dietary failures. We utilize the nasopharyngeal tube or nCPAP in the pre- and postoperative periods and until the patient has achieved sufficient weight loss which, as you mention, is usually rapid.

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Right Ventricular Volume Assessment
An Alternative Perspective

To the Editor:

In the December issue of Chest, Biernacki and co-workers report the results of a study using blood pool radionuclide measured right ventricular ejection fractions and thermodynamics measured stroke volumes to calculate right ventricular systolic volumes. In an accompanying editorial, Albert reviews some of the difficulties in using gated blood pool ventriculography to measure right ventricular ejection fractions, implying that this is the major problem in Biernacki et al's study. It is my thesis that blood pool radionuclide ventriculography has been validated substantially better than the more widely accepted first pass methods.

Most new fellows in the cardiac catheterization laboratory discover that there are potential errors in using the Sandler and Dodge formulae to measure the left ventricular ejection fraction from either single plane or biplane contrast left ventriculograms. Small differences in the way an end-systolic image is traced can lead to large differences in ejection fraction. Small differences in magnification factors beget significant differences in volumes. The formulae are based on volume assumptions (an ellipse of rotation) which sometimes do not appear warranted (especially in dilated hearts). Premature beats stimulated by the power injection through an intracardiac catheter can lead to large differences in ejection fraction. In the same way that an average of multiple thermoludion cardi output measurements is likely more accurate than a single injection, the most accurate left ventricular ejection fraction is likely an average of several calculations. Nevertheless, most physicians or technicians will trace (and planimeter) one time. Despite these and other problems, contrast ventriculography is the "gold standard" for left ventricular ejection fraction measurement. The upshot of all of these factors is that clinicians develop "a sense of humor" with which to take any clinical data. They also appreciate the need to validate new testing procedures and to consider the reproducibility of their methods.
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. 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 pool 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.,
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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 Biernecki 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

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