The Miracle of Echocardiography: A Clinician's Retroprospective

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I am honored to be here today and to have been selected to present the Seventh Annual Inge Edler Lecture. As you know, this lecture commemorates the clinical pioneer whose seminal work has blossomed into the comprehensive diagnostic technique that echocardiography is today. Dr. Edler's investigations antedate by a decade the era and subsequent events that I will describe in greater detail—the 1960s—the time when echocardiography made its initial appearance on the American scene and the role that it has since played in the evolution of the practice of cardiology during the past four decades.

One of my favorite sayings attributable to the British cleric John Henry Newman is, "To live is to change, and to be perfect is to have changed many times." The transition from the 1960s to the 1990s has been one of monumental change in the history of the understanding, diagnosis, and treatment of heart disease. Much of what we take for granted today was hardly established when I entered medical school at the beginning of this era. For instance, as with the very recently settled debates with regard to treatment for hypercholesterolemia, it was then seriously debated whether asymptomatic hypertension should be treated. It was argued whether coronary thrombosis was the cause or the result of acute myocardial infarction. Although penicillin had made significant inroads into the prevention of acute rheumatic fever, it was unclear that this would have an impact on the ultimate disappearance of rheumatic heart disease. Valve-replacement surgery was in its infancy, implantable cardiac pacemakers were nonexistent, and no one spoke of coronary bypass.

Coinciding with our improved understanding of heart disease and therapeutic interventions that we apply to it has been the array of new diagnostic techniques that have emerged concurrently, and echocardiography prevails as the most widely used, most versatile, and least costly of them all. Indeed, echocardiography has played a preeminent and spectacular role in the evolution of cardiac imaging techniques, and I believe it is deserving of the designation *miracle* in the sense that it describes such an amazing contribution.

For you to appreciate further how important the contribution of echocardiography has been to cardiac diagnosis, it is useful to explore the status of cardiology in the 1960s, when echocardiography was first used in the United States, to the present era, for not only have diagnostic techniques changed importantly but so have our concepts of pathophysiology, our patients, their diseases, medical and surgical treatments, the very manner in which we practice medicine, and the outcomes of our ministrations and interventions (Table 1).

The 1960s embrace the time span that occurred two decades after the advent and subsequent widespread use of penicillin. As a result, acute rheumatic carditis, the illness that filled specialized hospitals dedicated to its treatment with children and adolescents in the decades before, was dramatically on the wane. However, rheumatic heart disease, the long-term consequence of acute rheumatic fever that took such a toll in the prepenicillin era, continued to be a prevalent cause of heart failure. To be sure, acute coronary syndromes and nonvalvular heart failure were ever present, but heart failure as a result of rheumatic heart disease was the principal reason for admission of the youngest adults to an acute general hospital. The underlying lesion in all cases was some degree of mitral stenosis. A typical scenario would be a young woman in the prime of life, mid twenties to late thirties, previously with minimal or mild symptoms, having her first episode of atrial fibrillation, and this precipitated a life-threatening bout of pulmonary edema. Another common scenario was a person of similar age with a young family having an incapacitating stroke. Contrast this to the 1990s, when acute coronary disease dominates the reasons for cardiac admission, whereas rheumatic heart disease in most areas of

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TABLE 1 Hospital practice of cardiology: characteristics of the early 1960s

Practice	Characteristic
Pathophysiology	Earlier and less refined concepts
Patients	Many less than age 50 years
Diseases	Rheumatic valvular disease was prominent cause of CHF
Care delivery	Master clinician was role model
Noninvasive diagnostic methods	ECG, vectorcardiograms, Master's test, cardiac series, carotid and jugular pulse tracings, apexcardiograms, systolic time intervals, ballistocardiograms
Cardiac catheterization	Hemodynamic assessments, contrast and coronary angiography introduced
Treatments	
Medical	CHF: mercurial diuretics, multiple digitalis preparations Acute MI: bed rest, 3 wk hospitalization Arrhythmias: quinidine, procainamide, digitalis
Surgical	Mitral commissurotomy, early phases of valve replacement CAD, internal mammary implant operation Bradyarrhythmias, permanent pacing introduced late in decade
Outcomes	Modest palliation, heart disease usually limited life expectancy

CHF, Congestive heart failure; *ECG,* electrocardiogram; *MI,* myocardial infarction; *CAD,* coronary artery disease.

the United States has nearly disappeared as a cause for hospitalization.

Since the 1960s, not only have the entities comprising the spectrum of heart diseases changed in prevalence, but the patients we treat have also changed dramatically. The age range for the limited interventions that we performed then on adults (i.e., mitral commissurotomy and valve-replacement surgery) was approximately 30 to 50 years. Contrast this to the dominant decade for cardiac intervention in my hospital, which is now 70 to 80 years, and the wide range of procedures dominated by coronary bypass and angioplasty. Thus along with the passage of a 30-year span, the patients we now treat are on the average three decades older than they were in the 1960s.

Health-care delivery has also changed dramatically during this time. The 1960s was a time when medical education continued to feature a trend that had been prevalent for nearly a century: the master clinician as a role model. This was an individual whose clinical acumen, unsurpassed expertise in physical diagnosis, and impeccable judgment played a central role in health-care delivery. Their names are legendary, and every teaching institution had at least one of them. Contrast this to the 1990s—the age of the health care team in which the physician functions with other physicians, surgeons, physician assistants, nurse practitioners, nurses, technologists from many disciplines, dieticians, social workers, and home care coordinators to deliver rapid diagnosis and effective and continuing therapy. In contrast to the 1960s, when the opinion of the master clinician was the dominant arbiter of therapeutic strategy, in the 1990s the results of investigation produced by instrumentation, particularly imaging techniques, provide the evidentiary basis for medical decision making.

Decades ago we provided poor to modest palliation of heart diseases, most of which limited life expectancy when a serious problem was diagnosed. Today we can offer improved quality of life to patients with many forms of heart disease; for a great number of them, although they live with heart disease, their life expectancy is not limited by it. Thus it is commonplace for many patients with coronary or valvular heart disease to undergo angioplasty or bypass or valve-replacement surgery only to die years later of metastatic cancer or Alzheimer's disease.

In pursuing cardiac diagnosis in the 1960s, the catheterization laboratory was the key place for definitive diagnosis of almost all forms of heart disease. In the 1990s catheterization laboratories, although playing a diagnostic role in determining the severity and distribution of coronary disease, have increasingly become areas of therapy for interventional cardiologists and electrophysiologists.

With regard to noninvasive cardiac diagnosis in the 1960s, the electrocardiogram was our most valuable, and arguably it can now be claimed our only valuable, noninvasive diagnostic method. As a result, the electrocardiogram was universally applied in attempts to establish all forms of anatomic diagnosis. Much electrocardiographic diagnosis, particularly outside the realm of acute coronary disease, however, was highly

inferential and often reduced to truisms such as, the patient with cardiomyopathy rarely has a normal electrocardiogram, a patient with dominant mitral stenosis will not have left ventricular hypertrophy on the electrocardiogram, or the patient with severe aortic stenosis should have left ventricular hypertrophy on the electrocardiogram.

From this impressionistic overview comparing present-day cardiology to that of the 1960s, let us take a closer look at the dominant heart problems we then saw in hospitalized adults. Most were consequences of valvular heart disease, acute coronary disease, and nonvalvular heart failure. In that era, valvular heart disease was dominantly rheumatic, and mitral stenosis played the central role in this process. The patients who were most fortunate had exclusively or "pure" mitral stenosis, whereas the less fortunate had mitral stenosis complicated by mitral regurgitation or involvement of other valves, common combinations being mitral and aortic, mitral and tricuspid, or sometimes all three. For more than a decade, mitral commissurotomy had been shown to be an effective palliation for pure mitral stenosis (i.e., those patients with stenotic noncalcified mitral valves who had little or no mitral regurgitation and no other involvement of cardiac valves). Indeed it was this scenario that spawned the birth of echocardiography in Inge Edler's clinic. The challenge was twofold: both overestimating the importance of any accompanying mitral regurgitation and on occasion failing to recognize that it was present and predominant.[1] By the 1960s, artificial valves were coming into play, but valve-replacement surgery was offered to patients with New York Heart Association class III-IV heart disease. Mortality rates were high, often exceeding 10% to 20%; early mechanical prostheses had flaws and thromboembolism, poppet problems such as ball variance, valvular obstruction, and prosthesis dehiscence wreaked havoc by producing substantial morbidity and mortality rates after initially successful operation. Indeed we also learned to our regret that patients often survived with a properly functioning prosthesis but with no improved quality of life compared with their preoperative state; class IV and advanced class III heart failure were just too late for many patients to reap the benefit of value-replacement surgery.

Patients with heart failure, particularly those who did not have valvular disease, were doomed to an uncomfortable, as well as a rapidly inexorable, downhill course. Among other measures, acute heart failure was treated with phlebotomy and rotating tourniquets. Anasarca and even jaundice caused by chronic heart failure were frequently encountered. Mercurials were the best of our diuretics, and the response to these agents often lived up to the name. Indeed the most gratifying immediate responses to treating pulmonary edema were often in that prototypical young woman with mitral stenosis I described previously: going to an emergency room with life-threatening pulmonary edema and heart rates of 160 to 170 beats/min produced by a first bout of atrial fibrillation. The diagnosis, if not known previously, was usually ascertained from the history or appearance of the heart on chest x-ray film, because in such an acutely ill patient (gasping for breath) it was impossible to hear the characteristic diastolic rumble at such rapid heart rates. In these patients the repeated administration of intravenous digitalis glycosides over a period of 6 to 12 hours produced considerable slowing of the ventricular rate with extremely gratifying results. After appropriate control of heart rate and diuresis, such patients were often restored to their preatrial fibrillation cardiac class. In contrast, as their disease progressed, most patients with other forms of heart failure, both valvular and nonvalvular, often became refractory to mercurial diuretics and, if they were not a candidate for heart operation, died of pulmonary edema or an arrhythmia contributed to by anoxemia and acidosis resulting from advanced pulmonary congestion or poor cardiac output. The distinction between diastolic and systolic dysfunction had not transpired. Digitalis glycosides in many forms (digitalis leaf, digitoxin, digoxin, lanatoside C, ouabain, and acetylstrophanthidin) were used liberally, although perhaps not as indiscriminately as in prior times. Nevertheless, pushing digitalis glycosides to treat advanced heart failure frequently led to cardiotoxic arrhythmias, and this form of iatrogenic heart disease (i.e., digitalis intoxication) accounted for 10% to 15% of admissions for heart disease in many institutions.

Although the public was clearly much less attuned to reporting immediately to emergency rooms for chest pain syndromes, acute coronary disease, particularly myocardial infarction, was indeed diagnosed readily by clinicians. Once recognized, however, the physician virtually took a bystander role in treating this disease as the patient spent 3 weeks in the hospital, most of the first 2 weeks in bed recovering from what was for many working men a career-ending illness.

Our armamentarium to treat the patient with tachyarrhythmias consisted of digitalis, quinidine, and procainamide. Severe bradyarrhythmias, particularly complete heart block, were highly problematic. Patients hospitalized with Stokes-Adams syncope were treated with external pacemakers; those who

Journal of the American Society of Echocardiography
March 1997

were unfortunate enough to have transthoracic pacing from external electrodes, if they did not die after 24 hours of this treatment, surely begged to do so.

It is also pertinent to relook at the 1960s to examine further the diagnostic armamentarium that we had at that time. In that era, great premium was placed on the medical history and the physical examination. The four components of physical examination (inspection, palpation, percussion, and auscultation) were nowhere better applied than to the cardiac examination, and a plethora of eponyms was applied to every aspect of cardiac physical diagnosis, reflecting the abiding reverence to the master clinician. Auscultation, in particular, was held in the highest regard, and one could not expect to live up to the role model (the master clinician) unless one was also an accomplished auscultator. The chest x-ray film was expanded into a cardiac series—an examination almost always performed on patients who had heart murmurs or enlarged hearts. With a cardiac series, one outlined the esophagus with barium and right and left oblique films of the chest were taken along with the standard posteroanterior and lateral films. This facilitated differential evaluation of chamber enlargement, from which one was expected to deduce the dominant lesion or lesions in patients who had murmurs. Cardiac fluoroscopy was often an adjunct to this examination. Beyond the resting electrocardiogram, we had the Master's two-step test to evaluate patients suspected of having coronary artery disease.[2] This was an important conceptual advance in that the postexercise electrocardiogram would prove to be highly valuable in diagnosing exercise-induced myocardial ischemia. Nevertheless, it was a clumsy affair in which the patient, with electrodes attached, climbed over a pair of stairs and then had his or her electrocardiogram recorded after a prescribed number of trips. From postexercise variations in ST segments, one inferred the likelihood of ischemic heart disease.

In this era, surface recordings of heart sounds and pulse waveforms achieved a peak in popularity. Phonocardiograms, apex cardiograms, jugular and carotid pulse tracings, and systolic intervals were used to provide inferential and often weak information about underlying heart disease. Those most deeply committed to in-depth noninvasive diagnosis studied vectorcardiograms and ballistocardiograms in the hope of ascertaining incremental diagnostically important information not available with other techniques. In short, any time something depolarized, pulsated, or quivered as a result of cardiac activity, we were there to measure it.

This decade also witnessed the birth of nuclear cardiology. The latter technique was initially applied to trace blood flow through the heart and to ascertain abnormal flow patterns, particularly as occurred with intracardiac shunts. Moreover, the nuclear technique proved valuable for large organ perfusion and soon evolved to be particularly useful as a screening test for pulmonary embolic disease. Overall, however, the 1960s was an era of diagnostic difficulty unless you took the patient to the catheterization laboratory. Even then, if we did establish a definitive diagnosis with hemodynamic measurements or angiography, we had little to offer therapeutically outside of the realm of mitral commissurotomy or valve replacement with a problematic prosthesis.

Cardiac catheterization and angiography were the accepted and ultimate standards to make a definitive diagnosis of valvular or congenital heart disease, as well as to define coronary anatomy. Coronary angiography, however, had its greatest value in identifying patients who did not have the disease, because if they had obstructive coronary disease, little could be offered in the way of effective revascularization. Although the internal mammary implant operation was popular by the middle of the 1960s, it was not until the end of this decade that coronary bypass procedures started to take place.

Another even more difficult diagnostic problem was pericardial effusion. It was often suspected from the globular appearance of the heart on chest x-ray filming and the diagnosis was confirmed by either contrast angiography or a diagnostic pericardiocentesis, the latter often fraught with problems of cardiac penetration or coronary artery laceration from the pericardial needle.

In the midst of this scene of diagnostic difficulty, echocardiography came about as an exquisitely sensitive technique to diagnose pericardial effusion. One could suspect a pericardial effusion from the globular shape of the heart, but confirmation required invasive intervention with either catheter and contrast techniques or an empiric diagnostic pericardiocentesis. The landmark contribution of Feigenbaum et al.[3] demonstrating echocardiographic detection of pericardial effusion (Figure 1) has become a historic starting point for progressive contributions by a multitude of investigators that has characterized echocardiography during the succeeding three decades of its application for cardiac diagnosis on the American scene. These earliest observations in pericardial effusion clearly established that the diagnosis of pericardial effusion could be made safely, without invasive intervention, and, in addition, could be used to guide pericardiocentesis, significantly lessening the risk of heart penetration and coronary artery lacerations.

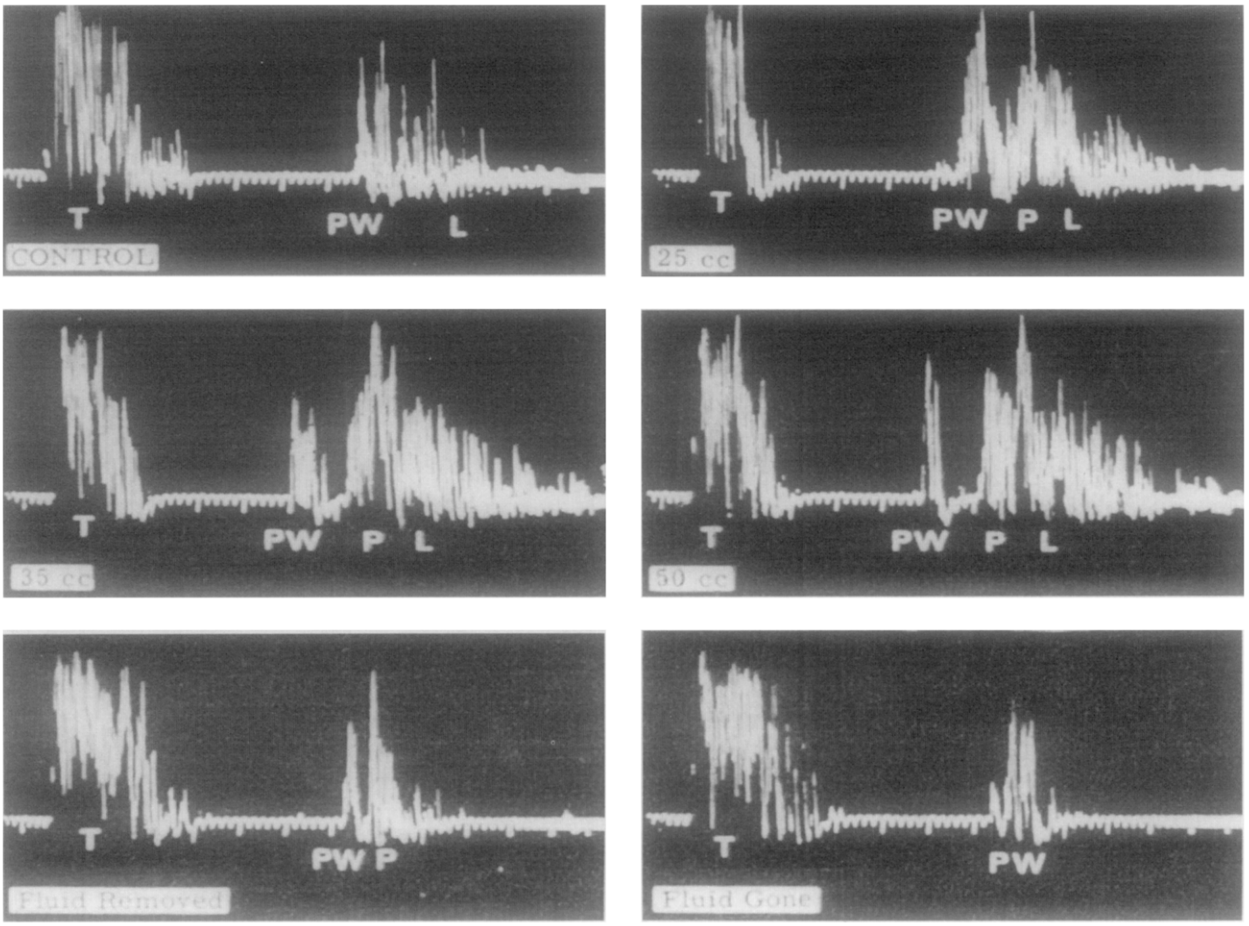

Figure 1 Canine experiment shows effect of injection of saline solution into pericardial sac on A-mode echocardiogram. With progressive instillation of fluid, there is separation of signals of the posterior wall *(PW)* of heart from posterior pericardium *(P),* which is anterior to lung *(L)* echoes. Note that posterior wall and pericardium fuse into single signal as fluid is removed. (*T,* Transducer signal.) (From Feigenbaum H, et al. Ultrasound diagnosis of pericardial effusion. JAMA 1965;191:9;109.

TABLE 2 Highlights of the historic role of echocardiography in pericardial effusion and cardiac tamponade

Echocardiography	Role	Year
M-mode	Detection of posterior pericardial effusion	1965
	Physical basis for electrical alternans	1966
	Elucidation of physiologic basis of pulsus paradoxus	1975
Two-dimensional	RV compression in tamponade	1977
	RV diastolic collapse and RA inversion as more subtle signs of impending tamponade	1983
	IVC plethora as a sensitive sign of tamponade	1988
Doppler	Reciprocal respiratory flow variations across mitral and tricuspid valves; altered IVC flow in tamponade	1988
Transesophageal	More refined detection of atrial tamponade	1990s

RV, Right ventricular; *RA,* right atrial; *IVC,* inferior vena caval.

With pericardial effusion used as an example, let us now move forward through the ensuing years to see the depth of understanding that echocardiography has unveiled about this problem (Table 2).

Further observations of cardiac motion with large pericardial effusions unveiled the underlying pathophysiology of electrical alternans,[4] long regarded as an electrocardiographic sign of likely pericardial effu-

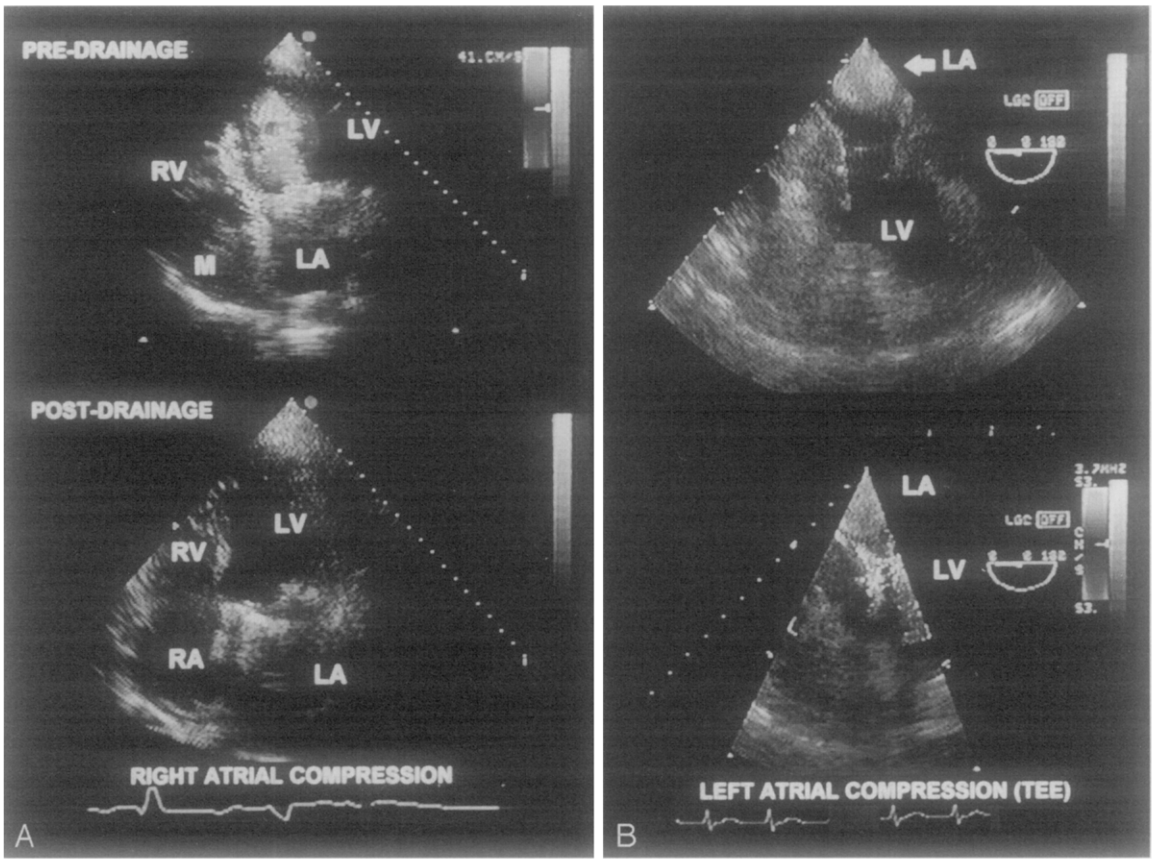

Figure 2 Atrial tamponade in two different hypotensive patients after surgery. **A,** Isolated right atrial tamponade *(top)*. **B,** Isolated left atrial tamponade. Note deviation of blood flow around obstructing extrinsically compressing masses (*M* and *arrow*) in color Doppler pictures. **A,** Effect of removing hematoma on reexpansion of right atrial cavity *(bottom)*. In both patients, evacuation of hematoma relieved hypotension and facilitated recovery. (*RV,* Right ventricle; *LV,* left ventricle; *RA,* right atrium; *LA,* left atrium.)

sion. Shortly thereafter we documented the importance of ventricular interaction as the basis for pulsus paradoxus[5]; during inspiration, the leftward septal shift underlay less left ventricular filling, producing a concurrent drop in systolic blood pressure. More understanding of the dynamics of pericardial effusion and tamponade became evident as two-dimensional echocardiography came into wider use in the late 1970s and early 1980s. Compression of the right ventricle by pericardial fluid was identified.[6] Thereafter, from inspection of heart wall motion, particularly the right ventricle and right atrium, we saw inappropriate inversion of external chamber surface contour[7,8] and repeatedly demonstrated that this finding was a useful sign of impending cardiac tamponade. In essence, these heart walls were acting as transducers, invaginating as a result of excess intrapericardial pressure as intracardiac pressures dropped transiently during the phases of the cardiac cycle. However, even more elucidation of the pathophysiology of impending pericardial tamponade was yet to come. With Doppler flow imaging, abnormal reciprocal flow was demonstrated across the mitral and tricuspid valves, flow decreasing with inspiration on the left side and increasing on the right side.[9] Concurrently, there is hepatic venous plethora with disappearance of inspiratory collapse and expiratory loss of antegrade blood flow in these structures.[10] Moreover, we have repeatedly been able to ascertain that tamponade, the stoppage of blood flow through the heart, can be caused by local collections of blood as may occur with penetrating chest trauma or after heart surgery. In the 1990s transesophageal echocardiography has added important refinement to this technique, because it is possible to detect cardiac chamber compression more readily with this approach than with the transthoracic approach early after surgery (Figure 2). Indeed the use of echocardiography after heart surgery for the

hypotensive patient with isolated atrial or ventricular tamponade caused by local accumulation of blood can be the first important life-saving step that leads the surgeon to relieve the compression by surgical reexploration and drainage.

So it was in the midst of the 1960s, a decade of groping for more precise noninvasive cardiac diagnosis, that echocardiography was born in America. The continuing evolution of change and refinement that I just described for the application of echocardiography to pericardial disease is really just a paradigm for all of the advances that echocardiography has brought to identification of most other forms of heart disease as well: valvular heart disease, where we can measure valve areas and pressure gradients precisely, coronary disease, where we can unmask risk areas and viable myocardium with stress interventions, cardiomyopathy in all of its forms, congenital abnormalities, both simple and complex, many of which can be diagnosed even before birth, and intracardiac masses, from the most subtle of valvular vegetations to the most exuberant of tumors.

What echocardiography has contributed during the past three decades is indeed a marvel to behold. In contrast to resting 12-lead electrocardiography, which derives information exactly as it did 30 years ago, echocardiography has continued to change, and indeed it has changed many times, in this period of three decades. The 1970s saw the advent of practical two-dimensional echocardiography. The 1980s saw the birth of fetal echocardiography and implementation of the Doppler techniques and Doppler color flow and, at the end of that decade, two-dimensional transesophageal echocardiography. Now in the 1990s we are seeing clinical application of myocardial contrast echocardiography, as well as intracoronary and intravascular applications, and promising new methodologic advances such as second harmonic imaging and better-than-ever three-dimensional reconstructions.

The incremental contribution of echocardiography to the status of 1960 cardiac diagnosis has been monumental. Clearly it has become a substitute for cardiac auscultation, at least on the part of internists and general practitioners. Rarely do I see a precise diagnosis of a heart murmur when the latter group of physicians refers a patient to the echocardiography laboratory; the usual referral says "murmur" or "systolic murmur" and the outcome of the study can show anything from a normal heart to complex structural lesions. Echocardiography has eliminated the use of the cardiac series and in most instances cardiac fluoroscopy. It has been well more than a decade since I can remember seeing a cardiac series on a patient. It is only now in the rarest of instances that we do cardiac fluoroscopy to the exclusion of other procedures in our cardiac catheterization laboratory. All cardiac graphics recordings—the phonocardiogram, apex cardiogram, pulse recordings, systolic intervals, vectorcardiograms, and ballistocardiograms—have become historic curiosities. I can now look back nostalgically on how valuable some of these techniques were, particularly apexcardiography, in sharpening my physical diagnostic acumen. I can also look back philosophically on them as testimony to the cadre of cardiologists who were committed to the notion that precise cardiac diagnosis could be made noninvasively. In this sense, these individuals were the pioneers who spurred many of us to look for more revealing noninvasive methods. In the end, as a pragmatist, I am all too pleased to recommend these earlier noninvasive methods to a prominent place in the medical archives of the Smithsonian Institution.

Echocardiography has revolutionized our appreciation of valvular heart disease and has become the standard means to establish its diagnosis and severity. Indeed, echocardiography can provide specific and life-saving information about valvular and cardiac involvement when all other measures fail, including cardiac catheterization and angiography. A typical example is a ruptured papillary muscle head in the course of acute myocardial infarction. Left ventricular angiography may establish the severity of the associated regurgitation, but it rarely defines the specific cause that is readily apparent with transthoracic or, if needed, transesophageal echocardiography. The same is true in a number of acute life-threatening situations, such as a dissecting aneurysm, intracardiac masses, or defining the complexity of valvular and perivalvular involvement in infective endocarditis or identifying atrial tamponade.

As we look at our patient population in the 1990s, echocardiography has become an important, although in my opinion underused, means to approach the problem of acute myocardial infarction, because it allows immediate recognition of the amount of myocardium at risk during acute ischemia and has clearly been shown to have important prognostic implications in multiple studies from independent investigators. Moreover, the facile bedside recognition of the mechanical complications of myocardial infarction is unparalleled by any other technique. Present-day advances in cardiac surgical technique allow a number of these patients to be saved, and echocardiography has become the linchpin in moving these patients expeditiously toward the operating room.

Conceptually, in nonvalvular heart failure we have learned to distinguish systolic from diastolic dysfunc-

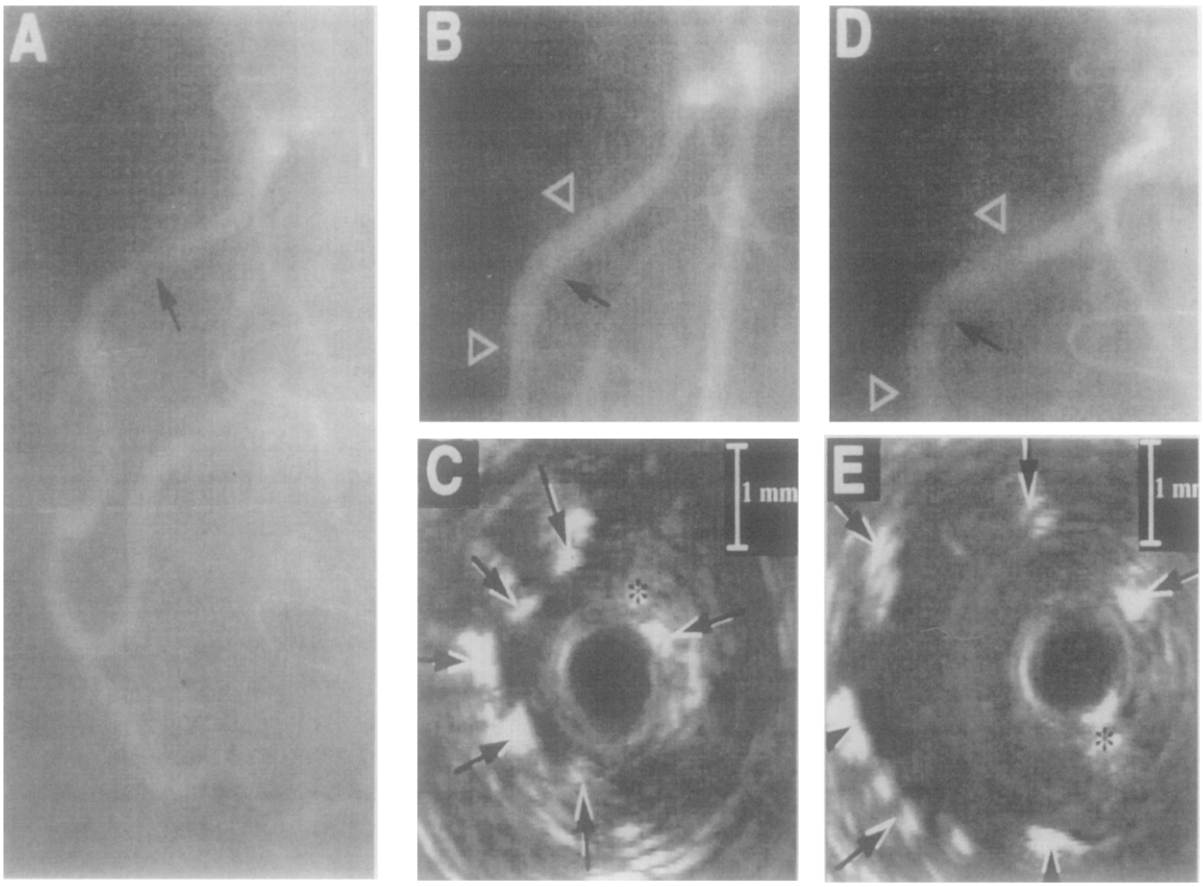

Figure 3 Role of intracoronary ultrasonography in stent deployment. **A,** Predilation angiogram with high-grade stenosis in proximal right coronary artery *(arrow)*. **B,** Angiogram after dilation of Palmaz-Schatz stent *(white triangles)* with 3.5 mm noncompliant balloon inflated to 10 atm. *Black arrow* denotes slight indentation in stent. **C,** Intravascular ultrasound image obtained at site of *black arrow* in *B* shows underexpanded stent with cross-sectional area of 3.1 $mm.^2$ Stent struts are demarcated by *black* and *white arrows* and guide wire with *asterisk*. **D,** Angiogram after final dilation of stent *(between white triangles)* with 3.5 mm noncompliant balloon inflated to 15 atm reveals minimal angiographic improvement. **E,** Subsequent intravascular ultrasound image recorded from same site *(black arrow* in *D)*. Palmaz-Schatz stent struts are well visualized (*black* and *white arrows*), and stent is now well expanded, with intrastent cross-sectional area of 9.3 $mm.^2$ (From Colombo A, et al. Intracoronary stenting without anticoagulation accomplished with intravascular ultrasound guidance. Circulation 1995;91:6; 1864.

tion, echocardiography once again playing a pivotal role in this distinction. Establishing the diagnosis of intracardiac masses, unheard of in the 1960s except occasionally by angiocardiography and usually at postmortem examination, has become an everyday phenomenon. Echocardiography is frequently called on to evaluate patients with atrial fibrillation, not only for its ability to identify the form of heart disease underlying this arrhythmia but also, for those patients with acute disease, it offers another means to evaluate the risk of early cardioversion, because it is possible to image intraatrial thrombi, particularly in the region of the left atrial appendage.

Above and beyond the breadth of application of echocardiography, it is truly amazing that echocardiography has continued to improve during the past three decades because of methodologic and instrumental innovations. In fact, echocardiography has changed more than any other cardiac diagnostic technique in continuous use during the past 30 years. What is even more exciting is that echocardiography continues to change and evolve; exciting new innovations such as second harmonic imaging and Doppler tissue imaging remain to be explored to their full potential.

We now see in this last decade of the twentieth

century the application of ultrasonography from intravascular and intracoronary sites. Intracoronary ultrasonography has played a recent pivotal role in the understanding of stent underexpansion as a cause of subacute thrombosis after implantation of these devices (Figure 3).[11] As a result, intracardiac ultrasonography is now advocated to ensure the adequate deployment of intravascular stents, and stents appear to be the single greatest advance in the history of interventional cardiology since the introduction of percutaneous transluminal coronary angioplasty in the late 1970s. It is most important to keep in mind that we are still within the first decade of clinical applications of ultrasound imaging with intravascular transducers. Based on the general improvement in cardiac imaging with ultrasound over the years, it is reasonable to expect that the images of the arterial wall that we see today will be relatively primitive compared with the type of images of the arterial wall that we will see emerge in the future. The ability to provide refined definition of atheromatous plaques of differing composition is truly within the potential of this technique, and I would predict that in decades to come echocardiography will play a key role in promoting further understanding of the biology of atherosclerosis.

Another area in which echocardiography has changed significantly and continues to change is the area of contrast echocardiography. First recognized in the 1960s as a means of identifying blood flow within and through the heart, thus facilitating the diagnosis of intracardiac and, in particular, right-to-left shunting, this technique is now being applied clinically to the detection of myocardial perfusion abnormalities (Figure 4)[12] New ultrasonic contrast agents are being developed that can image myocardial perfusion patterns after intravenous injection of contrast material. Gated and second harmonic imaging techniques appear to have important potential for further refinement of visualization of myocardial contrast. Thus contrast echocardiography with peripheral venous injections offers great promise to assess myocardium at risk during spontaneous or stress-induced ischemic episodes, as well as to define the viability of akinetic segments based on the presence or absence of ultrasonically detected myocardial perfusion.

As I near the end of this talk, I would like to return to the aphorism with which I began this lecture: "to live is to change, and to be perfect is to have changed many times." Following this definition, it is readily apparent how alive echocardiography has been during the past three decades. Your presence here today is compelling testimony to the vitality of this technique, a vitality I believe will continue to grow in breadth of application and richness of diagnostic information for many decades to come.

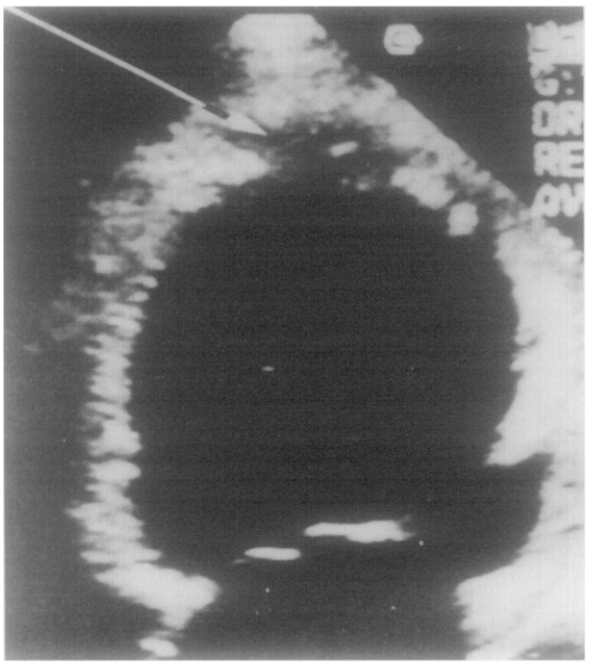

Figure 4 Contrast echocardiogram shows partial-thickness perfusion in patient with apical infarction and patent infarct-related artery from apical four-chamber view. At apex, note evidence of contrast effect in epicardial region with lack of evidence of perfusion in subendocardial area. (From Ragosta M, et al. Microvascular integrity indicates myocellular viability in patients with recent myocardial infarction: new insights using myocardial contrast echocardiography. Circulation 1994;89:6;2564.

Those of us who completed medical school training in the early 1960s and had a strong interest in cardiology were imbued by the importance of the stethoscope, then the ultimate bedside instrument of the master clinician. The word stethoscope derives from a combination of words, Latin and Greek: Latin scopium, seeing, and Greek stethos, meaning chest. The stethoscope thus was conceived to be an instrument that allowed one to see within the chest. What a misnomer that was. As we move into the twenty-first century, I believe that echocardiography will continue to grow in its application both at the bedside and in specialized laboratory settings and will really be the stethoscope of our successors.

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