and control of hemorrhage technically much easier. Those who are unfamiliar with the instrumentation will find themselves lost; those who are overly enthusiastic will find themselves in trouble; and those who lose sight of their priorities will do harm to their patients. Video thoracoscopy rationally practiced by qualified, responsible physicians offers a great deal to our patients. With continued improvements in instrumentation and increased familiarity with the technique, we can expect to see expanded utilization of this procedure with direct benefit to patients. Its role in the management of primary lung cancer is very limited at present.

Douglas J. Mathisen, M.D., F.C.C.P.
Boston

General Thoracic Surgical Services, Massachusetts General Hospital

Whither Goest the Right Ventricle in Obstructive Sleep Apnea?

Since the initial definition of obstructive sleep apnea (OSA), numerous attempts have been made to determine the relationship between cardiovascular function and this syndrome. However, much of the data concerning both right and left ventricular manifestations is inconclusive. The study by Hanly et al in this issue of Chest (see page 100) adds to the dilemma.

Hypoxic pulmonary vasoconstriction has been recognized since 1947.1 Chronic intermittent hypoxemia is a frequent nocturnal occurrence in OSA, and it may be present continuously as the syndrome worsens. Elevation of pulmonary artery pressure demonstrated in OSA may be the result of this hypoxemia, and cor pulmonale is not uncommon in OSA.5,3 Therefore, right ventricular hypertrophy (RVH) in patients who present to a sleep laboratory should be an expected result of this elevation in pulmonary artery pressures. Bradley et al4 have suggested that daytime hypoxemia, in addition to OSA, is necessary to develop cor pulmonale in OSA patients. Krieger et al5 confirmed that impaired daytime pulmonary function characterized by diffuse airway obstruction contributed to pulmonary hypertension, hypoxemia, and hypercapnia in OSA. They showed also that the severity of sleep-related breathing disorders played a significant role. In addition, leftward shift of the interventricular septum has recently been noted in OSA.6 This phenomenon is secondary to increased right ventricular volume during obstructive inspiratory efforts, with exceedingly negative values of intrathoracic pressure. Such effects on the right ventricle with increases in both preload and afterload may also lead to RVH.

This background of data has led to an attempt to determine the status of the right ventricle in newly diagnosed sleep apnea. In this issue of Chest, Hanly et al report the absence of echocardiographic evidence of RVH in OSA. In a similar study, we found the incidence of isolated RVH to be 71 percent in patients with newly diagnosed sleep apnea.7 Since our initial study, we have enlarged our group to 39 patients with OSA, with a 69 percent incidence of RVH. Such a discrepancy between two reputable sleep laboratories requires an attempt at an explanation.

A difference between the two populations in these two studies may be significant. We excluded patients with obstructive disease and restrictive abnormalities not secondary to obesity, but did not exclude patients with daytime hypoxemia and/or hypercapnia. The difference in exclusion criteria may have led to some of the disparity in the incidence of RVH. Krieger et al5 clearly pointed out the significance of daytime hypoxemia and hypercapnia in OSA in causing pulmonary hypertension. Bradley et al4 demonstrated similar findings in the development of cor pulmonale in this group. However, OSA may result in RVH secondary to nocturnal hypoxemia. Progression to persistent pulmonary hypertension and cor pulmonale may ensue only with the development of daytime hypoxemia and associated pulmonary function abnormalities.

Despite these findings, we feel that most of the differences are due to the relatively imprecise echocardiographic methodology of determining right ventricular wall thickness (RVWT). Many inherent problems are encountered in measurement of RVWT, including the complex geometry of the chamber (truncated and crescentic), the variable trabecular pattern of the free wall, asymmetric hypertrophy, retrosternal location, and axial orientation. The degree of accuracy also depends upon technique and equipment selection. Although RVWT greater than 5 mm is the recognized critical measurement for RVH, other parameters have been cited, including 5.9 ± 0.9 mm using M-mode echocardiography.8 M-mode echo criteria have a sensitivity of 93 percent and a specificity of 95 percent when correlated with autopsy data.10 However, some investigators have found a sensitivity of only 67 percent.11 For mild RVH, two-dimensional echocardiography values for RVWT of 8 mm or greater have been cited.12 Visualizing the RVW with two-dimensional echocardiography is difficult, and this technique should be used in conjunction with M-mode imaging.13 A weak correlation between echocardiographically determined RVWT and pulmonary artery pressure in chronic obstructive lung disease has been noted.14 In view of these many difficulties, we elected to use an RVWT of 6 mm or greater; Hanly et al selected a cutoff of 5 mm. A question still exists regarding the true thickness that reflects RVH.

Echocardiographic interpretations of RVH vary
greatly and are difficult to reproduce. For this reason, we chose to use two cardiologists, each blinded to the interpretations of the other, since inherent flaws in interpretative technique would be perpetuated with one observer, but would likely be canceled out with two observers. Hanly et al used one cardiologist with technicians for backup verification, a technique that may have led to inbred errors. Obviously, the evaluation of RVWT by echocardiography is far from exact. These difficulties may account for some of the differences between the study by Hanly et al and ours.

The echocardiographer must determine the line of separation between the right ventricular wall and the sternum, a determination that is not exact and may lead to fairly large relative errors. A misreading of 1 mm will markedly distort the incidence of RVH in OSA. Determination of the true incidence may have to await improved techniques of measuring RVWT. Perhaps advances in magnetic resonance imaging, three-dimensional echocardiography, transesophageal echocardiography, or cine computed tomographic scanning, or even placement of a transducer within the right ventricle capable of measuring wall motion and thickness, may be necessary.

The relationship of RVWT, RVH, and OSA remains elusive, but the incidence of RVH undoubtedly will be found between the 0 percent of Hanly et al and the 71 percent in our study. Determining the true incidence awaits further refinement in the methodology and technique of determining RVH and in better delineation of the population under study.

Robert J. DiBenedetto, M.D., F.C.C.P.;
Lloyd Goodman, M.D.;
Robert Rollings, M.D., F.C.C.P.;
Earl Berman, M.D.;
Don Causey, R.C.P.T., C.R.T.T.
Savannah, Georgia

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The Adverse Effect of Blood Transfusion in Lung Cancer

We do not err because truth is difficult to see. It is visible at a glance. We err because this is more comfortable.—Solzhenitsyn

What is the effect of perioperative blood transfusion on recurrence and survival in early-stage non-small cell lung cancer? This question has been posed numerous times, and several studies have attempted to answer it,1-8 the latest being the study by Pena et al in the current issue of this journal (see page 84). In one regard, the article by Pena et al is mistitled in that it speaks to the "Significance of Perioperative Blood Transfusions..." (emphasis mine), as if another small retrospective study would be the definitive one. In another sense, the article is properly titled in that it seems to deal primarily with the statistical significance of blood transfusion as a prognostic factor. Some other authors have made the same methodologic error in equating clinical effect with statistical significance. However, significance testing does not answer the basic biologic question. More about this below.

The Hypothesis

The origins of the blood transfusion hypothesis are well founded enough to make chest surgeons uncomfortable. Blood transfusions have a well-established beneficial effect on renal transplants, probably because