Historical Perspectives

The Vineberg Legacy

Internal Mammary Artery Implantation from Inception to Obsolescence

Joseph L. Thomas

At a time when cardiac surgery was still approached with hesitation, Arthur M. Vineberg developed the procedure of direct implantation of the internal mammary artery into the left ventricle for the relief of myocardial ischemia. The Vineberg operation, as it became known, had merit but never received broad endorsement from the medical and surgical communities. Its physiologic benefits were inconsistent and for years were documented by little more than anecdotal evidence, until coronary angiography (newly developed by Mason Sones) was able to demonstrate that the procedure did in fact increase perfusion in the diseased heart. This supporting evidence came rather late, for within the next decade direct aortocoronary artery bypass grafting overtook the Vineberg operation as a more efficient means of revascularizing the myocardium. Thousands of patients, however, had benefited from internal mammary artery implantation at a time when options were few; and the procedure was an aggressive move towards current (and similarly aggressive) treatments for myocardial ischemia. Moreover, the characteristics of the myocardium that Vineberg sought to exploit may form the basis for future therapy. A reappraisal of the implant is warranted, as today's physicians and surgeons inherit the last remaining recipients of Vineberg implants. ***(Tex Heart Inst J 1999;26:107-13)***

To have any other malady is to be sick;
to have this is to be dying.

—Lucius Annaeus Seneca (d. 65 A.D.),
in describing his own anginal symptoms[1,2]

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For centuries, ischemic heart disease has been a formidable enemy, known by its symptomatology and devastating effect. In 1947, when it appeared that social and medical advances had lessened the threat to human longevity posed by many diseases, George Bankoff[3] was prompted to write that "It now remained to combat the most dreaded heart disease which spares no human it attacks in middle or advanced age"—coronary artery disease (CAD). Arthur M. Vineberg, a Canadian, endeavored to answer that challenge by developing the 1st procedure that was documented to increase blood flow to the ischemic myocardium.[4,5] Although the astonishing progress made in cardiac surgery during the 1960s nearly consigned Vineberg's technique to the museum of curiosities, his internal mammary artery (IMA) implant proved to be a launching point for the treatment of CAD.

Work with Animal Models

In 1945, Vineberg (Fig. 1) began experimenting with IMA implantation in dogs.[6,7] In what would become known as the "Vineberg operation," he dissected the IMA free from the chest wall and pulled it into a tunnel created in the superficial myocardium.[7,8] The vessel was sutured directly into ventricular myocardium in the hope that it would arborize and develop communications with the native coronary circulation. It was his vision that he might create, in this manner, a "third coronary artery."

He founded his procedure on his belief that the myocardium contains relatively large venous sinusoids that would absorb the flow from the bleeding mammary vessels, but at the same time myocardial hemorrhage or rupture would be averted. Vineberg's procedure was a logical exploitation of the

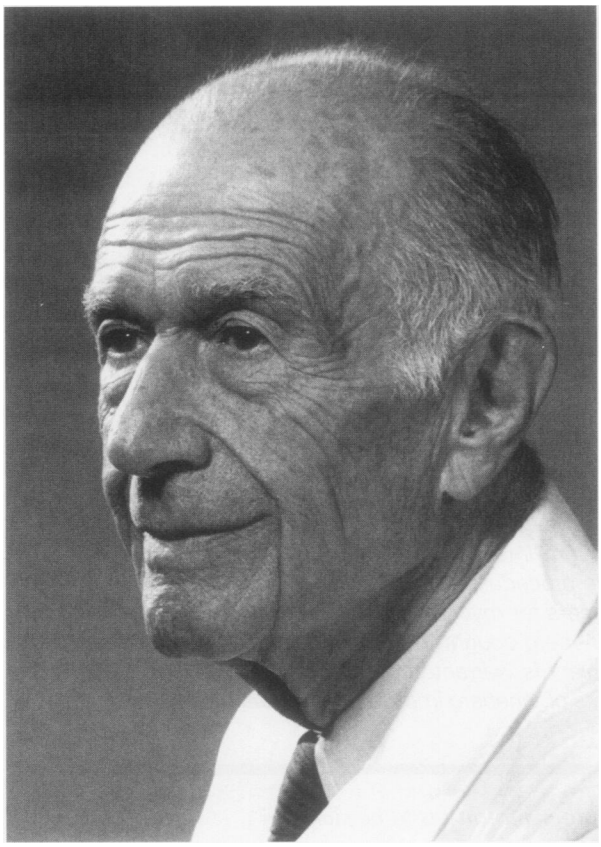

Fig. 1 *Arthur M. Vineberg (1903-1988)*

(Reproduced by courtesy of Royal Victoria Hospital, Montreal.)

small anastomoses known to exist between mammary arteries in the mediastinum and the epicardium.[9]

In the early series of tests conducted in dogs, he implanted varying lengths of the IMA with freely bleeding intercostal branches and terminal ligation of the main vessel. In the first 2 experimental groups, only 1 of 13 animals developed a communication between the grafted vessel and the coronary circulation as shown by injection of the IMA with radiopaque solution.[8] That solitary positive result provided enough inspiration to continue the trials.

By 1950, Vineberg had greatly refined his technique of implanting the IMA in dogs. He proved his theory that, in poorly perfused myocardium, the degree of collateralization could be increased by wrapping the test animal's left anterior descending (LAD) artery in cellophane; later, he used an amaroid constrictor to mimic the progression of atherosclerosis. Vineberg's implanted IMAs exhibited a collateralization rate of 75% in these experimental models. One month after implantation, he ligated the LAD to show that the grafted IMA could protect against myocardial infarction.[4]

Early Experience with the Vineberg Operation

In April 1950, Vineberg performed the 1st IMA implantations in human beings—which he reported the following year.[10] The left IMA was pulled into a myocardial tunnel parallel with the LAD (Fig. 2).[11] Although his 1st patient survived for only 62 hours after the procedure, postmortem examination revealed a patent IMA and no evidence of infarction, hemorrhage, or hematoma. The results of his 2nd attempt were more gratifying: the patient lived for 10 years after surgery.[12]

In the 1st decade of the Vineberg operation, enthusiasm was, at best, restrained. The emphatic renunciations of surgery on the heart by Theodor Billroth and Sir John Erichsen[13] might have been ringing in the ears of skeptics, but, more likely, the recent futile attempts at myocardial revascularization had taught the surgical community a lesson in caution. Throughout the 1950s, Vineberg's IMA implant procedure received limited use outside his own institution, and he remained one of its few dedicated proponents.[4]

Animal studies conducted concurrently outside of Vineberg's Montreal laboratory did not produce the same positive results. In fact, one study showed that only 16% of animal models formed graft–coronary communication. In defense, Vineberg noted that his critics had performed the IMA implantations on animals in possession of normal coronary circulation. In his own experiments, he had mimicked the atherosclerotic process. His contention was that an IMA

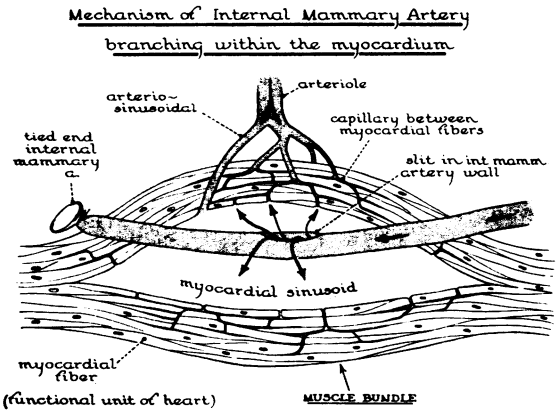

Fig. 2 *Drawing of implanted internal mammary artery in left ventricle with opening in side of vessel. Blood escapes from internal mammary artery into myocardial sinusoids, which is why the implanted vessel remains open until its own branches join the coronary arterioles.*

(Illustration and caption from: Vineberg A. Coronary vascular anastomoses by internal mammary artery implantation. Can Med Assoc J 1958;78:871-9. Copyright © by the Canadian Medical Association. Reprinted by permission of the publisher.)

implantation would be poorly received if it were in competition with normal coronary arteries.[4] Graft patency and collateralization were somehow dependent on tissue ischemia.

Vineberg in the Era of Early Surgical Experimentation

The Vineberg operation was a bold step in the direction of our contemporary treatment of CAD. The 1st half of the 20th century saw a number of surgical procedures intended to relieve angina pectoris, with or without increasing myocardial perfusion. In 1916, Jonnesco tried cervicothoracic sympathectomy in a human subject.[14] This procedure was thought to dilate the coronary vessels,[9] but it did little to increase blood flow. Sympathectomy did, of course, attenuate the chest pain that had served as a useful warning against exertion,[15,16] and forms of cardiac denervation remained in use as recently as the early 1960s.[9] Blumgart[9,17] followed a similar path by advocating, in 1948, iatrogenic hypothyroidism induced by radioactive iodine 131. Beck[18] and others advocated a combination therapy (the Beck I operation) of epicardial abrasion and irritation, grafting of pectoralis muscle and omental pedicles onto the heart, and partial ligation of the coronary sinus. Later, the Beck II operation involved an aortocoronary sinus vein graft with partial coronary sinus ligation.[1,16]

Perhaps nothing took more credibility away from Vineberg and all future invasive treatments for CAD than did the IMA ligation procedure. Ligation of the distal IMA was proposed to increase myocardial perfusion via the pericardiophrenic branch of the IMA. Introduced in the late 1930s by Fieschi[1,19] and championed by Zoja[4] and Glover,[20,21] this operation was later proved useless by both Glover[21] and a double-blind study.[1] This was one of the 1st demonstrations of the placebo effect of open surgery, and its results reduced the supportive value of symptomatic relief, as reported by patients who had undergone the Vineberg operation.

Vineberg parted ways with Beck and the others in one crucial regard. He knew that the solution lay in "a powerful new source of extracardiac blood . . . brought to the ischemic myocardium in such a manner as to bypass the occluded proximal portions of the major coronary vessels."[4] He concluded that the rival procedures merely created inflammatory channels that had neither longevity nor heavy flow. The Vineberg IMA implantation sought to take advantage of the myocardial sinusoids described by Wearn and associates in 1933.[22,23] Proof of true endothelium-lined sinusoids has remained elusive: digestion-casting and histologic study of the myocardial vasculature have yielded varying results. Nonetheless, in embryologic development, the heart possesses a primitive sinusoidal circulation that is later obliterated as the coronary system forms. The IMA implant is an aggressive attempt to use those remnants in the diseased heart; Vineberg proposed that these sinusoidal spaces would behave as a sponge for IMA run-off.[22]

In 1958, Vineberg reported experience with implants in 57 patients.[24] In that series, 40 patients suffered preoperatively from exertional angina, and the remaining 17 from angina at rest. In the former group, 68% (27/40) experienced no or slight pain at follow-up, and 80% (32/40) returned to work. Thirty of the 40 (75%) were alive at up to 7 years.[24] Vineberg cited both pain relief and exercise tolerance as evidence of efficacy. Results were less impressive for the group suffering from angina at rest, and only 24% (4/17) were alive at up to 7 years.[24] The Vineberg operation became a topic of boisterous debate among the medical and surgical communities.

The Discovery of Coronary Angiography and the Need for Proof

In 1958, Sones and Shirey developed selective coronary angiography[20,25]—a move that revolutionized cardiac care and gave Vineberg some respite from criticism. Cineangiography provided objective evidence of a sound physiologic basis for the operation.[26] In 1968, The Cleveland Clinic reported on the angiographic assessment of 1,100 IMA implants: the artery was patent in 92% of cases, and IMA–coronary artery communication was observed in 54% of patients.[4,27] Vineberg's was the 1st documented success in myocardial revascularization, and he was generous in his gratitude to the pioneering angiographers.[28]

Following the early angiographic studies, use of the IMA implant grew impressively: 10,000 to 15,000 procedures were carried out between 1958 and 1975.[4,29] From its inception, variations on the implant procedure were myriad. Vineberg[28] himself grafted the free omentum to the posterior left ventricle—a vestige of the Beck era. Vineberg's countryman, Wilfred Bigelow, left the free end of the artery patent,[30] and, during operation, resected the lower left stellate ganglion.[31] Effler[28] tunneled the IMA into the posterior wall. Sewell[28] used a graft consisting of the IMA, its corresponding vein, and chest wall tissue. Favalaro[32,33] performed a double implant with both the right and left IMAs. Vineberg[34] also described using the right gastroepiploic artery in the absence of a suitable IMA.[20] However, every advance that the Vineberg operation made was received with widespread skepticism.

The Vineberg operation had already served to afford some protection from anginal pain, and objective evidence mounted to support its ability to increase myocardial blood flow. Ochsner and associates concluded that, as Vineberg suggested, the success of the implant was dependent on the degree

of tissue ischemia. They found that the implants were patent 95% of the time when the LAD and circumflex arteries were greater than 75% stenosed. Conversely, there was a 25% patency rate when the coronaries were less than 75% stenosed.[35]

In a series of 55 patients in whom Ochsner and associates performed 73 IMA implants, the findings seemed additionally encouraging. On angiography, the IMA implants caused myocardial blushing or small vessel filling in 21% (15/73). There was filling of a major coronary artery in a further 42% (31/73).[35] The question remained whether the perfusion from the IMA implant was metabolically and therapeutically useful, and Vineberg attempted to answer it with a case-control study of angiographically-similar CAD patients.[36] The 4-year survival rate for the surgical group was 86.5%, compared with 53% and 60% for the 2 medically treated groups.[4]

In a 1966 publication on combining the IMA implant with coronary thromboendarterectomy, Viking Björk described the Vineberg operation as "at present, the best available alternative."[37] In 1966, Bigelow speculated that the IMA implant could serve as a prophylaxis in patients whose atherosclerosis had yet to invade the left coronary system.[30] Gorlin and Taylor[38] calculated that the operation afforded a 50% reduction in the reoccurrence of myocardial infarction, when outcomes were compared with those of controls with similar CAD.

Other studies were less reassuring. One implant follow-up discovered a 50% patency rate at 1 year and 58% mortality rate at 10 years.[12] The inconsistency of both the clinical and research results heralded a need for a large-scale, controlled clinical trial. In 1966, the Veterans' Administration sponsored such a study. Only 146 patients took part, and the operative mortality was 12%—much higher than in previous reports. Implant patency at 1 year was only 67%, and there was no difference in cumulative survival between study and control patients.[12] The VA trial was abandoned, and there would be no further opportunity to evaluate the Vineberg operation. It was 1968, and coronary artery bypass grafting (CABG) had taken center stage.

The Physiologic Basis of the Vineberg Operation

The magnitude of Vineberg's contribution to cardiac surgery is best appreciated in the context of the brave experimentation of the 1950s and 1960s. Through application of his broad knowledge of physiology, he became a trailblazing clinician driven by vivid memories of his own father's terminal battle with CAD. Although there was never a definitive dismissal or vindication of the Vineberg operation, it does appear to have benefited a great many people at a time when options were few.

Prior to 1900, there had been only 9 documented cases in which human arteries had been anastomosed, with establishment of flow. None had remained patent.[1] Even when Vineberg began his work in the 1940s, atherosclerosis was largely a mystery. Moreover, there was no hypothermic arrest, no modern imaging, and little precedent. When from today's perspective we see that the technical distance between IMA implantation and direct IMA–coronary artery anastomosis is small, and wonder why Vineberg did not take the extra step, we must remember that he did not consider the diseased coronary artery to be amenable to direct manipulation.

The idea that an implanted IMA, with or without distal ligation, could remain patent was a source of amazement to Vineberg's contemporaries and made them hesitant to embrace the procedure. Vineberg contended that the myocardium absorbed the run-off from the IMA, thereby preventing vascular thrombosis. In 1967, when Sparks investigated factors that appeared to affect graft patency in the Vineberg operation, he emphasized the value of an intact intima and speculated, further, that antithrombogenic factors could be at work.[6] Subsequent research[39] has indicated that damaged vessels do indeed liberate antithrombogenic factors and vasodilatory metabolites, which helps to explain the increased efficiency of Vineberg's implants in underperfused heart muscle.[35]

Tunneling the IMA into the substance of the myocardium took advantage of the compression-relaxation effect of the cardiac pumping cycle. When the free end of the IMA was left open, systole served to nearly empty the artery of blood.[6] It was also hypothesized that the rhythmic compression and agitation of the vessel's contents dispersed fibrin, thus preventing thrombosis.[22,40]

Questions remain about the physiologic consequences of IMA implantation: specifically, how much blood can the implant deliver and what is its contribution to myocardial metabolism? Direct and indirect flow measurements indicate that the flow rate of an IMA implant is highly unpredictable: impressive rates of up to 59 mL/minute are tempered by dismal reports of average flows well below 10 mL/minute.[4]

The pumping action of the myocardium upon the implanted artery was studied in an animal model.[6] Anterograde flow was observed during diastole and retrograde flow during systole. If one assumes that the tunneled portion of the IMA reliably fills with 0.1 mL of blood and that the heart rate is 72 beats/minute, the myocardial pump produces an alternating flow of 7.2 mL/minute in each direction.[6]

In a 1996 report[41] of follow-up of a patient who had a single left IMA implantation 23 years earlier, perfusion studies produced results generally supportive of Vineberg. The flow pattern was biphasic, with

a dominant diastolic component analogous both to the normal coronary circulation and to a directly-anastomosed IMA. The implanted IMA conveyed 70% as much blood as a directly anastomosed IMA. Under increased demand, the Vineberg graft was able to respond with an additional flow capacity of 60% over basal levels. In comparison, a directly-anastomosed IMA could increase its basal flow by 160%.[41] This demonstrates the Vineberg implant's capacity for adequate perfusion despite its attenuated responsiveness.

The aim of Vineberg's operation was to establish retrograde coronary perfusion via myocardial sinusoids and neovascularization. In support of this rationale, the heart of Vineberg's 1st patient revealed, at necropsy, a patent IMA and no hematoma around the implanted vessel.[12] From his early experiences with IMA implants at Hahnemann, Bailey remarked, "In no instance, human or animal, with intramyocardial implantation of an actively bleeding internal mammary artery have we ever observed any tendency toward hematoma formation within the myocardium."[11] He cited this as evidence that "the spongy structure of the myocardial wall renders it well-adapted to drain off the entire amount of additional arterial blood . . . introduced in this manner."[11] To show that these properties were unique to the myocardium, the investigators at Hahnemann implanted the femoral artery into the adductor muscle in a series of dogs. In all cases, there was significant hematoma formation, and all vessels were expediently thrombosed.[12]

In contrast, other investigators found that hematoma did occur, but that it was conducive to neovascularization. On histologic evaluation, Trapp and associates found evidence of hematoma around the periphery of the IMA implants. According to their observations, it was in the hematoma that small vascular channels arose and spread into surrounding viable tissue. This array of new channels resembled a hemangioma.[42]

The occasional high efficacy of Vineberg's operation has renewed interest in its potential. We now know that collateralization from the implant to the LAD is augmented by exogenous platelet-derived growth factor.[22] Advances in the understanding of angiogenesis give credence to the possibility of an "improved Vineberg"[43] in which the implant is aided by angiogenic growth factors, to yield consistently better outcomes. Such pharmacologic manipulation may well be the foundation for the next generation of revascularization procedures.

The novel transmyocardial laser revascularization (TMLR) procedure has disarming similarities to the Vineberg operation. Although the mechanism of TMLR is not entirely clear, both of the alternative physiologic explanations for its success call to mind the Vineberg operation. One explanation maintains that drilling holes in the myocardial wall enables blood from the cardiac chambers to access the sinusoidal "sponge" that is the myocardium[44]—which is to say that many small Thebesian-like tunnels accomplish what Vineberg did with a single large tunnel. The other explanation for TMLR's success is that the channels stimulate angiogenesis,[44] and this of course corresponds to the findings of Trapp and associates regarding the Vineberg operation.[42] These similarities almost ensure that proponents of TMLR will face much the same skepticism that Vineberg encountered.

The knowledge gained from the trial-and-error application of the Vineberg operation contributed to the growth of the procedure that superseded it—CABG. It was Vineberg who recognized the value of the IMA as an "expendable artery."[8] He understood, albeit superficially, the unusual properties that render the IMA resistant to thrombosis and atherosclerosis.[4,22,45] Vineberg established the IMA as a preferred high-flow conduit with which to connect the systemic circulation with a compromised coronary circulation. Undeniably, his experience catalyzed the early use of the IMA for direct CABG by Kolessov,[46] Goetz,[47] and others.[22,43,48]

For many well-founded reasons, current surgical practice does not employ the Vineberg operation. Nonetheless, there exist patients with severe, diffuse arterial disease, deeply-imbedded coronary arteries, and poor surgical fitness. Their arterial lesions may preclude angioplasty, and bypass surgery may be contraindicated. Because there are few therapeutic alternatives for such patients, it is not unreasonable to suggest that they would benefit from an evolved Vineberg-type procedure.

Living Proof: Coronary Artery Bypass in Vineberg Recipients

Some of the most intriguing support for Vineberg comes from cardiac surgeons who, even in the current decade, have inherited patients with Vineberg implants. There have been several reports of CABG in patients who had received IMA implants as long as 21 years earlier,[12,49] and this anecdotal evidence is both reaffirming and surprising.

Performing a coronary artery bypass on a patient with an old Vineberg implant is rife with complications. During preoperative angiography, the IMA graft may appear to be nonfunctional. Even in this circumstance, cardioplegic arrest can be difficult, because coronary flow may continue via collaterals from the IMA, despite aortic cross-clamping.[8,31,50] In such a case, the only way to stop the heart is to temporarily interrupt the IMA graft. This difficulty indicates, of course, that a Vineberg implant can maintain enough flow to support cardiac action.

Special care must also be taken during sternotomy in patients with a Vineberg implant. The grafted IMA may have shortened or developed adhesions in the anterior mediastinum, so that it is easily severed, which can result in arrhythmia and a high risk of intraoperative death.[35] Perioperative infarction in the zone of interrupted Vineberg implants has also been observed.[31] To avoid these pitfalls, the IMA implant is best left undisturbed (but not unheeded) during subsequent bypass surgery.[12]

The Vineberg Legacy

For millennia, it has been known that a palpable threat to vitality lurks in the mediastinum,[51,52] and many civilizations have attributed spiritual properties to the heart. Even within the 1st half of the 20th century, the heart was virtually off-limits to the surgeon. Now that this boundary is no longer within the recollections of most practicing physicians, it is difficult to understand the significance of Arthur Vineberg's contribution within its historical context. The Vineberg operation was not a tangent, but a stepping stone on the path toward the current "gold standard" of myocardial revascularization, coronary artery bypass grafting. More than any other pioneer of his era, Vineberg showed that the diseased heart requires a powerful supplementation of arterial blood. It is on that premise that today's cardiologists and surgeons aggressively and successfully treat their patients. It cannot be denied that the frustratingly inconsistent Vineberg operation was sometimes very effective: even a conservative summation of its beneficiaries would number in the thousands.

Coronary artery bypass surgery is an extraordinarily expensive treatment for an ordinarily common disease,[53] and there may come a time when this procedure is pushed into obsolescence by medical or transcatheter amelioration. Even so, the 20th century's truly dazzling advances in cardiac surgery will surely rank among the greatest feats of our time, when they are seen from the perspective of the millennium to come. Accordingly, historians cannot neglect such pioneers as Arthur Vineberg. We must remember, too, that progress in therapy can follow a circular path, when old therapeutic principles are given new applications. (Consider, for example, the current variations on Hunter's technique of exclusion for the treatment of popliteal aneurysms.) Vineberg's procedure set a precedent that still gives momentum to surgical progress.

References

1. Brewer LA III. Open heart surgery and myocardial revascularization. Historical notes. Am J Surg 1981;141:618-31.
2. Willus FA, Dry T. The heart and the circulation. Philadelphia: WB Saunders, 1948:15-7.
3. Bankoff G. The story of surgery. London: Arthur Barker, Ltd., 1947:223.
4. Shrager JB. The Vineberg procedure: the immediate forerunner of coronary artery bypass grafting. Ann Thorac Surg 1994;57:1354-64.
5. Sones FM Jr, Shirey EK. Cine coronary arteriography. Mod Conc Cardiovasc Dis 1962;31:735-8.
6. Sparks CH. Factors responsible for success of the Vineberg operation. An experimental study. Ann Thorac Surg 1967; 3:455-9.
7. Vineberg AM. Development of an anastomosis between the coronary vessels and a transplanted internal mammary artery. Can Med Assoc J 1946;55:117-9.
8. Dobell ARC. Arthur Vineberg and the internal mammary artery implantation procedure. Ann Thorac Surg 1992;53:167-9.
9. Mueller RL, Rosengart TK, Isom OW. The history of surgery for ischemic heart disease. Ann Thorac Surg 1997;63:869-78.
10. Vineberg AM, Miller G. Internal mammary–coronary anastomosis in the surgical treatment of coronary artery insufficiency. Can Med Assoc J 1951;64:204-10.
11. Bailey CP. Surgery of the heart. Philadelphia: Lea & Febiger, 1955:983-90.
12. Topaz O, Pavlos S, Mackall JA, Nair R, Hsu J. The Vineberg procedure revisited: angiographic evaluation and coronary artery bypass surgery in a patient 21 years following bilateral internal mammary artery implantation. Cathet Cardiovasc Diagn 1992;25:218-22.
13. Edlund Y. The triumph of complex operations. In: Haeger K. The illustrated history of surgery. Sweden: AB Nordbok, 1988:223-56.
14. Jonnesco T. Angine de poitrine guerie par la resection du sympathique cervicothoracique. Bull Acad Med Paris 1920; 84:93-102.
15. Drott C. The history of cervicothoracic sympathectomy. Eur J Surg Suppl 1994;572:5-7.
16. Marks C. A history of coronary artery surgery. South Med J 1973;66:249-53.
17. Blumgart HL, Riseman JEF, Davis D, Berlin D. Therapeutic effect of total ablation of normal thyroid on congestive heart failure and angina pectoris: early results in various types of cardiovascular disease and coincident pathological states, without clinical or pathological evidence of thyroid toxicity. Arch Int Med 1933;55:117-9.
18. Beck CS, Leighninger DS. Operations for coronary artery disease. JAMA 1954;156:1226-33.
19. Fieschi. Quoted by Battezzati M, Tagliaferro A, DeMarchi G. The ligature of the internal mammary arteries in disorders of vascularization of the myocardium. Minerva Med 1958; 46:1173.
20. Connolly JE. The history of coronary artery surgery. J Thorac Cardiovasc Surg 1978;76:733-44.
21. Glover RP, Kitchell JR, Kyle RH, Davila JC, Trout RG. Experiences with myocardial revascularization by division of internal mammary arteries. Dis Chest 1958;33:637-57.
22. Tsang JC, Chiu RC. The phantom of "myocardial sinusoids": a historical reappraisal. Ann Thorac Surg 1995;60:1831-5.
23. Wearn JT, Mettier SR, Klumpp TG, Zscthesche LJ. The nature of the vascular communications between the coronary arteries and the chambers of the heart. Am Heart J 1933; 9:143-64.
24. Vineberg A. Coronary vascular anastomoses by internal mammary artery implantation. Can Med Assoc J 1958;78:871-9.
25. Sones FM Jr, Shirey EK, Proudfit WL, Westcott RN. Cine-coronary arteriography. Circulation 1959;20:773-4.
26. Effler DB, Groves LK, Sones FM Jr, Shirey EK. Increased myocardial perfusion by internal mammary artery implantation: Vineberg's operation. Ann Surg 1963;158:526-34.

27. Fergusson DJ, Shirey EK, Sheldon WC, Effler DB, Sones FM Jr. Left internal mammary artery implant—postoperative assessment. Circulation 1968;38(4 Suppl):II24-6.
28. Vineberg AM. Discussion following: Favolaro RG, Effler DB, Groves LK, Sones FM Jr, Ferguson DJ. Myocardial revascularization by internal mammary artery implant procedures. Clinical experience. J Thorac Cardiovasc Surg 1967;54:359-68.
29. Preston TA. Coronary artery surgery: a critical review. New York: Raven Press, 1977:7-26.
30. Bigelow WG, Aldridge HE, MacGregor DC. Internal mammary implantation (Vineberg operation) for coronary heart disease: cineangiography and long-term follow up. Ann Surg 1966;164:457-64.
31. Goldman BS. The Vineberg procedure [letter]. Ann Thorac Surg 1994;58:1793-4.
32. Favaloro RG, Effler DB, Groves LK, Fergusson DJ. Revascularization of the left ventricle by double internal mammary artery implant. Geriatrics 1969;24:95-100.
33. Favaloro RG, Effler DB, Groves LK, Fergusson DJ, Lozada JS. Double internal mammary artery–myocardial implantation. Clinical evaluation of results in 150 patients. Circulation 1968;37:549-55.
34. Vineberg AM. Medical News Section. JAMA 1975;234:693-7.
35. Ochsner JL, Moseley PW, Mills NL, Bower PJ. Long-term follow-up of internal mammary artery myocardial implantation. Ann Thorac Surg 1977;23:118-21.
36. Vineberg AM. Evidence that revascularization by ventricular internal mammary artery implant increases longevity. J Thorac Cardiovasc Surg 1975;70:381-93.
37. Björk VO, Björk L. Internal mammary artery implantation for angina pectoris: angiographic evaluation of Beck and Vineberg procedures. Ann Surg 1966;164:236-42.
38. Gorlin R, Taylor WJ. Myocardial revascularization by internal mammary artery implantation: current status. JAMA 1969;207:907-13.
39. Moncada S, Gryglewski R, Bunting S, Vane JR. An enzyme isolated from arteries transforms prostaglandin endoperoxides to an unstable substance that inhibits platelet aggregation. Nature 1976;263:663-5.
40. Selmonosky CA, Ellison RG. Hemodynamics of the tunneled segment of a myocardial vascular implant. Ann Thorac Surg 1971;12:171-8.
41. Nasu M, Akasaka T, Chikusa H, Shoumura T. Flow reserve capacity of left internal thoracic artery 23 years after Vineberg procedure. Ann Thorac Surg 1996;61:1242-4.
42. Trapp WG, Burton JD, Oforsagd P. Detailed anatomy of early Vineberg anastomosis. J Thorac Cardiovasc Surg 1969;57:450-4.
43. Shrager JB. The Vineberg procedure [reply to Goldman BS, above]. Ann Thorac Surg 1994;58:1794.
44. Whittaker P, Rakusan K, Kloner RA. Transmural channels can protect ischemic tissue. Assessment of long-term myocardial response to laser- and needle-made channels. Circulation 1996;93:143-52.
45. Nguyen HC, Grossi EA, LeBoutillier M 3rd, Steinberg BM, Rifkin DB, Baumann FG, et al. Mammary artery versus saphenous vein grafts: assessment of basic fibroblast growth factor receptors. Ann Thorac Surg 1994;58:308-11.
46. Kolessov VI. Mammary artery–coronary artery anastomosis as a method of treatment for angina pectoris. J Thorac Cardiovasc Surg 1967;54:535-44.
47. Goetz RH, Rohman M, Haller JD, Dee R, Rosenak SS. Internal mammary–coronary artery anastomosis. J Thorac Cardiovasc Surg 1961;41:378-86.
48. Santos GH. Early use of internal thoracic artery [letter]. Ann Thorac Surg 1993;56:1438-9.
49. Hayward RH, Korompai FL, Knight WL. Long-term follow-up of the Vineberg internal mammary artery implant procedure. Ann Thorac Surg 1991;51:1002-3.
50. Salerno TA, Keith FM, Charrette EJ. Cardioplegic arrest in patients with previous Vineberg implants. J Thorac Cardiovasc Surg 1979;78:769-71.
51. Boisaubin EV. Cardiology in ancient Egypt. Tex Heart Inst J 1988;15:81-5.
52. Willerson JT, Teaff R. Egyptian contributions to cardiovascular medicine. Tex Heart Inst J 1996;23:191-200.
53. Treasure T. 25 years of cardiac surgery. Br J Hosp Med 1991;46:214-5.