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Presidential Address

Observations on the coronary circulation

With tributes to my teachers

David C. Sabiston, Jr., M.D.,* *Durham, N. C.*

At intervals it is helpful to repeat a simple yet incisive quotation from Shakespeare: "What's past is prologue." Moreover, the great philosopher at Harvard, Santayana, reinforced this message, saying: "Those who cannot remember the past are condemned to repeat it." With these comments in mind, it is appropriate to recall some of the milestones from the past in discussing the coronary circulation.

In his commentaries published in 1768, the famous English physician William Heberden[1] (Fig. 1) provided the first clinical description of angina pectoris. Heberden described, in an exceedingly poignant manner, the clinical symptoms of angina as well as its prognosis. This description is such a classic that it bears frequent repetition (Fig. 2). Heberden said:

> There is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it, and sense of strangling, and anxiety with which it is attended may make it not improperly be called angina pectoris.

Read at the Sixty-fifth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., April 29–May 1, 1985.

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They who are afflicted with it, are seized while they are walking (more especially if it be up hill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or continue; but the moment they stand still, all this uneasiness vanishes.

In all other respects, the patients are, at the beginning of this disorder, perfectly well, and in particular have no shortness of breath from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the sternum, and often more inclined to the left than to the right. It likewise very frequently extends from the chest to the middle of the left arm. The pulse is, at least sometimes, not disturbed by this pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. Males are most liable to that disease, especially such as have passed their fiftieth year.

After it has continued a year or more, it will not cease so instantaneously upon standing still, and it will come on not only when the persons are walking, but when they are lying down, especially if they lie on their left side, and oblige them to rise up out of their beds. In some inveterate cases it has been brought on by the motion of a horse, or a carriage, and even by swallowing, going to stool, or speaking, or any disturbance of the mind . . . .

The termination of the angina pectoris is remarkable. For if no accidents intervene, but the disease go on to its

Fig. 1. William Heberden.

COMMENTARIES

ON

THE HISTORY AND CURE

OF

DISEASES.

BY

WILLIAM HEBERDEN, M.D.

Fig. 2. Heberden W.: Commentaries on the History of Cure of Disease. London, 1802, T. Payne.

height, the patients all suddenly fall down, and perish almost immediately. Of which indeed their frequent faintness, and sensations as if all the powers of life were failing, afford no obscure intimation.*

*Heberden W: Commentaries on the History of Cure of Diseases, London, 1802, T. Payne.

Fig. 3. Alfred Blalock.

Most regard Sir William Osler as being among the greatest physicians of the late nineteenth and early twentieth centuries. In 1897, he[2] delivered a series of lectures on "Angina Pectoris and Allied States," and these were published as a monograph.[2] His ability to clearly associate clinical signs and symptoms with pathological lesions is skillfully illustrated in these presentations. Osler likened the pain of myocardial ischemia to intermittent claudication of the lower extremities, stating quite simply that *claudication* is due to obstruction of the iliofemoral system and appears following excessive muscular contraction. Similarly, he said, angina pectoris occurs in patients with coronary arterial obstructive lesions, and when the heart is forced to overwork, as during exercise or in the presence of emotional stress, there is insufficient blood supply to deliver the oxygen needed and anginal pain results. Osler therefore provided a sound pathophysiological basis for this very important clinical disorder.

It is rather paradoxical that it was not until 1912 that an antemortem diagnosis of acute myocardial infarction was made. Herrick[3] described a 55-year-old patient with intractable substernal pain, pallor, a rapid and weak pulse, and with a low urinary output. Herrick made a diagnosis of acute coronary occlusion and 52 hours later the patient died. Hektoen, the famous pathologist, performed the postmortem examination and found a

Fig. 4. Former chief residents of Alfred Blalock who were the author's teachers: Mark M. Ravitch, William P. Longmire, Jr., H. William Scott, Jr., C. Rollins Hanlon, William H. Muller, Jr., Denton A. Cooley, Henry T. Bahnson, and Andrew G. Morrow.

severe stenosis of the left main coronary artery superimposed upon which was a fresh thrombus.

The first suggestion that a surgical approach might have a role in the treatment of angina pectoris was made by a professor of physiology at the University of Paris, Francois-Franck,[4] in 1899. He predicted that excision of the cervical sympathetic chain would interrupt the pain fibers to the heart and therefore provide relief for patients with anginal pain. The procedure was actually performed in 1916 by Jonnesco[5] of Bucharest with a successful result. Despite the relief of anginal pain which occurred in some patients undergoing this procedure, this approach did not provide additional blood supply and was ultimately abandoned.

A number of early pioneers responded to the challenge of augmenting coronary blood flow, including O'Shaughnessy[6] of London, Fauteux[7] of Montreal, Beck[8] of Cleveland, Harken[9] of Boston, and many others who devised procedures designed to revascularize the myocardium in an attempt to increase the number of collaterals and add new vessels to the coronary circulation. Pedicles of tissue such as muscle or omentum were sutured to the surface of the heart, and epicardial abrasion was tried, but none of these techniques ever became widely adopted. Later, Beck and associates[10] attempted to add new arterial blood with a more extensive procedure by creating an aorta-coronary sinus anastomosis with a vein graft designed to perfuse the coronary circulation retrogradely. This approach was also unsuccessful.

In 1946, Vineberg[11] implanted the internal mammary artery into the left ventricular myocardium and showed, to the astonishment of many, that this vessel developed arterial communications with the coronary arteries. Despite the fact that these vessels remained patent for prolonged periods, direct measurement of blood flow in these internal mammary implants performed in patients whose chests were later reopened for other reasons indicated that the blood flow in them was minimal.[12] Therefore, this technique became infrequently utilized.

I wish to pay special tribute to my teachers, to whom I owe much, and they will be cited in the time frame of their influence and impact. My greatest teacher was clearly Alfred Blalock, a master surgeon, innovative investigator, thoughtful counselor, and personal friend (Fig. 3). My respect and admiration for him are boundless and my gratitude is of equal proportions. My personal thoughts, together with an account of his many contributions to surgical science and to the training of a school of surgery, have previously been published as the Presidential Address before the American Surgical Association in 1978.[13]

At the time I was a medical student and resident in surgery, Dr. Blalock had surrounded himself with a number of extremely bright young men who were also my teachers including Drs. Ravitch, Longmire, Scott, Hanlon, Muller, Cooley, Bahnson, and Morrow (Fig. 4). With each of them I had the privilege of working clinically, both helping them and they assisting me in the operating room. In addition, there were many memora-

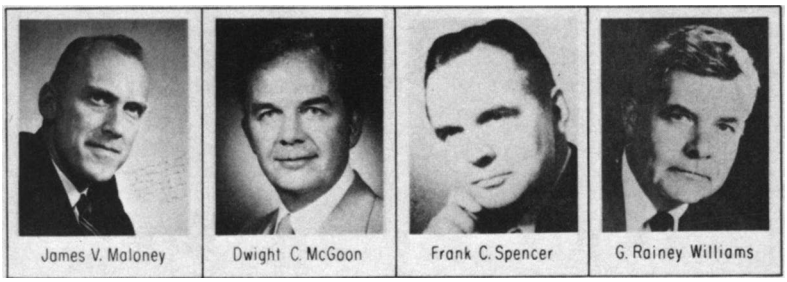


Fig. 5. Former chief residents of Alfred Blalock who were contemporaries of the author and have remained close colleagues: James V. Maloney, Jr., Dwight C. McGoon, Frank C. Spencer, and G. Rainey Williams.

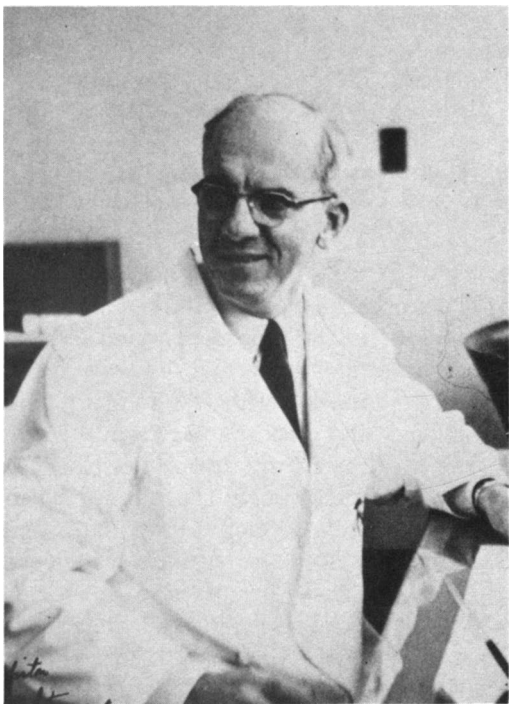


Fig. 6. Donald E. Gregg.

ble experiences in the experimental laboratory and in the preparation of clinical and research manuscripts.[14-18] Dr. Blalock had taught us all to work together, and this was another of his basic principles that was to remain with us. There were many others in the training program, too numerous to mention individually, but several who have been especially close through the years and are known for their original contributions. They include Dwight McGoon, Jim Maloney, Frank Spencer, and Rainey Williams (Fig. 5), and for their positive influence I pay to each grateful tribute.

I shall never forget a day in June of 1953, near the completion of the surgical residency at Hopkins, when Dr. Blalock asked me to remain on the faculty specifically to develop a myocardial revascularization program. While today such an appointment would be considered an optimal one, at that time there was little of consequence that could be done surgically to improve coronary blood flow. Nevertheless, the field provided great challenge and opportunity, and I quickly accepted the offer with considerable excitement.

Shortly thereafter I was called to active duty in the U.S. Army Medical Corps and sent to the Walter Reed Army Medical Center in Washington, D. C. It was fortunate that I was assigned to the Army Medical Service Graduate School there, where I had the privilege of spending 2 full-time years working with the world-recognized leader in coronary physiology, Donald E. Gregg (Fig. 6). He[19] had written the most respected and most frequently cited monograph on the subject and this book was considered the most authoritative reference in the field. I owe much to this distinguished scientist, who was not only a brilliant investigator, but also quite patient with his students. In that stimulating environment we worked side by side in the laboratory daily for 2 years.

At the outset, he thought my first task should be a review of those basic physiological and metabolic features of the coronary circulation that had become accepted as established facts. He had just been invited by a leading journal to contribute an in-depth review of the coronary circulation and asked me to join him in preparing it.[20] Then came a study of those specific aspects of the coronary circulation that were quite unlike the anatomic, physiological, and metabolic features of other body organs. These included the fact that the large arteries are primarily on the *surface* of the heart, and that quite paradoxically the most important organ in the body is endowed by nature with exceedingly few collateral vessels of much functional significance. Moreover, the heart has *multiple* anatomic pathways of venous

Table I. *Coronary arterial stenosis: Intraoperative measurement of distal pressure (39 coronary arteries)**

Stenosis (%)	*No.*	*Distal pressure (mm Hg)*
79-89	24	18
90-99	7	33
100	8	30

*Adapted from Oldham HN Jr, Rembert JC, Greenfield JC Jr, Wechsler AS, Sabiston DC Jr: Intraoperative Relationships between Aorto-Coronary Bypass Graft Blood Flow, Peripheral Coronary Artery Pressure and Reactive Hyperemia, *in* Primary and Secondary Angina Pectoris, A Maseri, GA Klassen, M Lesch, eds., New York, 1978, Grune & Stratton, Inc.

Table II. *Myocardial oxygen consumption**

	MVO_2 (ml/100 gm/min)
Normal heart	8-10
Beating, nonworking, heart	3.4
Ventricular fibrillation	3.8
Arrested heart	2.3

*Average utilization for body as a whole: 0.35 ml/100 gm/min.

drainage, and the oxygen extraction of the heart is quite remarkable, since even at rest, some three quarters of all oxygen delivered to the capillary bed is extracted. Finally, the coronary arteries are known to have an extraordinary capacity to undergo massive vasodilation even at a constant arterial perfusion pressure, and this major compensatory feature has often been underemphasized.

Much has been written concerning arterial *collaterals,* and it is generally accepted that arterial stenosis throughout the body favors the development of collateral channels. However, in the human heart this is usually minimal. In studies conducted with my colleagues, H. Newland Oldham, Joseph C. Greenfield, and others, the pressures in the coronary arteries were directly measured *distal* to severe stenoses at the time of operation in a group of patients undergoing operative procedures for myocardial revascularization.[21] It was interesting that the distal pressures were generally low, even in the vessels that were completely occluded proximally with a patent lumen beyond as well as those with a 70% or greater occlusion (Table I). Thus, even in the vessels with total proximal occlusion the mean distal pressure averaged only 30 mm Hg. In other arterial systems with this magnitude of occlusion, the distal pressure would usually be considerably greater.

Of primary importance is an appreciation of the

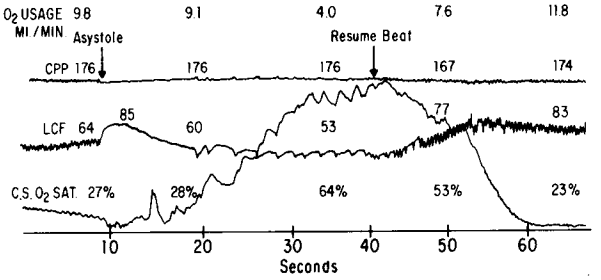


Fig. 7. The effect of asystole on myocardial oxygen consumption. Direct recording of data obtained from open-chest dog with cannula inserted in left coronary artery and continuous monitoring by rotameter. The rotameter was also supplied from a gravity source of arterial blood for perfusion at an identical pressure. Oxygen saturation in the coronary sinus was measured through a small catheter with continuous withdrawal through a cuvette densitometer. Following the induction of asystole, the coronary circulation is perfused from the reservoir. The left coronary flow rises and then slowly falls. The oxygen saturation in the coronary sinus increases while the oxygen usage of the myocardium falls from a control value of 9.8 to 4.0 ml/min. Following resumption of the beat, the oxygen saturation in the coronary sinus falls slightly below the control level and the coronary blood flow rises. *CPP,* Coronary perfusion pressure. *LCF,* Left coronary flow. *C.S.O_2 SAT.,* Oxygen saturation in coronary sinus. (From work performed with Dr. Donald E. Gregg.)

oxygen requirements of the myocardium. During normal cardiac output the heart requires a tremendous amount of oxygen, 8 to 10 ml/100 gm/min (Table II). Moreover, in the beating, *nonworking* heart such as during extracorporeal circulation with no cardiac output, the oxygen requirement is still quite high, and is even higher during ventricular fibrillation. Finally, when the heart is absolutely motionless, the basal metabolic rate of this organ at normothermia is about one fourth of that required for the normal cardiac output.

Donald Gregg was deeply committed to objective and reproducible studies of the moment-to-moment oxygen requirements of the myocardium. For this reason, he suggested development of a device designed to place an indwelling catheter in the coronary sinus allowing blood in it to be continuously withdrawn through an appropriate recorder such that the oxygen levels in the blood could be continuously recorded. He set me, Edward Khouri, and others to work on the development of the project, and ultimately it was possible to utilize a cuvette densitometer that provided accurate calibration curves for determination of myocardial oxygen consumption.[22] Using this instrument, we were able to perform instantaneous measurements of oxygen saturation at varying

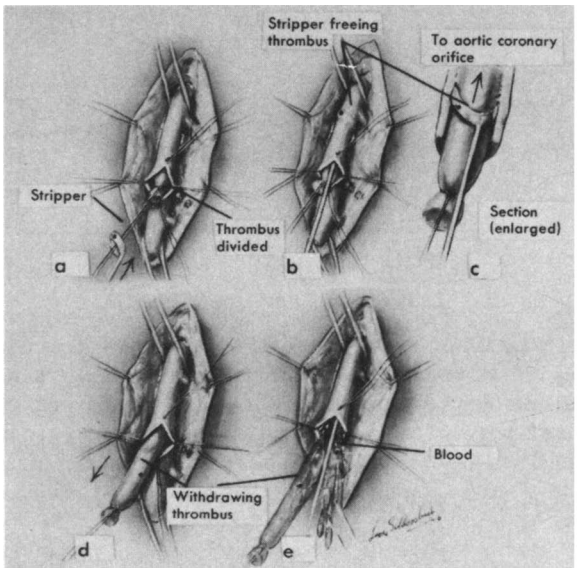


Fig. 8. *a,* The thrombus is divided and a ligature is placed on both ends. *b,* The ligature is pulled through the orifice of the coronary stripper, and the stripper is advanced toward the aorta freeing the thrombus from the muscularis of the coronary artery. *c,* Illustration of coronary stripper employed for separation of the thrombus from the arterial wall. *d,* After complete separation of the thrombus from the arterial wall, traction is applied to the ligature, and the thrombus is removed from the artery. *e,* following removal of the thrombus, a sudden jet of blood escapes from the artery. (From Sabiston DC Jr: Coronary Endarterectomy. Am Surg 26:217-226, 1960.)

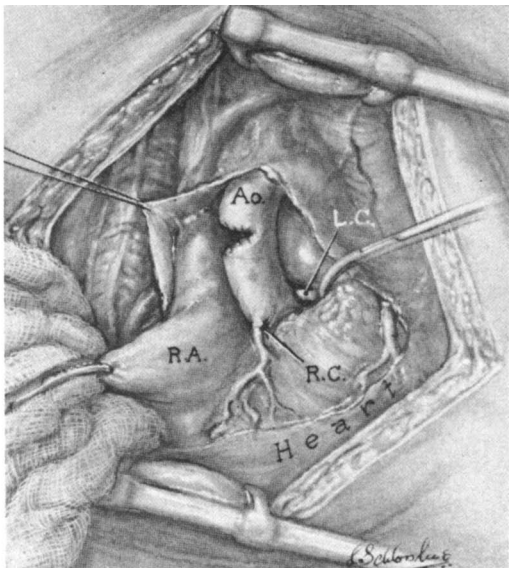


Fig. 9. Illustration of technique employed in the creation of a coarctation of the aorta a short distance above the orifices of the coronary arteries. *Ao.,* Aorta. *L.C.,* Left coronary. *R.C.,* Right coronary. *R.A.,* Right atrium. (From Sabiston DC Jr, Smith GW, Talbert JL, Gutelius J, Vasko JS: Experimental Production of Canine Coronary Atherosclerosis. Ann Surg 153:13-22, 1961.)

degrees of cardiac performance (Fig. 7). A number of additional studies with him then followed.[23-26] For this experience, and to Donald Gregg especially, I will remain ever grateful.

On return to Baltimore to assume the assignment that Dr. Blalock had originally given me, I was soon asked by the cardiologists to see a number of patients with severe angina pectoris. During a short period, a series of epicardial operations and Vineberg procedures were performed, but the results were disappointing. However, in 1958, Bailey[27] and, independently, Longmire[28] described direct coronary thromboendarterectomy. When Longmire published his early results, I felt that we should attempt this procedure, and we used this technique in a series of patients in Baltimore (Fig. 8).[29]

In 1962, Connolly and associates[30] reported successful transaortic endarterectomy of the right coronary ostium using cardiopulmonary bypass, and Effler and colleagues[31] did a similar procedure the same year. Our enthusiasm with endarterectomy, as well as that of others, was to greatly diminish, however, within a year or so because of the recurrence of anginal pain in a number of the patients, and when repeat arteriograms were performed there was evidence of occlusion of the previously endarterectomized site in most instances.

After our disappointment with endarterectomy, we turned attention to the basic problem of the pathogenesis of atherosclerosis and the effects of endarterectomy in healing of such lesions after surgical intervention. In order to achieve this, canine and later primate colonies were established for experimental production of severe experimental coronary atherosclerosis (Fig. 9).[32] Lesions quite similar to those in man developed in these animals. It was also interesting that with creation of experimental supravalvular aortic stenosis, such that the pressures in the proximal aorta rose as high as 300 mm Hg, the coronary lesions were much more severe (Fig. 10).

While the experimental studies were being conducted on the pathogenesis of atherosclerosis, I was fortunate in being able to obtain a Fulbright Research Scholarship to study in England at the University of Oxford. The work there was in association with the Department of Surgery with Professor Philip Allison, the Nuffield Professor of Surgery, and with Sir Howard Florey, the Sir William Dunn Professor of Pathology at Oxford. Florey had

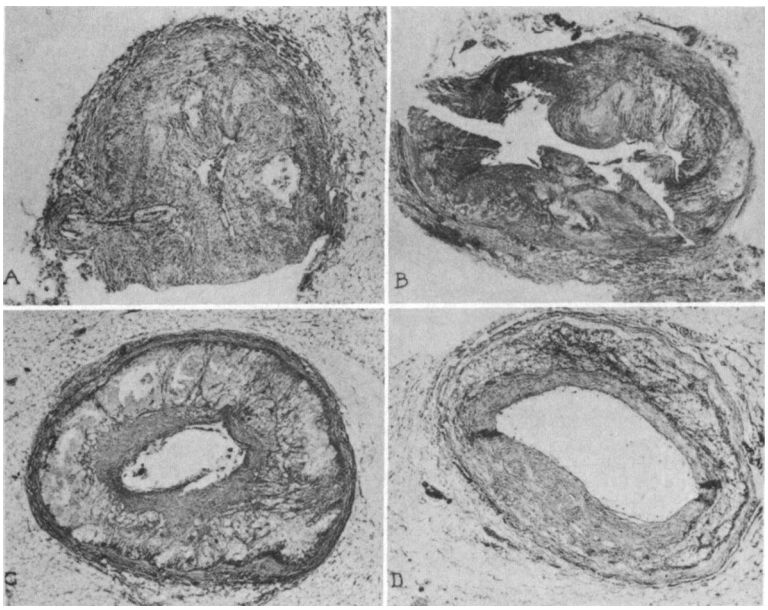


Fig. 10. *A,* Photomicrograph of right coronary artery showing total occlusion by atherosclerosis. *B,* Photomicrograph of left main coronary artery at its origin illustrating marked atherosclerosis with considerable diminution in the size of the lumen. *C,* Photomicrograph of anterior descending coronary artery showing marked atherosclerosis. *D,* Photomicrograph of circumflex coronary artery showing only minimal changes in this vessel. (From Sabiston DC Jr, Smith GW, Talbert JL, Gutelius J, Vasko JS: Experimental Production of Canine Coronary Atherosclerosis. Ann Surg 153:13-22, 1961.)

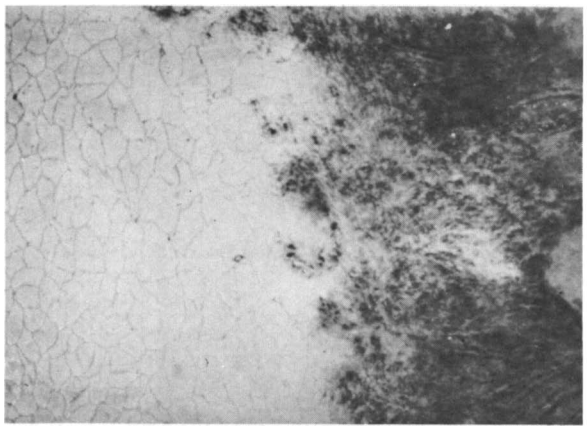


Fig. 11. On the *left* is the proximal aorta lined with normal endothelium. The section toward the *right* is the site of an endarterectomy, and endothelium is growing from the proximal aorta by cell division and spreading distally to cover the endarterectomized site.

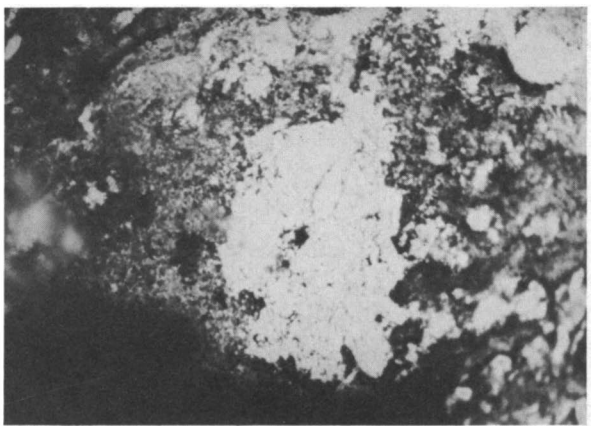


Fig. 12. Island of endothelium spreading inward to cover the endarterectomized site in the aorta.

expressed an interest in our studies on experimental atherosclerosis, and the work with his group was another unique and unparalleled privilege. Florey had created a distinguished laboratory in the Department of Pathology and had developed an outstanding research team originally committed to studies on antibiotics. For the fundamental work in penicillin he was awarded the Nobel Prize in 1945. However, at the time I was in Oxford, Florey had directed his research interest to the field of atherosclerosis and its effect upon the healing process. I worked with him and his associates, primarily John C. F. Poole, on endothelium and its growth in atherosclerosis and in the lining of prosthetic grafts.[33] In

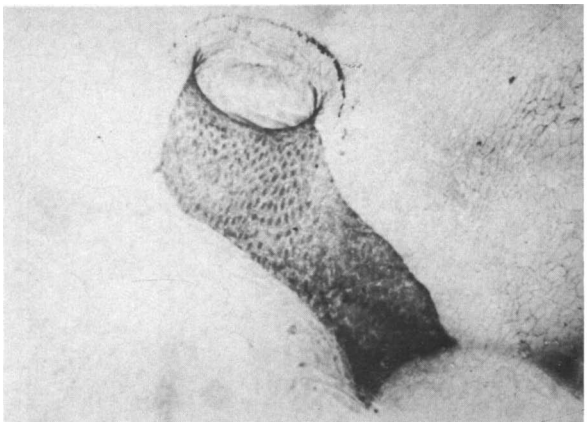
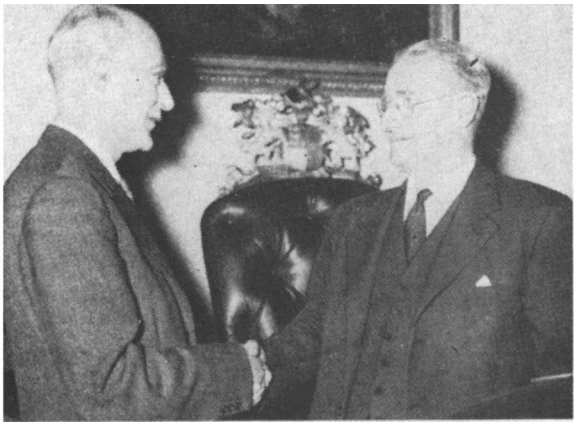


Fig. 13. A small branch entering the aorta; endothelium spreads concentrically to cover an endarterectomized site.

Fig. 14. Section of knitted Dacron graft covered with endothelium. The fibers and the pores in the graft can be seen.

these studies, it was found that endothelium usually spread by replicating itself cell by cell over the endarterectomized or grafted site. The endothelium grew from the native vessels proximally and distally (Fig. 11), ultimately meeting in the center of the graft or the endarterectomy site (Fig. 12). In addition, *islands* of endothelial growth in these specimens were observed, which enlarged concentricially and could be demonstrated to originate from vasa vasora spreading through the wall of the endarterectomized site (Fig. 13). The ingrowth of endothelium from vasa vasora was demonstrated by phase microscopy (Fig. 14). While I was working with Florey, he received the highest honor that can be accorded a British academician, namely, election to the presidency of the Royal Society (Fig. 15). I had already learned from him, as I had from Drs. Blalock and Gregg, that great men are basically very kind and



Sir Cyril Hinshelwood (left) with his successor, Sir Howard Florey, who was elected President of the Royal Society yesterday.

Fig. 15. Sir Howard Florey. (*The London Times*, December 1, 1960.)

Fig. 16. Anastomosis *(arrow)* of the end of the subclavian artery to the end of the circumflex coronary artery. At autopsy 5 months later, the anastomosis was patent. The remainder of the heart was normal. *R.V.*, Right ventricle. *L.V.*, Left ventricle. (From Sabiston DC Jr, Blalock A: Physiologic and Anatomic Determinants of Coronary Blood Flow and Their Relationship to Myocardial Revascularization. Surgery 44:406-423, 1958.)

thoughtful, and he consistently showed this characteristic to me.

Upon my return from Oxford a great contribution, *selective coronary arteriography*, had recently been introduced in the United States. It is interesting that the first coronary arteriogram was reported experimentally in 1932 by Rousthoi,[34] and he predicted at the time that

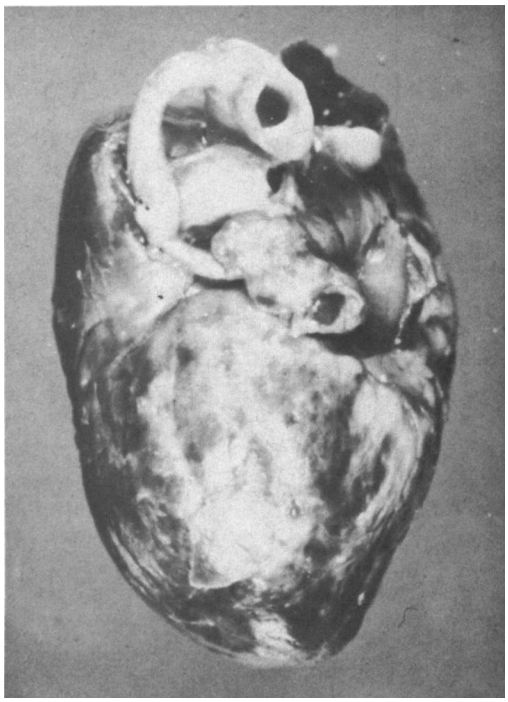


Fig. 17. Anastomosis of the end of the subclavian artery to the end of the circumflex coronary artery.

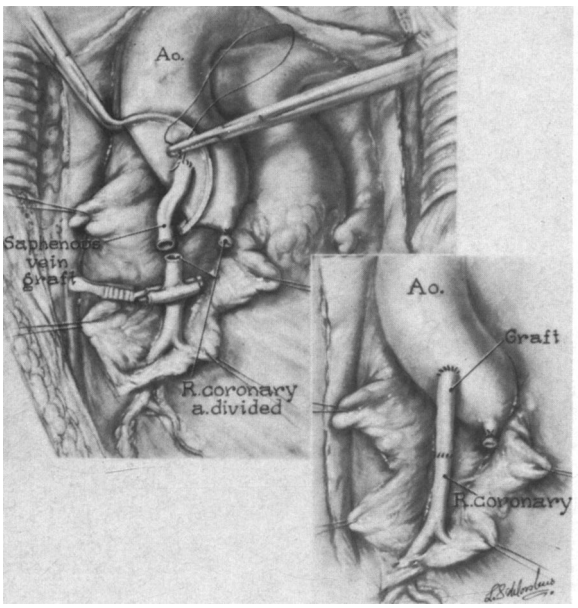


Fig. 18. Use of saphenous vein autograft anastomosed from the ascending aorta *(Ao.)* to the right coronary artery for proximal coronary arterial occlusion. (From Sabiston DC Jr, Ebert PA, Friesinger GC, Ross RS, Sinclair-Smith B: Proximal Endarterectomy, Arterial Reconstruction for Coronary Occlusion at Aortic Origin. Arch Surg 91:758-764, 1965.)

the technique would ultimately have clinical use. However, to Mason Sones[35] is due tremendous credit for the pioneering technique of *selective coronary arteriography,* and for this work he was subsequently chosen to receive the Lasker Award. The advent of coronary arteriography refocused attention on a *direct* surgical approach to the coronary circulation, and we and others turned attention to direct vascular anastomoses of the coronary vessels. In a series of experiments, the left subclavian artery of the dog was ligated distally and the proximal end was rotated inferiorly for direct anastomosis with the severed end of the left main coronary artery (Fig. 16). Similarly, the left subclavian was anastomosed directly to the circumflex coronary artery (Fig. 17).[36] After many studies on animals, and with the results usually showing patent anastomoses, we felt prepared to apply these principles clinically.

On April 5, 1962, we operated upon a patient with severe angina pectoris and total occlusion of the right coronary artery. A saphenous vein graft was obtained from the leg and anastomosed from the ascending aorta with the use of a partial occlusion clamp. This technique was illustrated at the time by Leon Schlossberg, the student of Max Brodel and the medical artist who has contributed greatly to all fields of surgery (Fig. 18).[37]

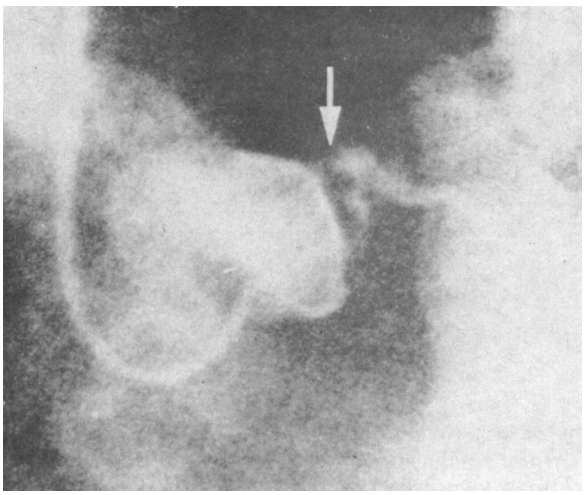


Fig. 19. Coronary arteriogram demonstrating severe stenosis of left main coronary artery. The catheter tip could not be made to enter the coronary artery because of the severity of the stenosis. Contrast medium injected into the aorta fills the left coronary artery. (From Sabiston DC Jr, Ebert PA, Friesinger GC, Ross RS, Sinclair-Smith B: Proximal Endarterectomy, Arterial Reconstruction for Coronary Occlusion at Aortic Origin. Arch Surg 91:758-764, 1965.)

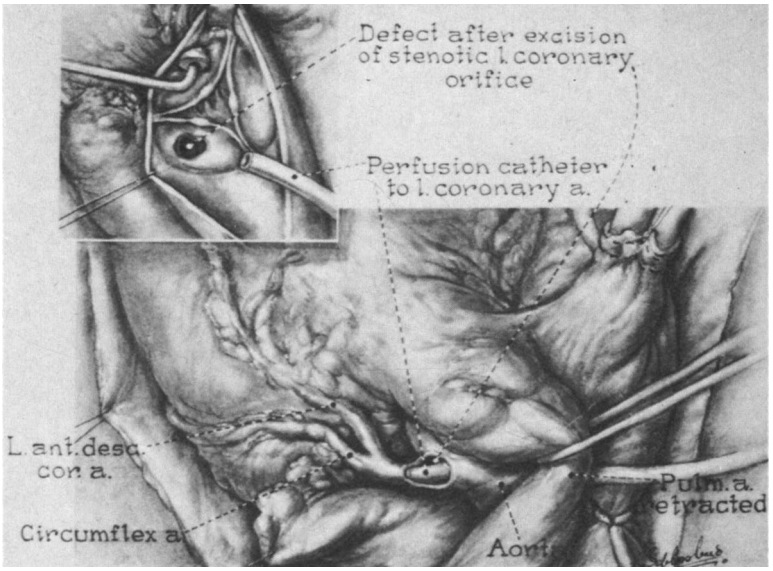


Fig. 20. Operative procedure performed for severe stenosis of left main coronary artery employing extracorporeal circulation. An aortotomy was made for excision of stenotic lesion from within the aorta. A coronary perfusion cannula was inserted for constant perfusion while the stenotic lesion was further managed by excision and insertion of a pericardial graft. (From Sabiston DC Jr, Ebert PA, Friesinger GC, Ross RS, Sinclair-Smith B: Proximal Endarterectomy, Arterial Reconstruction for Coronary Occlusion at Aortic Origin. Arch Surg 91:758-764, 1965.)

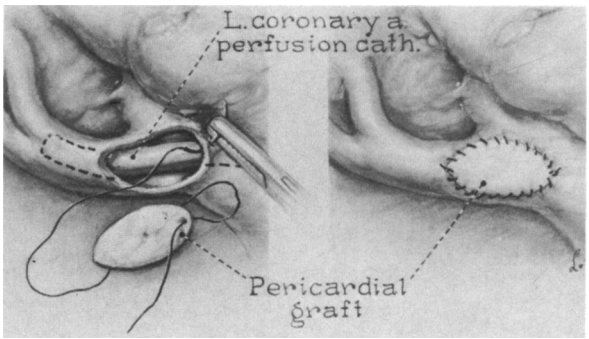


Fig. 21. Pericardial graft to left main coronary artery at site of endarterectomy. (From Sabiston DC Jr, Ebert PA, Friesinger GC, Ross RS, Sinclair-Smith B: Proximal Endarterectomy, Arterial Reconstruction for Coronary Occlusion at Aortic Origin. Arch Surg 91:758-764, 1965.)

We were quite pleased with the immediate results and were hopeful that this technique might open an entirely new approach in the management of such patients. Moreover, the cardiologists were also enthusiastic about its potential application. However, our optimism was dampened when the patient developed a cerebrovascular accident postoperatively and died. At autopsy thrombus was present at the aortic end of the anastomosis. We assumed that the embolus from the site was probably responsible for the cerebral event and unwisely did not pursue this procedure until several years later.

The first successful bypass for a left main lesion was performed by Garrett, Dennis, and DeBakey[38] in 1964. Great credit is due Favaloro[39] and Johnson[40] for bringing this procedure to full fruition, since both championed the coronary bypass procedure simultaneously in the late 1960s.

In 1964, we saw a patient with severe, unremitting angina and a 95% stenosis of the left main coronary artery (Fig. 19), who was in obvious need of an urgent procedure that would provide additional blood flow to the myocardium immediately. It was elected to correct the stenosis by placing the patient on cardiopulmonary bypass and, through a direct aortotomy, excising the lesion of the left main ostium from within the aorta (Fig. 20).[37] Our earlier experimental work had emphasized the necessity for continuous coronary perfusion to protect the myocardium, and it will be noted that both the right and left coronary arteries were continuously perfused while the procedure was being performed (Fig. 20). The stenosis in the left main coronary artery was an extensive one, and after excision a large defect on the anterior surface of the left main coronary artery was apparent (Fig. 21). Therefore, a pericardial graft was used to close this defect to prevent narrowing of the

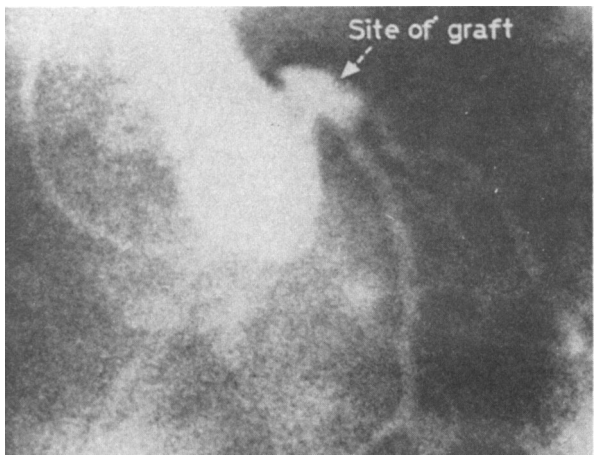


Fig. 22. Postoperative coronary arteriogram from patient demonstrated in Fig. 19, showing correction of the stenosis of the left main coronary artery. (From Sabiston DC Jr, Ebert PA, Friesinger GC, Ross RS, Sinclair-Smith B: Proximal Endarterectomy, Arterial Reconstruction for Coronary Occlusion at Aortic Origin. Arch Surg 91:758-764, 1965.)

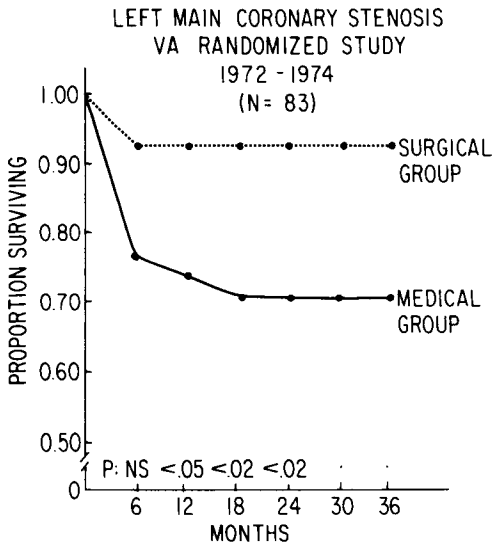


Fig. 23. Cumulative survival rates of 83 patients with significant left main lesions who were randomly allocated into medical or surgical treatment groups in 1972, 1973, and 1974. There were 41 patients in the medical group and 42 patients in the surgical group at zero time. (From Takaro T, Hultgren HN, Lipton MJ, Detre KM, Participants in the Study Group: The VA Cooperative Randomized Study of Surgery for Coronary Arterial Occlusive Disease. II. Subgroup With Significant Left Main Lesions. Circulation 54:Suppl 3:107-117, 1976.)

arterial lumen. Postoperatively, the patient did very well and a late postoperative arteriogram showed excellent patency of the left main coronary artery (Fig. 22). This patient has been followed at the Vanderbilt University Medical Center. The most recent report, received in 1984, indicated that there were no cardiovascular complaints, there were only nonspecific changes in the electrocardiogram, and there were no ventricular wall abnormalities seen on the radionuclide angiocardiogram. A personal telephone call quite recently, 21 years after the operation, revealed the patient to be asymptomatic and working daily in managing a business. This may be the longest survival of a direct coronary procedure, and one wonders if this technique might not be reconsidered for some patients with this lesion in view of the *late* results of occlusion of bypass grafts.

At that time we were unaware of the unfavorable prognosis of severe left main coronary lesions, but in 1972, J. Willis Hurst and I were asked to be the medical and surgical consultants to the Veterans Administration's randomized study conducted by Dr. Timothy Takaro and associates. The results were exceedingly impressive, and since then coronary bypass grafts have generally been recommended for significant left main lesions (Fig. 23).[41]

In recent years, the improvements in myocardial protection, the conduct of the anesthesia, the technique used for anastomosis, the choice of graft, the use of inotropic agents, and more complete revascularization have each contributed to a striking lowering of surgical mortality. A major advance was made in 1955 by Melrose and associates[42] with the introduction of potassium arrest. In retrospect, perhaps the high dosage employed and lack of sufficient topical hypothermia might have accounted for the discontinuance of the procedure at that time.

However, later the work of Bretschneider and colleagues[43] in Germany, Hearse, Stewart, and Braimbridge[44] in England, and Gay and Ebert[45] in this country each provided sound metabolic evidence of the advantages in placing each cardiac cell in diastolic arrest. Under these circumstances and in the presence of topical hypothermia, oxygen metabolism is reduced to a very low level. My colleagues and I[46] obtained data indicating that potassium alone achieves a great reduction in metabolism, but nevertheless it remains sufficiently high that it is clear that the metabolic activity of the heart must be further diminished by lowering the temperature of the heart (Fig. 24).

To Bigelow goes much credit for his original concepts of hypothermia so well expressed in his recent monograph entitled "Cold Hearts—The Story of Hypothermia and the Pacemaker in Heart Surgery." In this superb work, he[47] states: "What would be the effects of

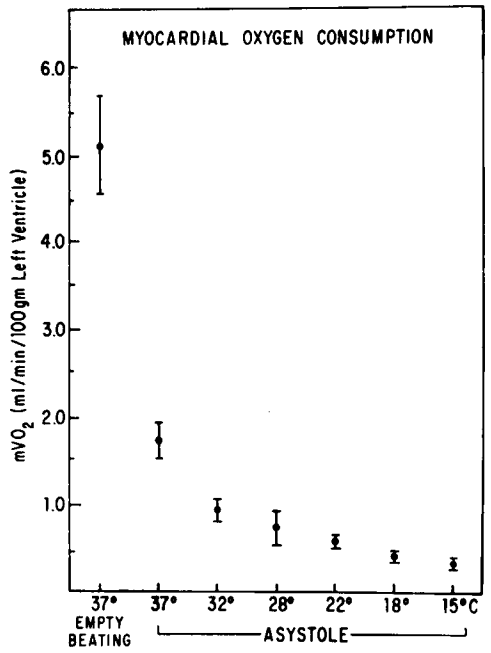


Fig. 24. Myocardial oxygen consumption *(mVO_2)* was determined for 11 dog hearts during empty beating normothermic conditions and following potassium arrest at several temperature levels (37° to 15° C). (From Chitwood WR Jr, Sink JD, Hill RC, Wechsler AS, Sabiston DC Jr: The Effects of Hypothermia on Myocardial Oxygen Consumption and Transmural Coronary Blood Flow in the Potassium-Arrested Heart. Ann Surg 190:106-116, 1979.)

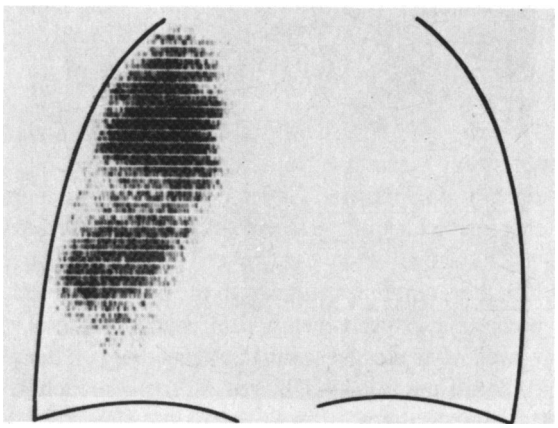


Fig. 25. Perfusion lung scan showing total absence of pulmonary flow to the left lung and with diminished pulmonary arterial flow in the right mid-lung field and at the right base. The level of the right hemidiaphragm is indicated. (From Sabiston DC Jr, Wolfe WG, Oldham HN Jr, Wechsler AS, Crawford FA Jr, Jones KW, Jones RH: Surgical Management of Chronic Pulmonary Embolism. Ann Surg 185:699-712, 1977.)

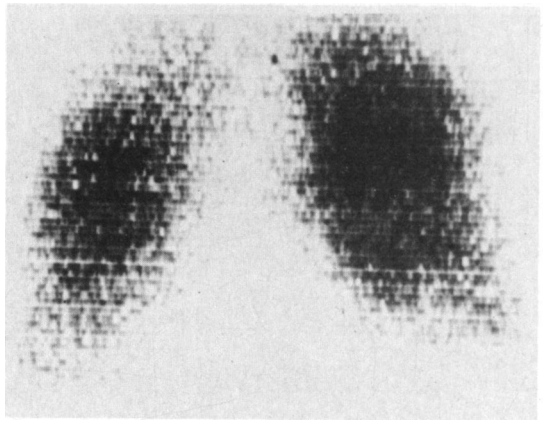


Fig. 26. Pulmonary scan showing good perfusion 8 years following pulmonary embolectomy. (From Sabiston DC Jr, Wolfe WG, Oldham HN Jr, Wechsler AS, Crawford FA Jr, Jones KW, Jones RH: Surgical Management of Chronic Pulmonary Embolism. Ann Surg 185:699-712, 1977.)

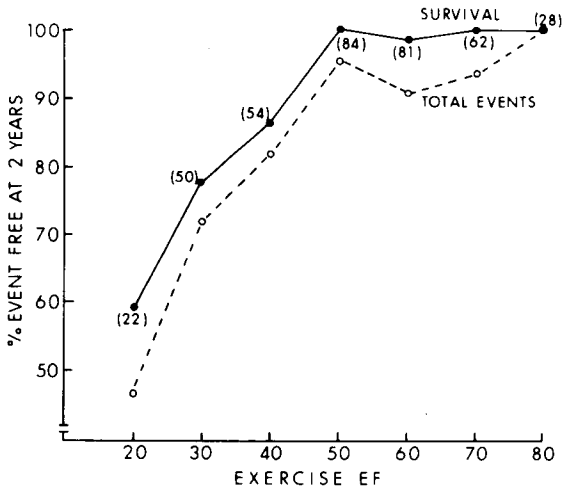


Fig. 27. Cardiac event-free probabilities in patients with a left ventricular ejection fraction during exercise greater than 50%. (From Pryor DB, Harrell FE Jr, Lee KL, Rosati RA, Coleman RE, Cobb FR, Califf RM, Jones RH: Prognostic Indicators From Radionuclide Angiography in Medically Treated Patients With Coronary Artery Disease. Am J Cardiol 53:18-22, 1984.)

general hypothermia on the metabolism of the body? I could hardly wait to return to Toronto in 1947 to investigate this simple and enchanting theory. The thinking may not sound particularly courageous today. However, one must realize that in those days a fall in body temperature was considered dangerous—something to be carefully avoided in surgery and in the

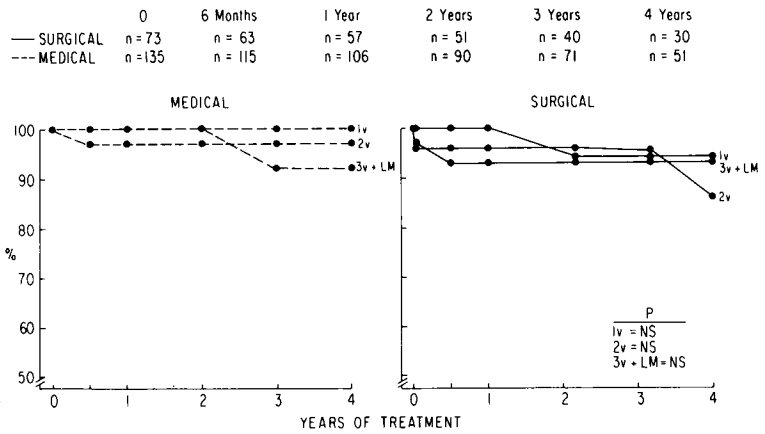


Fig. 28. Prognosis in 208 patients with coronary artery disease and no ischemia during exercise by radionuclide angiocardiography. (Courtesy of Dr. Robert H. Jones.)

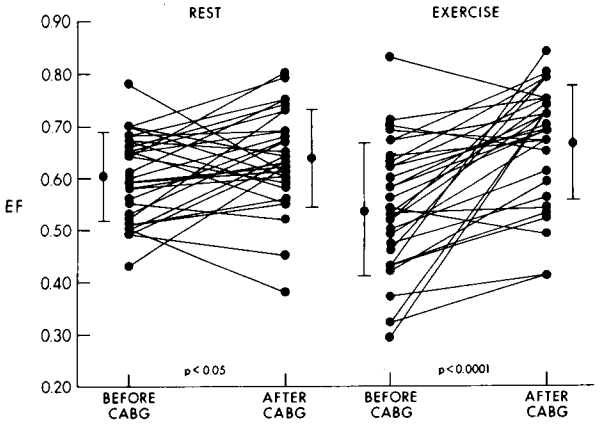


Fig. 29. Rest and exercise left ventricular ejection fraction *(EF)* before and after coronary artery bypass grafting *(CABG)* in 31 patients with no previous myocardial infarction and no perioperative QRS change. (From Floyd RD, Wagner GS, Austin EH, Sabiston DC Jr, Jones RH: Relation Between QRS Changes and Left Ventricular Function After Coronary Artery Bypass Grafting. Am J Cardiol 52:943-949, 1983.)

Fig. 30. Scinticor.

treatment of injury." His contributions will remain of epic proportions.

Many improvements have been made in the technical aspects, including more generalized acceptance of the fundamental contribution of Green[48] in 1970 emphasizing the superiority of the internal mammary artery as a graft, particularly in relation to long-term patency. This has been fully documented in several clinics, including a recent publication from the Cleveland Clinic.[49] In our institution, Rankin and associates[50] have performed some interesting recent studies using internal mammary grafts and have also demonstrated that early patency is quite high in addition to the fact that improvement in ventricular function can be demonstrated within several weeks following the operative procedure.

Among the recent advances in cardiac surgery, the fine work that has been established in the surgical approach to *intractable dysrhythmias* has been dramatic. Guiraudon, Harken, Cox, Lowe, and others have been pioneers in this field, and the results of therapy for

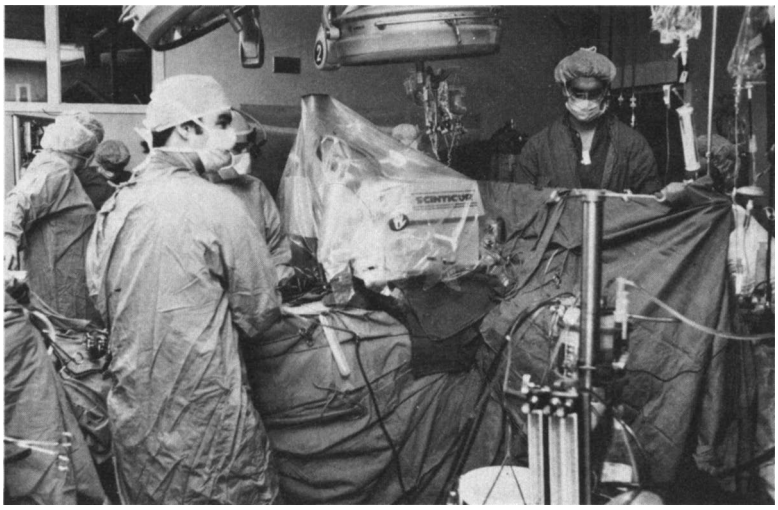


Fig. 31. Scinticor in the operating room.

ischemic ventricular tachycardia have been impressive.

Radionuclide angiocardiography has brought much objective, practical, and useful data in the daily practice of cardiac surgery. Moreover, its potential for the future is apt to be of monumental proportions, especially in view of some of the new technical advances that are currently available. Studies on performance of the left ventricle in normal and diseased hearts have been of paramount interest to cardiac surgeons, particularly in selection of patients for myocardial revascularization and in objectively assessing the postoperative results.

My interest in this field began 25 years ago while working in Oxford with Philip Allison, another teacher who taught me much and who became a close counselor and friend. During the course of studies on experimental pulmonary embolism it was apparent that a simple, safe, noninvasive, and relatively inexpensive assessment of the pulmonary circulation was much needed. On return from Oxford to Baltimore, I had the good fortune to work with Henry Wagner, a pioneer in nuclear techniques, in development of the pulmonary scan using radioactive microaggregates of human serum albumin.[51,52] After many studies in the experimental laboratory assessing its reliability and safety, the procedure was then employed clinically and has since remained the primary screening technique for the identification of pulmonary embolism in most instances.

In the past several years, my colleagues and I have had an increasing interest in *chronic, recurring pulmonary embolism* with progressive development of cor pulmonale and right ventricular failure. The radionuclide scan has been of considerable use in the preoperative diagnosis and postoperative follow-up of these patients. A preoperative film of a patient who had chronic occlusion of the entire left pulmonary artery and of much of the circulation to the right lower lobe is shown in Fig. 25, and a scan obtained 8 years later is shown in Fig. 26.[53] This patient is now well 17 years after the operation. Our experience with the surgical management of chronic pulmonary embolism has recently been reviewed with my colleagues, W. Randolph Chitwood and H. Kim Lyerly.[54]

One of our brightest and most effective members of the faculty, Robert H. Jones, was a medical student in Baltimore and worked with Henry Wagner and me in the laboratory at the time the pulmonary scan was being developed. Fortunately, Robert Jones chose Duke for his residency training, and he has become a world leader in the development of radionuclide angiocardiography. This technique provides accurate measurement of cardiac function and images of cardiac chambers at rest and especially during exercise. Dr. Jones has had an immense impact on the use of the radioactive angiocardiogram in assessment of a variety of cardiac disorders. One of his most interesting studies has been a multivariable analysis, which has documented the prognosis of 386 patients with coronary disease treated medically up to 4½ years. It was interesting that the *ejection fraction during exercise* was the *single* most important variable that could predict the survival of the patient as well as freedom from a future myocardial infarction. Moreover, recent data show this test to be more important than data about the coronary anatomy as seen on arteriograms in documenting the natural history of patients

Fig. 32. Surgical Staff, Duke University Medical Center, 1984-1985. (*First row, left to right*) Drs. R. D. Floyd, D. M. Estok, J. D. Sink, J. M. Williams, P. Van Trigt, R. C. Hill, B. D. Schirmer, D. C. Sabiston, Jr., G. K. Lofland, R. N. Jones, P. M. Thurlow, A. R. Scott, A. K. Das, S. M. Currin. (*Second row, left to right*) Drs. W. P. J. Peete, M. E. Kernberg, S. C. Lottich, D. C. Urquia, G. P. Fontana, A. P. Sanfilippo, D. P. Bolognesi, W. W. Shingleton, R. B. Peyton, W. L. Holman, H. Takai, O. E. Akwari, R. L. Hall, D. H. Harpole, Jr., H. R. Brown. (*Third row, left to right*) Drs. R. W. Leyen, T. E. Jordan, J. A. Spratt, M. W. Cloyd, F. S. Rotolo, D. M. Mahvi, D. S. Reintgen, M. Hensley, K. W. Small, M. K. O'Malley, T. B. Ferguson, Jr., D. J. Wermuth. (*Fourth row, left to right*) Drs. D. L. Stickel, L. G. Alexander, D. D. Glower, R. Silva, H. K. Lyerly, R. H. Jones, H. Xu, C. E. Murphy, T. D. Christopher, P-O. Hagen, W. G. Young, Jr., A. J. Colosimo, J. W. Gaynor. (*Fifth row, left to right*) Drs. R. J. Damiano, Jr., H. C. Filston, W. G. Wolfe, J. E. Lowe, S. J. Knechtle, G. W. Maier, B. A. Bowers, G. M. Stuhlmiller, V. A. Mantese, R. G. Cummings, H. J. Gerhard, C. L. Slingluff, Jr., R. L. Murrah, G. S. Leight, Jr., C. W. Cole, Y-F. Chen, J. E. Nitka, R. H. Cameron, D. Whitley, Jr.

with coronary disease. In 386 patients with a left ventricular ejection fraction greater than 50% during exercise, a very good *event-free* survival is seen (Fig. 27).[55] As the ejection fraction decreases below this level, there is a progressive worsening in prognosis such that patients with an ejection fraction of 20% during exercise had a 40% chance of death within 2 years. Moreover, these patients had a greater than 50% chance of myocardial infarction or death during this period. Such data are quite important in identifying patients likely to have a *stable course* in the future contrasted with those who are very apt to have *potential instability, complications, and death.*

In selecting optimal therapy for patients with coronary artery disease, the Duke computerized data base has been repeatedly utilized (Fig. 28). A group of 857 patients were identified who had *stable* coronary artery disease by coronary angiography. These patients were further subdivided into the 208 who showed *no ischemia during exercise* and compared with the 649 who *did* demonstrate ischemia during exercise. The survival curves show no difference in the outcome of *medical* or *surgical* therapy in patients with anatomic coronary disease who had *no* physiological ischemia by radionuclide angiography. Even patients with three-vessel and left main coronary disease did not benefit by bypass grafting if their preoperative radionuclide studies were normal. Moreover, relief of pain was *not* improved by coronary bypass in this subgroup of patients who had no documented physiological ischemia by scan. Therefore, one can draw the conclusion that exercise testing reveals potential problems based upon *physiological* alterations, whereas *anatomic* evidence alone is insufficient to predict outcome correctly.

Exercise angiocardiography can also be used to evaluate the influence of coronary artery bypass grafting on left ventricular function postoperatively as shown by the work of two of our residents, Richard Floyd and Erle Austin. In a study of patients following coronary artery bypass grafting, there is a very slight influence on *resting*

Fig. 33. Past Presidents of The American Association for Thoracic Surgery, 1918-1957.

function but a *dramatic influence* during exercise (Fig. 29).[56]

Another resident, Richard Peterson, studied a group of patients with Fontan procedures and showed that the cardiac index during exercise is not reduced in these individuals who do not have a functioning right ventricle. John Kirklin wrote this resident a highly complimentary letter which stimulated him to work even harder to further extend these basic observations. John Kirklin's thoughtfulness in praising work he regards as deserving is another of his many sterling characteristics. His contributions in introducing quantitative and objec-

Fig. 34. Past Presidents of The American Association for Thoracic Surgery, 1957-1985.

tive data in final assessment rank second to none and have set a high standard for everyone.

The use of radionuclide angiography in determining normal cardiac function during peak exercise has been quite revealing. One of our residents, Stephen Rerych, has attracted a group of world-class athletes to the laboratory for study of their cardiac function at rest and during peak exercise. As an example, he persuaded a Silver Medalist at the Montreal Olympics to have rest and exercise studies performed.[57] The heart rate rose from 51 at rest to 210 beats/min, the blood pressure increased as would be expected during exercise, and the ejection fraction, which was already a high 74% at rest, increased to an almost perfect 97% at peak exercise.

Moreover, the end-systolic volume was extraordinary in the left ventricle since it was 52 ml at rest with only 8 ml in the left ventricle at the end of systole during exercise. Finally, the cardiac output rose from 7.64 L/min at rest to an extraordinary level of 56.6 L/min at maximal exercise, the highest ever recorded in the literature.

In turning toward the future, through much hard work and dedicated commitment, and with the cooperation of the Baird Corporation, Robert Jones has recently been able to devise a new, highly refined multicrystal gamma camera, of which I am confident much will soon be heard (Fig. 30). This instrument can be used in the operating room to obtain measurements of cardiac function immediately after discontinuation of cardiopulmonary bypass and at varying times thereafter (Fig. 31). These data permit construction of pressure-volume loops for each individual heartbeat, from both the right and left ventricles. This type of measurement, which can be done early after coronary bypass grafting or other cardiac procedures, provides a new dimension of objective data available to the cardiovascular surgeon in the *immediate* situation in the operating room. This approach should be quite useful in providing objective end-points for clinical studies, including the effectiveness of myocardial preservation and the technical aspects of the operative procedure. It also may provide information concerning the clinical benefit that can be predicted following operation. Further studies are being conducted in the operating room and in the intensive care unit at this time on the important changes that occur immediately after surgical procedures.

Robert Jones is representative of the second period of my teachers and is an example of the faculty currently in the Department of Surgery at Duke who have been devoted colleagues in research and clinical work, and each is a close colleague and friend. They include H. Newland Oldham, Jr., Andrew S. Wechsler, Walter G. Wolfe, W. Glenn Young, Jr., Robert H. Jones, James E. Lowe, and J. Scott Rankin. Equally, in the Division of General Surgery, I owe much to my friends and colleagues who now are also my teachers including Onye E. Akwari, William G. Anlyan, R. Randal Bollinger, Howard C. Filston, Gregory S. Georgiade, John P. Grant, J. Dirk Iglehart, George S. Leight, Jr., Richard L. McCann, William C. Meyers, Joseph A. Moylan, William P. J. Peete, Raymond W. Postlethwait, Hilliard F. Seigler, William W. Shingleton, and Delford L. Stickel, as well as another group including R. Scott Jones, Samuel A. Wells, Jr., and Donald Silver, each of whom has left for promotion in an academic post elsewhere.

Next to my immediate family, those who have meant most to me are the residents who have trained in the Duke program and who are now my most recent teachers. To me this is a very important group indeed. They are all thorough gentlemen and close associates, and I feel confident they know how grateful I am to them. They are all teachers, both by instinct and design. During the past 15 years, 69 residents have completed the program in general or cardiothoracic surgery, and of these 54 currently hold university academic appointments and 10 are chiefs of service. At home, my primary interest centers around the trainees from the first year through the chief residency (Fig. 32). The spirit of inquiry in this bright group, their innovative minds, and their insistence upon objective data for conclusions rather than clinical impressions sharpen our ability as teachers, investigators, and clinical surgeons.

There is reason for some concern about the future of all our training programs, and our profession has a clear obligation to act thoughtfully, rapidly, and effectively to prevent any adverse economic or political consequences both for the quality of our training programs and for the proper training and well-being of our residents.

To gain strength to lead this venture successfully, we need to reflect upon those giants in our past, many of whom have been Presidents of this organization. During the past year, I have sought and located photographs of all the past Presidents of The American Association for Thoracic Surgery, among other reasons as an expression of my appreciation for your allowing me the privilege of being included in this group (Figs. 33 and 34). For this I express my lasting gratitude.

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