Auscultation and Don Quixote

To the Editor:

The well-conceived and nicely presented article by Barrett et al1 (August 2004) begs the question: are we merely jousting at windmills, as did the “Man from La Mancha”? Its references need not be repeated, are well established, and have been confirmed by many philosophic and scientific articles regarding the notorious and decremental decline in the teaching and acquisition of physical diagnostic skills with special reference to those of auscultation. In this scenario, we confront the continued debate regarding the value of cardiac auscultation (I suspect pulmonary auscultation fares no better).

In spite of the never-ending development of technologic diagnostic modalities, it nevertheless remains unquestioned by any master clinician that the physical examination (accompanied by an adequate history) still remains the solid keystone in our diagnostic armamentarium. The problem is, however, that its “decline and fall” has not been adequately remedied in spite of what is known, has become more obvious, and continues on the path of destruction and obsolescence. In 1950, Paul Wood2 stated, “There is already plenty of evidence to show that we are in danger of losing our clinical heritage, and of pinning too much faith in figures thrown up by machines. Medicine must suffer if this tendency is not checked.”

The incomparable Sir James Mackenzie (1853–1925) constantly feared that the combination of technology and instruments would divorce the cardiologist from looking at the whole person, replacing powers of observation and reasoning.3 The late George Burch,4 Fontex Maximus of cardiologists, researchers, and teachers (as was usual) in his presidential message to the American College of Cardiology stated,

“With the decreasing relative abundance of physicians and the increasing interest in gadgets, computers and dramatic procedures, the care of the average patient is apparently not improving in quality and elegance . . . a careful and meticulous clinical study would provide more, if not all, necessary information for diagnosis, treatment and understanding of the pathophysiologic disturbances. There is a tendency to rely more and more on complex procedures when simple ones done well are not only sufficient but necessary for careful and complete care of the patient.”

Those are the fortunate physicians who recall the pleasure of observing the cardiologists trained by Harvey and Perloff as they methodically and meticulously conducted their cardiac examination of the patient with perfection (like Toscanini conducting a Puccini opera) as they proceed through the four basic steps of their quest (not the impossible dreams of Don Quixote): inspection, palpation, percussion, and auscultation. Over the past 20 years, those pleasures have become almost nonexistent. A typical daily example recorded on the chart by attendings and consultants regarding the presence of a mitral insufficiency (regurgitation) murmur: “heart sounds normal, soft systolic murmur,” or slightly better—“heart tones normal, grade 2 systolic murmur at apex.” Shortcuts often occur, but the proper response should be as follows: the rhythm is normal sinus, first and second heart sounds are normal, with no abnormal splitting, no premature beats or gallops, a grade 2/6 soft, blowing, long (if not holosystolic), high-pitched systolic murmur, heard maximally at the apex, with slight (or no) increase in the left lateral position, with faint radiation toward the pulmonic area and heard faintly in the epigastrium (if such was the case). A murmur has intensity, duration, frequency, quality, configuration, timing, and radiation. Inspection, palpation, and percussion are either dying or dead. A properly performed cardiac physical examination can lead one to varying diagnoses: valvular heart disease, nonvalvular obstructive disease, congenital heart disease, ischemic heart disease, pericardial disease, various cardiac arrhythmias, cardiac decompensation, peripheral and central thromboembolic disorders, and offer clues to some metabolic and infiltrative disorders. Various reasons (or excuses) have been offered for the decline in cardiac auscultation and examination. Foremost among them are the following: the overlecturing and underteaching of clinical medicine, the displacement of clinical skills by technology, the lack of structured teaching of cardiac examination in most residency programs, the lack of teaching of physical examination skills in the training of medical students, the absence of third party

References

payment for phonocardiography, and lastly but certainly not least, the decline in the presence of senior teachers and master clinicians.7–9 As stated by Adolph,10 “We must remedy the situation before it is too late. When the senior teachers are gone, who will then teach?” In an excellent review on this subject, Craig11 stated, “The present trend toward the denigration of auscultation may soon leave us with a whole new generation of young physicians who have no confidence in their own ability to make worthwhile bedside diagnoses.” Unfortunately, his prophetic wisdom has come to pass. Cardiac auscultation is an art and a science. This is one of the few pleasures that can be derived from the ever-changing practice of medicine. It stimulates acquisition of a good doctor-patient relationship and provides a satisfying alternative to many of the idiopathic inconsistencies and inadequacies of medical practice. It is unquestionably an art and skill that must not be allowed to succumb to the way of the impossible dream of Don Quixote.

Basil M. Rudusky, MD, FCCP
Wilkes-Barre, PA

References
5 Mangione S, Nieman LZ. Cardiac ausculatory skills of internal medicine and family practice trainees. JAMA 1997; 278:717–722
6 Roldan CA, Crawford MH. How valuable is the physical examination for detecting valvular heart disease? Cardiol Rev 1997; 14:51–54
7 Hurst JW. The overculturing and underteaching of clinical medicine. Arch Intern Med 2004; 164:1605–1608
8 Tavel ME. Cardiac auscultation: a glorious past; but does it have a future? Circulation 1996; 93:1250–1253
9 Hurst JW. I view with alarm (the fall of 1999). Am J Cardiol 1999; 84:1339–1340

Epoprostenol and Nesiritide in Pulmonary Hypertension

To the Editor:

The case report by Kurian et al1 highlights several important misconceptions regarding the management of pulmonary hypertension that warrant attention. Firstly, one cannot address the optimal approach to therapy of pulmonary hypertension without first delineating its etiology. In the case presented, the disease process was postcapillary in origin, and therefore should have been treated as such. Although the authors refer to epoprostenol (prostacyclin) as “traditional therapy,” this drug is approved by the Food and Drug Administration for the treatment of patients with pulmonary arterial hypertension (PAH) who fall in to New York Heart Association functional classes III or IV; indeed, epoprostenol therapy is considered to be contraindicated in patients with pulmonary venous hypertension due to left-sided heart disease.2 At best, this would have to be considered off-label use that carries considerable risk without evidence of benefit for this condition.

Whether nesiritide may offer benefit in patients with PAH will require carefully performed clinical trials in a well-defined population using established criteria for disease definition. The current management of patients with PAH is presented comprehensively in the American College of Chest Physicians evidence-based guidelines, which accompanied the July issue of CHEST.3

Lewis J. Rubin, MD, FCCP
La Jolla, CA

The Importance of Clinical Registries for Pulmonary Epithelioid Hemangioendothelioma

To the Editor:

We read with great interest the article by Cronin et al1 in CHEST (February 2004) entitled “Pulmonary Epithelioid Hemangioendothelioma: An Unusual Case and Review of the Literature.” In their article, the authors commented on several aspects of pulmonary epithelioid hemangioendothelioma, including the rarity of epithelioid hemangioendothelioma (EHE) to the lung, the ability to make this diagnosis by transbronchial biopsy, the fact that EHE can affect various organs, the varied prognosis for patients with EHE, the varied response with chemotherapy, and, notably, the fact that some patients have shown full regression of EHE without any treatment. We agree with the authors that the rarity of this condition, the lack of clear standards for treatment, and the partial-to-complete spontaneous regression of EHE seen in some patients up to 15 years from initial detection makes it difficult to decide on the most appropriate treatment.2 However, we do not necessarily think that the involvement of other organs such as the liver translates into poor survival, as commented on by the authors. Long-term follow-up of ≥ 20 years has been reported in many cases of EHE involving both the lung and liver, with a survival time of up to 27 years reported.2

Partial hepatectomy and liver transplantation for diffuse involvement is now a standard treatment for localized and diffuse involvement of the liver with EHE. Marino et al2 reported a 76% 5-year survival rate for 10 patients with EHE involving the liver who underwent liver transplantation. Five patients who received transplants were free of metastatic disease, although two patients who underwent liver transplantation for diffuse involvement were free of metastatic disease, although two patients who underwent liver transplantation for diffuse involvement were free of metastatic disease.

Lewis J. Rubin, MD, FCCP
La Jolla, CA

References
1 Kurian DC, Wagner IJ, Klapholz M. Nesiritide in pulmonary hypertension. Chest 2004; 126:302–305
3 Diagnosis and management of pulmonary arterial hypertension: ACCP evidence-based clinical practice guidelines. Chest 2004; 126(Suppl):1S–92S

The Importance of Clinical Registries for Pulmonary Epithelioid Hemangioendothelioma

To the Editor:

We read with great interest the article by Cronin et al1 in CHEST (February 2004) entitled “Pulmonary Epithelioid Hemangioendothelioma: An Unusual Case and Review of the Literature.” In their article, the authors commented on several aspects of pulmonary epithelioid hemangioendothelioma, including the rarity of epithelioid hemangioendothelioma (EHE) to the lung, the ability to make this diagnosis by transbronchial biopsy, the fact that EHE can affect various organs, the varied prognosis for patients with EHE, the varied response with chemotherapy, and, notably, the fact that some patients have shown full regression of EHE without any treatment. We agree with the authors that the rarity of this condition, the lack of clear standards for treatment, and the partial-to-complete spontaneous regression of EHE seen in some patients up to 15 years from initial detection makes it difficult to decide on the most appropriate treatment.2 However, we do not necessarily think that the involvement of other organs such as the liver translates into poor survival, as commented on by the authors. Long-term follow-up of ≥ 20 years has been reported in many cases of EHE involving both the lung and liver, with a survival time of up to 27 years reported.2

Partial hepatectomy and liver transplantation for diffuse involvement is now a standard treatment for localized and diffuse involvement of the liver with EHE. Marino et al2 reported a 76% 5-year survival rate for 10 patients with EHE involving the liver who underwent liver transplantation. Five patients who received transplants were free of metastatic disease, although two patients who underwent liver transplantation were free of metastatic disease.

Lewis J. Rubin, MD, FCCP
La Jolla, CA

References
1 Kurian DC, Wagner IJ, Klapholz M. Nesiritide in pulmonary hypertension. Chest 2004; 126:302–305
3 Diagnosis and management of pulmonary arterial hypertension: ACCP evidence-based clinical practice guidelines. Chest 2004; 126(Suppl):1S–92S