take over the brain, liver, or kidneys following abnor-
mal passage through the pulmonary circulation; and
pulmonary angiography may show dilated precapillar-
ies as a "spongy" angiography appearance or discrete,
small arteriovenous connections most commonly in
the lung bases. Second, any of the above findings
occurring in the setting of liver disease and hypoxemia
(PaO₂ < 70 mm Hg) should be considered sufficient
clinical information to diagnose the syndrome.

Interestingly, many of these individuals may have
profound hypoxemia and a significant response to 100
percent supplemental oxygen. Therefore, the term
"intrapulmonary shunt" is really inappropriate in
terms of the clinical response to oxygen. For this
reason, we prefer the term intrapulmonary vascular
dilatation, and the physiologic condition is probably
best described by the inert gas elimination technique
as a diffusion-perfusion defect.7 These dilatations
anatomically assume at least three forms. First, there
are diffuse precapillary dilatations ranging from 15 to
160 μm in diameter; second, there are true arterio-
venous communications that may occur either at the
hilar level or in the lung bases and have an appearance
angiographically similar to arteriovenous malforma-
tions; and third, there are pleural-based arteriovenous
"spiders."1,8,9

Etiology of this syndrome is unclear at this time,
even though a variety of mediators are possibly
responsible for pulmonary vasodilations. Can pharma-

cologic approaches improve the hypoxemia? Ad-
mistration of almitrine bimesylate and the use of
therapeutic plasma exchange have not resulted in
consistent improvement.3 Recent studies have sug-
gested that somatostatin analogue may improve hy-
poxemia associated with this syndrome.10 A prospec-
tive clinical study is underway at our institutions
assessing the effect of somatostatin analogue in hep-
topulmonary syndrome.

Finally, liver transplantation has resulted in the
resolution of hypoxemia in this syndrome.2,3,9 but such
success is not universal.4,11 Transplantation, clinical
studies, and animal studies now provide an opportu-
nity to study this unusual pulmonary pathophysiologic
condition many years after the initial descriptions of
the abnormality.8,9

Michael J. Krowka, M.D., F.C.C.P., Jacksonville, FL*
Denis A. Cortese, M.D., F.C.C.P., Rochester, MN†

*Head, Section of Thoracic Diseases, Mayo Clinic Jacksonville.
†Professor of Medicine, Mayo Medical School, Mayo Clinic, Roch-
ester.

Reprint requests: Dr. Krowka, Mayo Clinic Jacksonville, 4500 San
Pablo Road, Jacksonville, FL 32224.

REFERENCES
1 Sherlock S. Disorders of the liver and biliary system, ed 8.
2 Eriksson LS, Soderman C, Wahren J, Ericzon BC, Eleborg L,
Hedenstierna G. Is hypoxemia in cirrhotic patients due to a
functional 'hepatopulmonary' syndrome? J Hepatol 1988;
7(suppl):529
3 Krowka MJ, Cortese DA. Pulmonary aspects of liver disease
4 Van Thielt DH, Schade RB, Gaveler JB, Shaw BW Jr, Iwatsuki
S, Starzl TE. Medical aspects of liver transplantation. Hepatol-
y 1984; 4(suppl):79-83
5 Maddrey WC, Van Thielt DH. Liver transplantation: an overview.
Hepatology 1988: 8:948-59
6 Stoller JK, Moodie D, Schiavone WA, Vogt D, Broughan T,
Winkelmann E, et al. Reduction in intrapulmonary shunt and
resolution of digital clubbing associated with primary biliary
cirrhosis after liver transplantation. Hepatology 1990; 11:54-8
7 Edell ES, Cortese DA, Krowka MJ, Rehder K. Severe hypoxe-
8 Berthelot P, Walker DG, Sherlock S, Reid L. Arterial changes
in the lungs in cirrhosis of the liver and lung spider nevi. N Engl J
Med 1966; 274:291-98
9 Williams A, Trewby F, Williams R, Reid L. Structural altera-
tions to the pulmonary circulation in fulminating hepatic failure.
Thorax 1979; 34:447-53
10 Salem O, Dindzans VJ, Freeman J, O'Dorisio T, Ruthardt F, Van
Thiel DH. Liver transplantation following preoperative closure
Intrapulmonary vascular dilatations (IPVD) in liver transplant
candidates: screening by two-dimensional contrast enhanced
echocardiography. Chest 1990; 97:1165-70

Thermodilution Right Ventricular
Ejection Fraction
Remain Questions

The ability to measure right ventricular volume and
ejection fraction at the bedside of critically ill patients
has renewed interest in this oft-forgotten component of
the central circulation. Seminal investi-
gators such as Fineberg and Wiggers1 and Guyton
et al2 recognized the critical role of the right ventricle
(RV) in supporting the circulation during respiratory
failure or pulmonary hypertension. The inability of
clinicians to easily and reliably measure right ventric-
ular function in their patients, however, has resulted
in a relatively limited understanding of its role in
clinical disease. Using thermal dilution techniques to
measure right ventricular volume and ejection fraction
was investigated intensively in the 1960s.3,4 Cum-
bersome calculations and slow thermistor response times
limited its usefulness. Fast computer algorithms which
account for the limits of a catheter-mounted
thermistor4 now offer the possibility of simply and
repeatedly assessing right ventricular function in critic-
ally ill patients.7,8 Such technical refinements are
crucial to improving the accuracy and reproducibility
of the technique.
Studies evaluating the thermodilution method have found that the technique is reasonably reproducible, with coefficients of variation ranging from 7 to 12 percent, and can be correlated with other methods of measuring right ventricular function, such as twodimensional echocardiography and radionuclide angiography. How can the reproducibility and accuracy of the method be improved? The technique, like other indicator dilution methods, assumes that the indicator is delivered as a bolus, mixes quickly and homogenously in a single chamber, and is sensed with high fidelity. The data from the thermistor must then be correlated with a signal of ventricular systole (such as the R-wave of the electrocardiogram), accurately analyzed, and interpreted. The current study by Spinale and coworkers (see page 1259) sheds light upon several systematic errors contributing to variations and inaccuracies in the thermodilution method. These workers report that the RV ejection fraction may be systematically underestimated if the thermistor is placed too far from the pulmonic valve or if the injectate port is placed too high within the body of the right atrium. The study suggests that careful and consistent positioning of the injectate port and thermistor will improve the accuracy and repeatability of thermodilution RV ejection fraction measurements. Since this study was performed using small anesthetized pigs, it remains to be seen whether the same effect is important in human patients.

Other important methodologic questions must still be evaluated. The effect of valvular lesions such as tricuspid regurgitation is uncertain; theoretically uneven mixing and loss of indicator might occur. Additionally, atrial arrhythmias and atrial fibrillation, common in patients with right ventricular dysfunction, may unacceptable increase the variability of the technique. The application of thermodilution RV volume and ejection fraction measurements also deserves careful study. Conceivably, knowledge of right ventricular volume may be useful in clinical states in which changes in intravascular pressure do not reflect equivalent changes in intravascular volume. This situation may be found in patients with acute lung injury, pulmonary hypertension, cardiac tamponade, direct right ventricular injury or infarction, or receiving positive pressure ventilation with high levels of PEEP. These patients might be managed more intelligently if right ventricular volume were known. Initial clinical investigations using thermodilution and other methods, such as radionuclide angiocardiography, to measure right ventricular function are promising. Martyn and coworkers suggest that right ventricular end-diastolic volume more accurately estimates right ventricular preload than does central venous pressure. Additionally, changes in the RV ejection fraction may reflect important alterations of cardiovascular function which were previously difficult to diagnose using routine hemodynamic monitoring. Studies such as that of Spinale and coworkers, which strive to identify and eliminate systematic errors in the method, may make the thermodilution technique more consistent and reliable, and improve the chance that the measurements made will be clinically useful.

William E. Hurford, M.D., F.C.C.P.
Boston

Department of Anesthesia, Massachusetts General Hospital, Harvard Medical School.
Reprint requests: Dr. Hurford, Department of Anesthesia, Massachusetts General Hospital, Boston 02114

REFERENCES
4 Rapaport E. Usefulness and limitations of thermal washout techniques in ventricular volume measurement. Am J Cardiol 1966; 18:226-34
14 Parker MM, McCarthy KE, Ogibene FP, Parrillo JE. Right ventricular dysfunction and dilatation, similar to left ventricular changes, characterize the cardiac depression of septic shock in humans. Chest 1990; 97:126-31