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The Patient With Heart Disease and the Cardiovascular Physician and Surgeon: 1958–1983

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The patient with heart disease and the physician providing cardiovascular health care have experienced dramatic change since the American College of Cardiology first published its journal 25 years ago. During the decade before 1958, cardiology and cardiovascular surgery emerged as specialties. Surgery by closed techniques flourished and open heart surgery began. Since 1958, spectacular progress has occurred. Closed chest massage and defibrillation, electronic monitoring, advances in electrophysiology and a new pharmacology have changed cardiology. The coronary care unit has evolved into a comprehensive coronary care system. Pacemakers, myocardial revascularization and open heart surgery have become commonplace and percutaneous angioplasty an option. As custodians of cardiology's historic advances, the cardiologist and cardiovascular surgeon are cast in a role of decision maker and problem solver. Today's diagnostic and therapeutic cardiology, used appropriately, has great potential for good—used inappropriately, for great harm. The patient has the right to expect the physician to act objectively and appropriately in dealing with problems that may threaten his or her livelihood or life. The physician who does less is an unworthy heir to cardiology's great legacy of 1983.

The patient with heart disease and the physician live in a different world from that of 25 years ago when the American College of Cardiology introduced its official journal. The College now begins its second quarter century of publication.

The year 1958 was a watershed in the history of cardiology. It had been a little more than a decade since the end of World War II. During that interim, cardiology had become a specialty in its own right. Departments of cardiovascular surgery emerged. There was unparalleled expansion in training and research in cardiovascular medicine and surgery, financed and stimulated largely by the new National Heart Institute. It was a period of ferment and change. The prospect of prodigious progress lay ahead.

One way to comprehend the change experienced by both patient and physician during this quarter century is to browse through Volume 1 of 1958 and then through Volume 49 which began the 25th year of publication by the American College of Cardiology. The number of articles has tripled. There is a striking increase in papers devoted to basic, applied and clinical research. Moving quickly from Volume 1 to Volume 49 creates a kind of Rip Van Winkle effect. It is as if the reader has awakened in a world where everything is strange and new.

Cardiology in 1958

In 1958, closed chest cardiac massage to maintain the circulation and external defibrillation to restore the heartbeat were not yet in clinical use (1–4). While catheterization of the right and left chambers of the heart was well known, techniques were primitive compared with those of today (5,6). Transseptal catheterization of the left heart chambers, now used rarely, had not been introduced (7). Contrast angiography and ventriculography had very limited clinical application. Coronary arteriography had not yet demonstrated the feasibility of myocardial revascularization by internal mammary artery implantation into the left ventricle (8,9). When the College journal first went to press, cardiologists had not yet recognized the vast clinical implications of visualizing coronary circulation and the potential for surgical revascularization. Intracardiac pacemaking and

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permanent transvenous pacemakers, commonplace today, were not in use (10). Indeed, external pacemakers were relatively new and not yet cast aside (11).

Cardiac surgery. The year 1958 marked perhaps the zenith of progress in the closed surgical treatment of congenital and acquired valvular heart disease (12). The previous decade had seen surgical relief of stenotic mitral, aortic and pulmonary valves as well as some successful reconstructive efforts (13–16). Congenital lesions, such as atrial and ventricular septal defects, were yielding to the surgeon's hand (17). Open heart surgery was done by hypothermia (18). The pump oxygenator and open heart techniques were coming into vogue (19,20). Some 40 articles on the subject were published in the English language in 1957. Titles still appeared in 1958 on internal mammary artery ligation (21), implantation of the internal mammary artery into the left ventricle and poudrage to treat coronary artery disease (22,23).

Diagnostic tests. Ballistocardiography was popular in the literature of 1958 and the subject of several symposia in the College journal. High technology meant electrokymography and intracardiac phonocardiography. The only mention of radioisotopes in the College journal of 1958 described treatment of angina pectoris with radioactive iodine to produce a hypothyroid state (24). How could the physician of 1958 anticipate that 25 years later, Volume 49 would print a spectrum of articles on imaging the myocardium to define anatomy and function using radionuclides?

Pharmacologic therapy. Compared with today, the pharmacology of 1958 was limited in scope and effectiveness. Mercurials were the leading diuretics. The thiazides, furosemide and ethacrynic acid were still in the clinical future (25,26). There were, indeed, many articles on the treatment of hypertension. They reported such agents as rauwolfia, cryptenamine and protoveratrine. Beta-receptor blocking agents, calcium antagonists and angiotensin-converting enzyme inhibitors were unknown. In a single supplement to the College journal of 1982, there were more articles on drug treatment than in the entire first volume of 1958.

Although the electrocardiographic diagnosis of arrhythmias was sophisticated in 1958, electrophysiologic studies since then have enhanced our understanding of mechanisms (27). The physician of today is startled to see intravenous quinidine advocated in the management of ventricular tachycardia (28). Therapeutic choices were limited. Procainamide was a relatively recent alternative. A quarter of a century later, if the galaxy of antiarrhythmic agents, including lidocaine, beta-receptor blocking agents and calcium antagonists failed, the cardiologist could resort to sophisticated electrophysiologic mapping and surgery (29,30).

Stress testing and diagnosis of coronary artery disease. In 1958, the Master two-step test (31) was the standard provocative exercise test to diagnose ischemic coronary artery disease. Today, the Master test has been replaced by the multilevel bicycle ergometer and treadmill. The decline of the Master test was dramatically illustrated at a recent presentation to a group of our fourth year medical students and junior house officers. The vast majority had never heard of the Master test. While stress in relation to coronary disease was discussed in 1958 (32), the term type A behavior pattern to describe the coronary-prone personality had not yet become a part of the common language (33). Since that time, interest in stress and the genesis and management of coronary disease has continued to increase (34). The entire field of myocardial imaging is new since 1958. Echocardiography and radionuclides have revolutionized the noninvasive approach to valvular, myocardial and indirectly to coronary heart disease (35,36).

Progress in Cardiology Since 1958

Although its literature did not anticipate many subsequent spectacular advances, cardiology in 1958 was poised for progress. By 1960, the surgical treatment of angina pectoris was already being assessed (37). In that same year, there was a report of segmental perfusion of coronary arteries with fibrinolysin in acute myocardial infarction (38). A special unit was proposed for the treatment of patients undergoing open cardiotomy (39). Surgical approaches to diseases of the great vessels became well established (40). Progress began to accelerate exponentially.

The Coronary Care Unit

After World War II, there was a great proliferation of electronic equipment. The ability to monitor the electrocardiogram introduced an entirely new dimension to the understanding of acute coronary heart disease. Closed cardiac massage and external defibrillation heralded a new era of cardiac resuscitation. This triad of monitoring, external defibrillation and closed chest massage made possible the coronary care unit which was introduced in 1962 (41–43). The coronary care unit was a major development of the past 25 years. Its impact on cardiology was monumental. From a modest beginning in a community hospital, the coronary care unit proved that progress and innovation were not entirely the province of the great centers. The recognition and treatment of cardiac arrhythmias changed from a somewhat esoteric pursuit to a practical requirement for every physician and nurse who treated patients with acute coronary disease.

The coronary care unit triggered a virtual revolution, not limited to coronary disease. The nursing profession rapidly assumed a major role in the care of the acutely ill patient. The responsibility of the nurse, and consequently nursing educational requirements, changed dramatically. The effectiveness of the coronary care concept was not lost on those interested in other areas of acute care. Medical and

surgical intensive care units, pulmonary care units, burn units and many others were developed. Success in the coronary care unit was realized from the control of arrhythmias, which caused many deaths early in the course of acute myocardial infarction. Recognition and treatment of life-threatening arrhythmias with lidocaine and prompt defibrillation and cardioversion reduced postmyocardial infarction arrhythmic deaths to a minimum.

The problem of late postcoronary care unit mortality was challenged by the intermediate coronary care unit (44,45). Surveillance was continued throughout the later phases of the postmyocardial infarction hospital stay. These units are still in use today, although their life-saving value has not been established with certainty (46).

Interest then turned to the point of entry into the coronary care system. Vast public educational programs created a population able to recognize the signs and symptoms of acute coronary heart disease and to assist in life support and resuscitation before arrival of professional help. Ambulances evolved into mobile coronary care units staffed by well trained paramedics. Electrocardiographic monitoring and a coronary care unit approach began in the hospital emergency room immediately on arrival of the patient with chest pain.

Bedside hemodynamic monitoring and therapy. The coronary care unit did not stand long on the laurels it achieved through the control of arrhythmias. In the early 1970s, the Swan-Ganz catheter made possible bedside hemodynamic appraisal (47). A new approach to diagnosis and treatment of acute myocardial infarction followed. It was the era of hemodynamic monitoring and management. Such terms as preload and afterload introduced a new language to coronary care. As often happens, effective therapy followed accurate diagnosis. Hemodynamic monitoring and a new pharmacology of potent diuretic drugs, vasodilators, beta-receptor blocking drugs, pressor agents and calcium antagonists helped to transform the coronary care unit into a physiologic laboratory. Concurrently, surgery became bolder and more successful (48). Complications of acute myocardial infarction, such as septal perforation and mitral apparatus injury, suddenly were treated surgically (49). Events moved rapidly. Some patients with acute myocardial infarction were taken directly to the catheterization laboratory. On occasion, medical revascularization was attempted with thrombolytic enzymes (50, 51). Other patients had emergency coronary arteriography and, after definition of anatomy, surgical revascularization (52). Transluminal angioplasty, undreamed of in 1958, added a nonsurgical alternative to the treatment of occlusive coronary disease (53). Angioplasty evolved so rapidly that present techniques are probably transitional. During the 1970s, coronary arteriography and surgical treatment of occlusive coronary disease became commonplace, even before consensus could be established for indications and long-term effects.

Care of the Patient: 1983 Versus 1958

Office Diagnosis and Procedures

The magnitude of change in the past quarter century in cardiology has been so great that a few pages can offer only the barest outline. One way to view this change is to compare the experience of the patient with heart disease in 1958 with that of today's patient. The process in the physician's office begins today much as it did then. The history, the physical examination, the electrocardiogram and the chest X-ray film remain fundamental. There the similarity ends. For the patient with a heart murmur or an abnormal cardiac silhouette, the echocardiogram and radionuclide imaging and dynamic studies provide information not imagined in 1958. Pericardial effusion, mitral valve prolapse and stenosis, other valvular abnormalities, septal and free wall hypertrophy, cardiac dilatation, ventricular dysfunction and a spectrum of congenital anomalies may often be diagnosed noninvasively in a matter of minutes in the ambulatory patient.

The limited surgical alternatives in 1958 made accurate diagnosis much less urgent than today. Although some surgical procedures were available, the first prosthetic valve had yet to be implanted (54). Open heart surgery was limited and not in general use because risks and results were prohibitive. For the patient with otherwise nonremedial valvular or myocardial disease, the option of cardiac transplantation was in the realm of science fiction both technically and immunologically. Today, there are few valvular and congenital lesions for which some surgical treatment is not available. Even cardiac transplantation under certain circumstances is a practical option (55).

Coronary Heart Disease

Angina pectoris. Although progress has been dramatic in all phases of cardiovascular disease, it is the patient with coronary heart disease who occupies the center stage of cardiology in 1983. Whether measured in terms of numbers, procedures, results, dollars or impact on medicine and society, it is the diagnosis and treatment of coronary disease that dominates cardiology. In 1958, the patient presenting with chest pain and suspected of having ischemic coronary disease required a relatively simple investigation. This included a history, physical examination, resting electrocardiogram and perhaps a Master two-step test. Except for nitroglycerin, therapy was not specific. The approach was indirect and supportive and emphasized control of pain, congestive heart failure and arrhythmias. Related syndromes such as hypertension, hyperthyroidism, diabetes and obesity were treated.

As is still the case, caveats included smoking, emotional and physical stress and fatigue. In 1958, there probably was less emphasis on physical exercise and behavior modification to control stress. Some controversies such as anticoagulation and diet remain. A commentary on coronary artery

disease (56), published in 1958, quoted an American Medical Association Council on Food and Nutrition opinion, ". . . quite a bit more study concerning dietary fads will have to be done before the average American eating habits are to be drastically altered." The author also recommended a low cholesterol diet despite the incomplete evidence. These statements could have been written today.

With the exception of nitroglycerin, drug therapy for symptomatic ischemic coronary disease is totally different from that of 1958. The long dormant concept of coronary artery spasm has been restored to clinical importance (57). Long-acting nitrates, orally and transcutaneously, beta-receptor blocking drugs, calcium antagonists, more effective diuretic drugs, peripheral vasodilators and psychoactive agents contribute today to better control of angina.

Unstable angina. For the patient with chest pain hospitalized in 1983 because of unstable angina, the experience is entirely different from that of 1958. Today, he or she will be placed in a cardiac care facility where electrocardiographic monitoring is constant and hemodynamic surveillance available. The immediate therapeutic objective is the control of symptoms and signs of clinical instability. In addition to the pharmacologic regimen already described for the ambulatory patient with angina, anticoagulant agents, intravenous nitroglycerin and narcotic drugs may be used. When the patient's condition has been controlled medically, elective coronary arteriography and ventriculography will be performed. A decision can then be made for definitive medical or surgical management. If the patient's condition cannot be stabilized medically, coronary arteriography will be carried out as a relative emergency.

Acute myocardial infarction. For the patient with acute myocardial infarction, the program is also dramatically different from that of 1958. The coronary care unit, with its electrocardiographic and hemodynamic monitoring, capability for resuscitation, defibrillation, cardioversion and transvenous pacing and intraaortic balloon assistance, was unknown in 1958. In 1958 the hallmark of treatment for acute myocardial infarction was protracted bed rest, perhaps 6 weeks. At that time, a radical innovation was to lift the patient out of bed in a chair (58). While papers were beginning to advocate a shorter stay for patients designated as "mild," (59) it was not until the middle 1970s that bed rest and length of stay were reduced materially (60).

Virtually every aspect of management of acute myocardial infarction in 1983 is different from that of 1958. One of the most important changes is philosophic. Today's approach to the patient with acute myocardial infarction looks beyond the acute phase. This attitude evolved after the development of significant medical and surgical means of improving long-term prognosis. From the moment the patient enters the coronary care unit, prognostic evaluation begins simultaneously with treatment. Clinical, electrocardiographic and hemodynamic evaluation helps to define short-term prognosis. Relatively unfavorable factors include anterior location of infarction, cardiac enlargement, intraventricular conduction defects, recurrent infarction, persistent tachycardia, severe and protracted pain (61,62) and an inordinately high rise in serum enzyme levels (63). From a hemodynamic point of view, hypotension not due to hypovolemia, pulmonary congestion, elevated left ventricular filling pressure or reduced ejection fraction suggests a more ominous short-term prognosis (64). Recognition of right ventricular infarction and the requirement for specific therapy are new since 1958 (65). The severity of the myocardial insult may be defined further by echocardiography (66) and by radionuclide techniques (67). Other ways of categorizing the acute infarction include the designations complete or incomplete, transmural or nontransmural and complicated or uncomplicated (60). Each of these categories is significant in defining treatment and prognosis. For example, in 1958 a nontransmural infarction was considered relatively benign. Today it is known that the future of patients with this condition may be even more uncertain than that of patients with a completed transmural lesion (68). The patient with uncomplicated myocardial infarction may be hospitalized for less than 2 weeks and the patient with complicated infarction rarely for more than 3.

Postinfarction management. After myocardial infarction, the ultimate prognosis depends on three factors—electrical stability, the integrity of the myocardium and the character of coronary artery stenosis. Long-term evaluation begins before discharge from the hospital. It is at this point that the decision-making process for patients with unstable angina and patients with a recent myocardial infarction converges. Here controversy remains. How important is the presence of symptoms in patients who have had unstable angina or recent myocardial infarction? What happens to the patient depends on the philosophy of his or her physician and how the physician interprets the published data and his or her own experience. Some physicians may select a high risk postmyocardial infarction group by the response to an exercise stress test (69). A 24 hour ambulatory electrocardiogram may suggest a high risk category because of electrical stability or an ischemic response to activity (70). Other physicians may consider that all patients without specific contraindications should have coronary anteriography after recovery from acute myocardial infarction (71,72). Still others may proceed with a medical regimen without even considering coronary arteriography.

Surgical therapy. One of cardiology's great dilemmas today is in the surgical management of coronary disease. Absolutes are few. Consensus currently favors surgical revascularization for patients with obstructive left main disease or symptomatic multivessel occlusive coronary disease. For patients with limited symptoms and less compelling combinations of obstructive disease and left ventricular dysfunction there is no consensus. The selection of a medical

regimen from available medications, exercise rehabilitation (73) and behavior modification programs is no less perplexing.

Cardiology: Present Status

This first issue of the *Journal of the American College of Cardiology* reviews and interprets the evolution of cardiology during the past 25 years. Topics vary from epidemiology to echocardiography, from enzymes to electrophysiology, from revascularization to rehabilitation and from arrhythmias to arteriography. Almost everything written is new since 1958. These advances are not confined to the great centers. Relatively remote hospitals throughout the country are staffed with highly trained cardiologists and offer sophisticated facilities.

Although it may seem that progress has made the work of the physician easier now than in 1958, this impression is misleading. Today, diagnosis is more accurate and intervention more decisive. The physician's responsibility is much greater. Failure to diagnose bacterial endocarditis, a dissecting aneurysm or an ulcerated carotid lesion, or to recognize a subtle anginal equivalent, may deny the patient a chance for a cure. As never before, the physician has the opportunity to do good and the possibility to do harm.

Decision-making process. In every stage of the diagnostic and therapeutic process, the cardiologist will make decisions and perform or advise procedures that will affect the quality of life and perhaps even patient survival. The decision-making process is the very essence of the practice of medicine. This is especially true for the cardiology of 1983.

In the practice of cardiology, the setting for a vital decision is not necessarily dramatic. It is more likely to be mundane and innocent. A 45 year old woman presents for physical examination. She may describe a typical chest pain. Her physician elects to perform a multilevel treadmill stress test. ST-T segment depression occurs. A pivotal decision must be made. Is the result of the exercise test definitive? Does it really suggest the probability of life-threatening occlusive coronary disease? Does it warrant arteriography? Is it a false positive result or a normal variation? Does it really correlate with the patient's history, symptoms and other findings? The physician's decision may subject the patient to an expensive invasive procedure with potential, though limited, risk of a major complication or even death. Failure to perform the test may cause the diagnosis of life-threatening coronary disease to be missed. A negative result may reassure the patient, but at a cost difficult to bear and which society is increasingly reluctant to absorb. This is true particularly if astute judgment could avoid the procedure. The arteriogram may show some degree of arterial narrowing. Again, a fateful decision must be made. One judgment may lead the patient to the expense, morbidity and danger of surgery. Another may choose a medical program. The decision for medical treatment when surgery is needed is no less inappropriate than unnecessary surgery. The practice of medicine and cardiology is both art and science; neither is exact. Effective decision making is as important to the patient and society as the development of the test and the procedure. The ingenuity, talent and energy that created coronary arteriography and open heart surgery are wasted if these procedures are applied inappropriately. The quality of the decision-making process must be comparable to that of the intervention it selects.

Evaluation of clinical trials. Since World War II, clinical studies have become standard for evaluating diagnostic and therapeutic procedures. Prospective, randomized, double-blind trials have been used to document drug safety and efficacy. However well intentioned, these studies are rarely perfect. Although it is attractive to think that a protocol can be designed to prove whether a drug or a procedure is effective, in practice, unequivocal answers are rare. Several searching commentaries on therapeutic trials have described their limitations (74–76). Statistical analysis may suggest a good result erroneously or may not recognize a favorable trend. The reasons for these errors are complex. They may be inherent in the design of the trial, in the execution or in the interpretation. There are many instances in cardiology where clinical trials are not definitive. Well known examples are the use of anticoagulant and antiplatelet agents, lipid-lowering diets and agents and, indeed, the role of myocardial revascularization. The evaluation of a diagnostic or surgical technique may be more difficult than that of a medication. The composition of a medication is constant. The result of a surgical procedure varies with the operator's skill.

Measures to improve decision-making process. Because definitive answers are few, how can the decision-making process be improved? Perhaps the first step is to recognize that few tests provide absolute answers. The electrocardiographic evolution of acute myocardial infarction, an elevated blood sugar level or a positive pathologic biopsy may be unequivocal. Many clinical tests on which decisions are made are not. The significance of an exercise stress test, a coronary arteriogram or radionuclide imaging may be uncertain. The significance of a test must be evaluated in the clinical context. The incidence of the disease in the population which the patient represents, the medications, physical findings and symptoms are vitally important in interpretation of a test. The physician must consider sensitivity, specificity and the positive and negative predictive value of each test. These usually cannot be expressed in finite numbers. Because decision making is generally not based on absolutes, the physician must have special qualities. These are knowledge, experience, empathy, judgment and character. They are vital to the decision-making process. Without knowledge and experience, the physician lacks the intellectual and practical balance to decide. Without empathy he will not understand the patient's symptoms, expectations

and life situation requirements. Judgment and character are the priceless ingredients. Unfortunately, some elements of judgment are inherent, difficult to learn and not distributed equally. Given a reasonable level of knowledge, experience, empathy and judgment, it is character or integrity that may be the most important intangible in the decision-making process. This quality is demanded of the physician to whom society has given the privilege of making vital decisions in a milieu where he or she may be in conflict of interest. A physician making decisions in cardiology in 1983 must put aside self-interest and at times academic curiosity and the desire of the virtuoso to perform in order to serve the best interest of his patient.

As custodians of cardiology's historic advances, the cardiologist and the cardiovascular surgeon are cast in a role of problem solving and not case finding. The patient with chest pain does not present to have a stress test or an arteriogram or open heart surgery. He or she presents to have a problem solved that may threaten a livelihood or a life. He or she has the right to expect that in dealing with this problem, the physician will draw appropriately from cardiology's vast diagnostic and therapeutic reservoir. The physician who does less is an unworthy heir to cardiology's great legacy of 1983.

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References

1. Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed chest cardiac massage. JAMA 1960;173:1065–7.
2. Kouwenhoven WB, Milnor WR, Knickerbocker GG, Chestnut WR. Closed chest defibrillation of heart. Surgery 1957;42:550–61.
3. Zoll PM, Linenthal AJ, Gibson W, Paul MH, Norman LR. Termination of ventricular fibrillation in man by external applied electric countershock. N Engl J Med 1956;254:727–32.
4. Lown B, Neuman J, Anarasingham R, Berkovits B. Comparison of alternating current with direct current electroshock across the closed chest. Am J Cardiol 1962;10:223–33.
5. Cournand AF, Riley RL, Breed ES, Baldwin E deF, Richards DW Jr. Measurement of cardiac output in man using the technique of catheterization of the right auricle or ventricle. J Clin Invest 1945;24:106–16.
6. Zimmerman HA, Scott RW, Becker ND. Catheterization of the left side of the heart in man. Circulation 1950;1:357.
7. Ross J Jr. Transseptal left heart catheterization of the left atrium; preliminary report. J Thorac Surg 1959;37:482–6.
8. Sones FM Jr, Shirey EF, Proudfit WL, Westcott RN. Cine-corconary arteriography (abstr). Circulation 1959;20:773–4.
9. Vineberg A. Revascularization of the right and left coronary arterial system. Am J Cardiol 1967;19:344–53.
10. Furman S, Robinson G. Use of intracardiac pacemaker in correction of total heart block. Surg Forum 1958;9:245–8.
11. Zoll PM. Resuscitation of the heart in ventricular standstill by external electrical stimulation. N Engl J Med 1952;247:768–71.
12. Friedberg CK. Progress in cardiac surgery: general survey—indications for surgery, surgical risks and results. Prog Cardiovasc Dis 1958;1:2–27.
13. Harken DE, Ellis LB, Ware PF, Normal LR. Surgical treatment of mitral stenosis. Valvuloplasty. N Engl J Med 1948;239:801–9.
14. Bailey CP. Surgical treatment of mitral stenosis (mitral commissurotomy). Dis Chest 1949;15:377–93.
15. Baker C, Brock RC, Campbell M. Valvulotomy for mitral stenosis: report of six successful cases. Br Med J 1950;1:1283–93.
16. Ellis LB, Harken DE, Black H. A clinical study of 1,000 consecutive cases of mitral valvuloplasty. Circulation 1959;19:803–20.
17. Lillehei CW, Cohen M, Warden HE, Varco RL. The direct vision intracardiac correction of congenital anomalies by controlled cross circulation. Surgery 1955;38:11–29.
18. Swan H, Virtue R, Blount SG Jr, Kircher LW. Hypothermia in surgery. Analysis of 100 clinical cases. Ann Surg 1955;143:382–400.
19. Gibbon JH Jr. Application of a mechanical heart and lung apparatus to cardiac surgery. Minn Med 1954;37:171–80.
20. Cooley DA, Latson JR, Keats AS. Surgical considerations in repair of ventricular and atrial septal defects utilizing cardiopulmonary bypass. Experience with 104 cases. Surgery 1958;43:214–25.
21. Kitchell JR, Glover RP, Kyle RH. Bilateral internal mammary artery ligation for angina pectoris. Am J Cardiol 1958;1:46–50.
22. Beck CS. Coronary heart disease—a report to William Harvey 300 years later. Am J Cardiol 1958;1:38–45.
23. Bakst AA, Loewe L. The surgical therapy of occlusive coronary artery disease—present status. Am J Cardiol 1958;1:340–4.
24. Segal RL, Silver S. Yohalem SB, Newburger RA. Use of radioactive iodine in the treatment of angina pectoris. Am J Cardiol 1958;1:671–81.
25. Brest AN, Likoff W. Studies in diuretic therapy. Am J Cardiol 1959;2:144–7.
26. Kim KE, Onesti G, Moyer JH, Swartz J. Ethacrynic acid and furosemide. Am J Cardiol 1971;27:407–15.
27. Fisch C, Knoebel SB, Feigenbaum H, Greenspan K. Potassium and monophasic action potential, electrocardiogram, conduction and arrhythmias. Prog Cardiovasc Dis 1966;8:387–418.
28. Sigler LH. Severe cases of ventricular tachycardia. Treatment with intravenous quinidine. Am J Cardiol 1958;1:637–9.
29. Josephson ME, Horowitz LN, Spielman SR, Waxman HL, Greenspan AM. Role of catheter mapping in the preoperative evaluation of ventricular tachycardia. Am J Cardiol 1982;49:207–20.
30. Josephson ME, Harken AH, Horowitz LN. Endocardial excision: a new surgical technique for the treatment of recurrent ventricular tachycardia. Circulation 1979;60:1430–9.
31. Master AM, Friedman R, Dack S. Electrocardiogram after standard exercise as a functional test of the heart. Am Heart J 1942;24:777–93.
32. Russek HI. Emotional stress and the etiology of coronary artery disease. Am J Cardiol 1958;1:129–33.
33. Friedman M, Rosenman RH. Association of a specific overt behavior pattern with increases in blood cholesterol, blood clotting time, incidence of arcus senilia and clinical coronary artery disease. JAMA 1959;169:1286–96.
34. Elliott RS, Forker AD. Emotional stress and cardiac disease. JAMA 1976;236:2325–6.
35. Feigenbaum H. Echocardiography. In: Ferrir MI, ed. Current Cardiology. Boston: Houghton Mifflin, 1979:225–58.
36. Willerson JT. Nuclear cardiology, 1981 and beyond. In: Yu PN, Goodwin JF, eds. Progress in Cardiology. Philadelphia: Lea & Febiger, 1972:253–68.
37. Dock W. Surgical treatment of angina. Am J Cardiol 1960;5:1–2.
38. Boucek RJ, Murphy WP Jr, Sommer LS, Voudoukis IJ. Segmental

perfusion of the coronary arteries with fibrinolysin in man following a myocardial infarction. Am J Cardiol 1960;6:525–33.

39. Hanks EC, Deterling RA Jr, Himmelstein A, Scheiner M, Papper EM. A special care unit for the postoperative open cardiotomy patient. Am J Cardiol 1960;6:778–82.
40. DeBakey ME, Crawford ES, Garrett HE, Beall AC Jr, Howell JF. Surgical considerations in the treatment of aneurysms of thorical or abdominal aorta. Ann Surg 1965;162:650–62.
41. Day HW. An intensive coronary care area. Dis Chest 1963;44:423–7.
42. Brown KWG, MacMillan RL, Forbath N, M'Grano F, Scott JW. An intensive care centre for acute myocardial infarction. Lancet 1963;2:349–52.
43. Meltzer LE. Concepts and systems for intensive coronary care. Acad Med NJ Bull 1964;10:349–52.
44. Gotsman MS, Schrire V. Acute myocardial infarction: an ideal concept of progressive coronary care. S Afr Med J 1958;43:829–32.
45. Weinberg SL. Complacency in coronary care. Dis Chest 1969;56:273–4.
46. Weinberg SL. Intermediate coronary care. Observations on the validity of a concept. Chest 1978;73:154–7.
47. Swan HJC, Ganz W. Forrester JS, Marcus H. Catheterization of the heart in a man with the use of a flow directed balloon tipped catheter. N Engl J Med 1970;283:447–51.
48. Favaloro RG, Effler DB, Groves LK. Severe segmental obstruction of the left main coronary artery and its divisions: surgical treatment by the saphenous vein graft technic. J Thorac Cardiovasc Surg 1970;60:469–82.
49. Kay JH. Observations on emergency surgery for complications of myocardial infarction. Heart Lung 1972;1:340–3.
50. Ganz W, Buchbinder N, Marcus H, Mondkar A. Intracoronary thrombolysis in evolving myocardial infarction. Am Heart J 1981;101:4–13.
51. Rentrop KP, Blanke H, Karsch KR, Rahlf G, Leitz K. Acute myocardial infarction: intracoronary application of NTG and streptokinase in combination with transluminal recanalization. Clin Cardiol 1979;2:354–63.
52. Berg R Jr, Selinger SL, Leonard JJ, Grunwald RP, O'Grady WP. Immediate coronary artery bypass for acute evolving myocardial infarction. J Thorac Cardiovasc Surg 1981;81:493–7.
53. Gruntzig AR, Senning A, Siegenthaler WE. Nonoperative dilatation of coronary artery stenosis. N Engl J Med 1979;301:61–8.
54. Starr A, Edwards ML. Mitral replacement: clinical experience with a ball valve prosthesis. Ann Surg 1961;154:726–40.
55. Barnard CN. Human cardiac transplantation. An evaluation of the first two operations performed at the Groote Schuur Hospital, Capetown. Am J Cardiol 1968;22:584–96.
56. Krause S. Coronary artery disease. Present concepts. Am J Cardiol 1958;1:334–9.
57. Maseri A, Mimmo R, Chierchia S. Coronary artery spasm as a cause of acute myocardial ischemia in man. Chest 1975;68:625–33.
58. Levine SA. Some puzzling points concerning angina pectoris. Am J Cardiol 1958;1:19–25.
59. Prinzmetal M, Weiner SM, Bhuyan MC. Mild myrocardial infarction, clinical features and new method of management. Am J Cardiol 1958;1:27–37.
60. Swan HJC, Blackburn HW, DeSanctis R, et al. Duration of hospitalization in uncomplicated completed acute myocardial infarction. An ad hoc committee review. Am J Cardiol 1976;37:413–9.
61. Col J, Weinberg SL. The incidence and mortality of intraventricular conduction defects in acute myocardial infarction. Am J Cardiol 1972;29:344–50.
62. Weinberg SL. Natural history six years after acute myocardial infarction. Is there a low risk group? Chest 1976;69:23–8.
63. Sobel BE, Bresnahan GF, Shell WE, Yoder RD. Estimation of infarct size in man and its relation to progress. Circulation 1972;46:640–8.
64. Forrester JS, Diamond G, Chatterjee K, Swan HJC. Medical therapy of acute myocardial infarction by application of hemodynamic subsets. N Engl J Med 1976;295:1356–404.
65. Strauss HD, Sobel BE, Roberts R. Influence of occult RV infarction on enzymatically estimated infarct size, hemodynamics and prognosis. Circulation 1980;62:503–8.
66. Corya BC, Rasmussen S, Knoebel SB, Feigenbaum H. Echocardiography in acute myocardial infarction. Am J Cardiol 1975;35:1–10.
67. Rigo P, Murray M, Strauss HW, Taylor D, Kelly D. LV function in acute myocardial infarction evaluated by gated scintiphotography. Circulation 1974;50:678–83.
68. Hutter AM Jr, DeSanctis RW, Flynn T, Yearman LA. Nontransmural myocardial infarction: a comparison of hospital and late clinical course of patients with that of matched patients with transmural anterior and transmural inferior myocardial infarction. Am J Cardiol 1981;48:595–603.
69. Theroux P, Waters DD, Halphen C, Degaisieux JC. Prognostic value of exercise testing soon after myocardial infarction. N Engl J Med 1979;301:34–42.
70. Moss AJ, Schnitzler R, Green R, DeCamilla J. Ventricular arrhythmias three weeks after acute myocardial infarction. Ann Intern Med 1971;75:837–41.
71. Karimian HO, Joffe CD, Marcus DR, Imm CW, Weinberg SL. The clinical importance of coronary arteriography after myocardial infarction. Heart Lung 1979;8:87–91.
72. Turner JD, Rogers WJ, Mantle JA, Rackley CE, Russell RO Jr. Coronary angiography soon after myocardial infarction. Chest 1980;77:58–67.
73. Hellerstein HK. Exercise therapy in coronary disease. Bull NY Acad Med 1968;44:1028–47.
74. Selzer A. On the limitation of therapeutic intervention trials in ischemic heart disease. The clinician's viewpoint. Am J Cardiol 1982;49:252–5.
75. Sachett DL. The competing objectives of randomized trials. N Engl J Med 1980;303:1059–60.
76. Lee KL, McNee F, Starmer CF, Harris PJ, Rosati RA. Clinical judgment and statistics: lessons from a simulated randomized trial in coronary disease. Circulation 1981;81:508–14.