tertiary centers with expertise in the management of PPH. This implies that in the hands of experienced clinicians appraised of optimal management strategies, hemodynamic evaluation and vasodilator therapy can be performed safely. However, the evolution of diagnostic strategies and effective therapies for PPH is rapidly changing. Until a consensus about evaluation and treatment is established by experts, it would be wise for patients with PPH to be managed at tertiary centers with extensive experience with this condition.

In summary, it is reasonable, perhaps even necessary, for the leaders in the field of PPH research to continue to work towards establishing a consensus regarding the basic methodologies and treatments used in the management of the disease. This will facilitate the effective design of future multicenter trials. Only when the results of such trials emerge can guidelines be established that may allow treatment by more than a limited number of experts in the field. In this way, the goal of establishing the safe, efficacious, and widely applicable treatment for PPH will be realized.

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REFERENCES

In Defense of the Stethoscope

My comments relate to bedside skills with a stethoscope in the 1990s in general and specifically to the article by Lok and colleagues in this issue of CHEST (see page 1283).

Anyone adept at auscultation will attest to the sorry state of the skill among students, house staff, and even young physicians (including cardiologists) who were trained in the past 20 years. Ironically, these years represent an exciting and unprecedented interval in cardiologic diagnosis and treatment, during which a variety of noninvasive technologies and invasive procedures were introduced.

I believe that at least three major factors contributed to the downgrading of bedside skills in auscultation of the heart. All had their origins in the late 1970s. First, on the recommendation of a committee on medical appropriateness in 1977, Blue Shield plans discontinued routine payment for phonocardiography, the graphic recording of heart sound and murmurs. Other insurance carriers and Medicare rapidly followed suit. Clearly, there had been instances of misuse of phonocardiography, but the net result of the action might have been expected. In our American culture, a test unworthy of reimbursement was abandoned in clinical practice and, unfortunately, in the training of young physicians as well. Publications on the value of auscultation have virtually disappeared from the medical literature, which reflects the lack of utilization of phonocardiography. Physicians previously trained in phonocardiography knew how to use a stethoscope.

Second, and more important, echocardiography, which had been applied clinically since the early 1960s,2 captured the imagination of physicians in the 1970s with reports of the clinical application of two-dimensional real-time and Doppler imaging of the heart.3 Echocardiography had the advantage of greater precision and objectivity and allowed graphic displays of anatomic detail and function. Unfortunately, diagnostic capabilities that are largely undeserved, for example, accurate quantification of valvular regurgitation, have been attributed to echocardiography. Diagnosis of several conditions, including congestive heart failure (CHF), mitral valve prolapse, constrictive pericarditis, cardiac tam-
ponade, acute pulmonary hypertension, innocent murmurs, and angina, may be best suspected by bedside examination.3

Third, elimination of bedside examination of patients as a hurdle in credentialing candidates for certification by the American Board of Internal Medicine has, in my judgment, contributed to the demise of skills in auscultation.

For these reasons and others, such as the vast difference in financial compensation for performing a procedure, as compared with performing a history and physical examination, skills in auscultation have waned.

Craig5 questioned whether auscultation should “be reserved for the occupational therapy of dwindling coteries of antiquarians, or ... promoted more vigorously as a viable part of our diagnostic armamentarium.” Those of us who learned bedside skills in the 1950s and 1960s and developed and maintained these skills in parallel with the growth of high-tech diagnostic procedures clearly represent a dwindling coterie. We have a responsibility to share our experience with younger practitioners who may have no basis for comparison of bedside examination with diagnostic tests.

Lok and colleagues conclude that agreement between observers and a phonocardiographic gold standard in identifying S3 and S4 gallops is poor. They confirm the appalling auscultation skills of trainees recently reported.6

Lumping S3 with S4 detection, however, is probably a mistake. Recordability of an S4 does not mean that it is audible.7 A phonocardiographic preamplifier with good filtering characteristics in the 25- to 50-Hz range records presystolic vibrations in most persons with or without heart disease. These vibrations result from left atrial filling of the ventricle in ventricular presystole. An S4 is a low-frequency sound and is difficult to hear. One hears the weaker overtones or harmonics. The usual mistake at the bedside is to confuse an audibly split S1, which is very common, for an S4-S1, which is uncommon. Phonocardiographically, this may be explained in part by mistaking detection of presystolic vibrations for audible sounds.

There is precedent for mistaking recordability of an S4 with audibility.7 Reectra and colleagues concluded that an S4 is common in otherwise normal adults. A better standard will be the finding of a palpable S4 gallop equivalent—a presystolic outward displacement of the chest wall superimposed on the early outward movement of the apical heave with the patient in a lateral decubitus position. Admittedly, this may be impossible in a patient with emphysema or a thick chest wall. A palpable S4 is a valuable clue to suspecting a stiff left ventricle secondary to hypertrophy or fibrosis, not a disease per se. It is positive in diastolic heart failure. An echocardiogram shows normal systolic function. For this reason, every patient with heart failure deserves at least a baseline echocardiogram. The relatively high negative predictive value for S4 detection (82%) noted by Lok and colleagues may have resulted from the generally acknowledged difficulty in hearing a soft, low-frequency S4, a possible small number of patients with an S4 in the study group, or the relatively infrequent phonocardiographic recording of an absence of presystolic vibrations.

Phonocardiography should be much more reliable in identifying an S3 gallop. False positives are uncommon. Indeed, the mean positive predictive value reported by Lok and colleagues was 71% (50 to 88%). Auscultation of an S3 in over 70% of patients with a recorded S3 shows that auscultation can be valuable as diagnostic or confirmatory evidence of CHF, a life-threatening condition. CHF is the leading cause of hospitalization and hospital cost in the elderly. We should not abandon a finding that is cost effective. Accurate bedside auscultation is clearly cost effective. Echocardiography will diagnose systolic dysfunction, but not CHF.

In summary, the study is flawed in design by the use of phonocardiography as the gold standard. Perhaps a truly well-designed study cannot be done. The number of patients is small. Reliance on statistical methods to compensate for inadequate numbers may result in false conclusions.

To revive interest and skills in auscultation will require experienced teachers. The best teachers currently are senior cardiologists trained in phonocardiography. The ages and specific training and experience of the staff who participated in the study would therefore be of interest.

Do I have suggestions? Chiefs of medicine must appreciate the scope of the problem and arrange regular auscultation rounds with senior cardiologists. Excellent CD-ROM computer-based programs have recently become available for home study and reinforcement. Testing of pattern recognition with recordings should be included in board examination as a special hurdle. We must remedy the situation before it is too late. When the senior teachers are gone, who will then teach? Will the blind then lead the blind?

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Integration of Catheter Thrombectomy Into Our Armamentarium To Treat Acute Pulmonary Embolism

Successful management of acute pulmonary embolism (PE) combines prompt risk stratification and decisive, early intervention. In the past, a normal BP and heart rate too often engendered a sense of complacency. Typically, clinically undetected right heart failure would worsen, cause pressor dependence, and lead to unremitting cardiogenic shock. At this point, as patients suffered rapid overt deterioration, desperate clinicians would consider thrombolysis or surgical embolectomy as a last resort, often with poor results.

Fortunately, we now recognize that right ventricular function is a critical determinant of prognosis.1,2 Moderate or severe right ventricular dysfunction is correlated with recurrent PE (including death from recurrent PE) despite adequate anticoagulation.3,4

After high-risk PE patients are identified, efforts should be redoubled to ensure adequate anticoagulation with heparin, generally requiring a partial thromboplastin time of at least 60 s. Then, patients should be screened to determine their suitability for pharmacologic thrombolysis.

Pharmacologic thrombolysis offers the opportunity to remove clot without the morbidity, risk, and expense of open surgical embolectomy. It also permits dissolution of thrombus in the distal pulmonary arterial tree (and in the pelvic and deep leg veins) that is not accessible to the surgeon. However, the same comorbid conditions that make patients poor thrombolysis candidates may also deem them unsuit-

able for an open surgical approach. Fortunately, for high-risk PE patients, contemporary catheter thrombectomy may be employed, especially when neither pharmacologic thrombolysis nor open surgical embolectomy is suitable.

Medical clot-debulking yields rapid improvement in pulmonary perfusion and right ventricular function,3 at a cost of increased bleeding, including the possibility of intracranial hemorrhage.5 In addition to standard contraindications such as stroke, trauma, or GI tract bleeding, additional risk factors that increase bleeding include older age, obesity, and pulmonary artery catheterization via the femoral vein.6 At least half of high-risk PE patients will be relatively unsuitable candidates for thrombolysis.

When pharmacologic thrombolysis is inadvisable, mechanical intervention should be considered. Inferior vena cava filters used to be the principal intervention that was offered; now, they are less often employed than previously for several reasons. First, they do not in any way treat the clot that already exists in the pulmonary arteries or deep leg veins. Second, they have not been shown to be superior to intensive anticoagulation among patients with “free floating” venous thrombus.7,8 Third, a recently published randomized controlled trial concluded that vena caval filters conferred no survival benefit compared with anticoagulation after 2 years of follow-up. Furthermore, those patients who had received filters actually suffered a higher rate of deep vein thrombosis over the ensuing 2 years compared with those treated just with anticoagulation.9 Nevertheless, patients with massive PE who are not candidates for thrombolysis often merit placement of inferior vena caval filters, especially if residual pelvic or leg deep venous thrombosis is detected.

Table 1—Contemporary Catheter Thrombectomy

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<th>Techniques</th>
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<tr>
<td>Fragmentation thrombectomy</td>
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<td>Pigtail (bimanual) rotation catheter</td>
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<tr>
<td>“Clot Buster” Amplatz Thrombectomy Device (Microvena Corp; White Bear Lake, MN)—8F catheter with enclosed impeller, driven at 150,000 rpm by an air turbine</td>
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<td>Rheolytic thrombectomy</td>
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<td>Angiojet Rapid Thrombectomy System (Possis Medical Inc; Minneapolis, MN)—a high-velocity saline solution jet from a dedicated expensive drive unit creates a strong Venturi effect at the tip of a 5F catheter</td>
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<tr>
<td>Hydrolyser-Cordis Thrombectomy Catheter (Cordis; Miami Lakes, FL)—uses a standard contrast medium-power injection to create a saline solution jet</td>
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<td>Aspiration thrombectomy</td>
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<td>Meyerovitz technique: 8F or 9F coronary guiding catheter (without sideholes) is placed through a 10F Arrow sheath; aspirate with a 60-mL syringe</td>
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<td>Greenfield embolectomy catheter</td>
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References
6 Mangione S, Nieman LZ. Cardiac auscultatory skills of internal medicine and family practice trainees: a comparison of diagnostic proficiency. JAMA 1997; 278:717-722