Continuing Education

Perfusion of the Descending Thoracic Aneurysm

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Dissecting aneurysm of the aorta was first described by Morgagni[1] in 1961. Laennec[2] in 1819, referred to it as "aneurysme dissequant" or dissecting aneurysm. In spite of early recognition of this condition it was not until the turn of the century that Carrell[3] demonstrated successful resection of thoracic aorta in animals was possible. He repaired the resulting defects by vessel grafts. It was not until 1935 that the technique of fenestration[4] was tried. In 1948, Paullin and James used cellophane to wrap the aorta in an attempt to strengthen the wall, but the method proved unsuccessful.[5] Gross in 1948, first used aortic homografts clinically for replacement of defects in the thoracic aorta.[6]

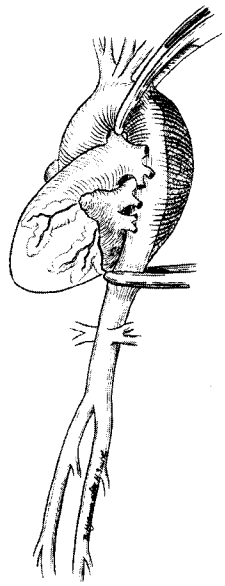
Thoracic fenestration was first described by DeBakey and associates in 1955.[7] *Fenestration* consists of creating a distal aortic re-entry so as to decompress the dissected aorta. There is reason to suspect that nonsurgical treatment may be more effective than fenestration in the treatment of aortic dissection. Juan, Beckwith, and Muller attempted *wrapping* the aneurysm with Orlon unsuccessfully.[8] In 1957, Cooley, et al.[9] and Gerbode and his associates utilized left atrial to femoral artery bypass during the excision of aneurysms of the descending aorta. Crawford[39] reports excellent results utilizing no shunts or partial bypass during resection and graft replacement (Figures 1 & 2). Other authors[5,4] advocate meticulous identification of the origin of the dissection and limiting graft replacement only to this area (Figure 3).

PATHOLOGY

The most common site of the thoracic aneurysm is the descending aorta just distal to the left subclavian artery (Figure 1). Less frequently the ascending aorta is involved as well as the aortic valve. Occasionally the aneurysm involves part or all of the aortic arch including the innominate and left carotid arteries. The aneurysm may extend and involve the abdominal aorta as a single dissection. Blood flow to the kidneys and splanchnic beds is often compromised.

Many investigators[2,4,11,12] believe that a tear or laceration in the intima initiates the dissecting process. Other investigators[13,14,15,16] support the 1920 observations of Krukenberg[17] that rupture of the vasa vasorum could initiate the dissection process. Bauer and Hirsch[18] stated that the repeated expansion and contraction of an aortic wall in which there is considerable difference in the elasticity of the media and adventitia layers would result in easy separation of these two layers. Because of unequal elasticity, the shearing force in the wall of the aorta at points of greatest mechanical stress produced dissection without intimal laceration. This explanation incorporates all theories of pathogenesis and explains established observations. The plane of dissection usually lies between the outer 2/3 and the inner third of the aortic wall.

Dissecting aneurysms predominate in males by a ratio of approximately 3:1 over females except in persons older than 80 years of age when this relationship is reversed. No age group is exempt; however, most authors report the highest incidence between the fourth and seventh decades. Hypertension is a common finding. Dissecting aneurysms in women are often associated with pregnancy (25-51%)[19,20,21] usually in the third trimester. Dissection is frequently associated with hereditary disorders particularly in the younger age group. The congenital cardiovascular disease includes Marfan's syndrome, Takayasu's disease, coarctation, bicuspid aortic valves, aortic hypoplasia, patent



1. Drawing of an aneurysm of the descending thoracic aorta. The aneurysm originates distal to the left subclavian artery. Occluding clamps have been applied above and below the aneurysm.

ductus arteriosus, interatrial septal defect, and tricuspid valve defects.[22-27, 8] Rarely a dissecting aneurysm follows trauma, and occasionally dissection arises in an atherosclerotic plaque. Cystic medial necrosis, syphilitic mesoaortitis, Cushing's disease, and pheochromocytoma have all been reported to be associated with dissecting aneurysm.

Resection and repair of aneurysms of the descending thoracic aorta involves cross-clamping the aorta. The clamp is usually applied just distal to the left carotid artery. During the period of aortic occlusion, the blood supply to the lower part of the body must be maintained *to prevent ischemic injury to the spinal cord* and distal organs and *to avoid acute left heart failure*. Ischemic damage of the spinal cord was not uncommon in the early days of excisional therapy of this disease. As early as 1944, Blalock and Park, had demonstrated ischemia of the spinal cord in 50% of the dogs following thoracic aortic occlusion.[28] Actually the first mention of this phenomena was in 1667 when both Stenonis[29] and Swammerdam[30] independently observed paralysis of the hindquarter in the rabbit after occlusion of the abdominal aorta. This observation is known as Stenonis' experiment.

Occlusion of the aorta may produce a distinct entity known as *the syndrome of the anterior spinal artery* or *the anterior spinal artery syndrome*. The Russian Preobrazhenskii is said to have originally described this syndrome in 1904;[31] however, a paper published by a Williamson on this subject appeared in the 1894 Lancet.[32] In 1909, Spiller presented this syndrome as a definite symptom-complex which could be diagnosed clinically. It consists of paraplegia, dissociated sensory (pain and temperature) loss below the upper level of the occlusion, and sphincter disturbances. Causes for the occlusion of the anterior median spinal artery include diseases of blood vessels; hypotension; hypotension with thrombosis; obstruction of the anterior median artery by tumors, intervertebral discs, bony spurs, and bone fragments; blood, air and fat emboli; and toxins.

In man, the duration of resistance to anoxia of the most sensitive structure of the spinal cord is unknown. Cooley[34] stated that 15 to 20 minutes of occlusion of the aorta is tolerable in man. Crafoord[35] stated that 27 minutes and Dubost[36] 30 minutes of aortic occlusion was permissable. Eiseman[37] thought that 52 to 60 minutes of aortic occlusion was safe while Adams and van Geerturyden[38] reported an occurrence of ischemic lesion after 18 minutes and 40 seconds. Crawford[39] reported a total aortic occlusion time of 64 minutes without paraplegia. He further states that other factors are more important in producing the anterior spinal artery syndrome. These factors are hypotension, removal of long segments of the aorta, and removal of aortic segments from which collateral circulation to the spinal cord had not had time to develop.

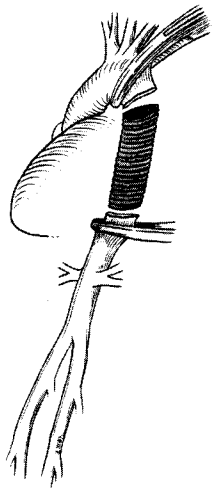


FIGURE 2.

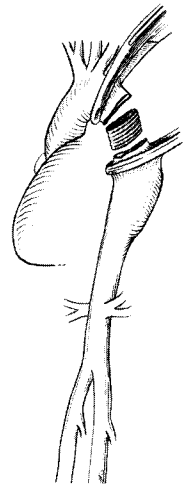


FIGURE 3.

2. Drawing showing a method of resection of a descending thoracic aneurysm and graft replacement. The entire aneurysmal area has been resected.

3. Drawing showing a method of repair of a descending thoracic aneurysm. The origin of the dissection has been identified and only this area has been resected.

ANATOMY

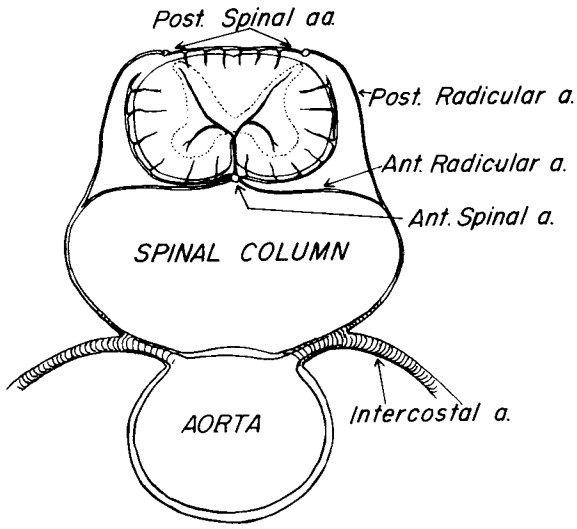
The blood supply to the spinal cord in the thoracic and lumbar regions is precarious at best. Blood is supplied to the spinal cord through three arteries. The arteries are the *anterior spinal artery* (anterior median spinal artery) and two smaller *posterior spinal arteries*. The anterior spinal artery is located along the anterior midline of the cord while the pair of posterior spinal arteries lie on either side posteriorly.

Various radicular arteries supply these cord vessels along its course through the spinal canal. In the cervical region, the cord vessels receive blood from the vertebral arteries, posterior inferior cerebellar arteries, and segmental branches. In the thoracic and lumbar regions radicular branches supply the cord. The radicular arteries are the anterior and posterior radicular branches of the spinal branch of the dorsal branch of the posterior intercostal artery (Figure 4). In the thoracic and lumbar regions these radicular branches supplying the cord vessels are scarce and the reserve blood supply is precarious. Between C 8 and C 9 only two small unilateral and inconsistently located small branches supply the long segment of the cord. Important branches contributing significantly to the anterior spinal artery are usually between T 11 and L 3. This artery is the *arteria radicularis magna* (major anterior radicular artery). This artery is the principle arterial supply of the lower 2/3 of the spinal cord. The anterior spinal artery provides the majority of the arterial blood supply to the spinal cord (75% of the cross-sectional area). The pair of posterior spinal arteries supply only a portion of the posterior columns and posterior horns (about 25%). Gillillan[40] in her fine presentation of the spinal cord blood supply indicates that the anastomoses between the anterior spinal artery and the posterior spinal artery are insufficient to sustain adequate circulation. Since the anterior spinal artery is in essence a chain of segmental arteries and adequate collateral circulation dues not exist within this one vessel, if the arteria radicularis magna is occluded, there is no collateral circulation available for the distal 2/3 of the spinal cord and neurologic sequlae is almost inevitable.

The extent to which collateral vessels have developed around the aortic lesion has some bearing on the tolerance to total aortic occlusion. Congenital coarctation, for example, is associated with a rich collateral circulation which begins to develop even before birth. An acute dissecting aneurysm on the other hand allows no time for collateral development.

HYPOTHERMIA

Various techniques have been employed in an effort to protect the spinal cord. Hypothermia was first used by Oudot[41] in aortic surgery. For a time general body hypothermia enjoyed wide spread popularity,[42,43] and indeed offered a measure of



4. Drawing of the blood supply to the spinal cord showing the anterior spinal artery and the posterior spinal arteries. The aorta, an intercostal artery and its branches are shown.

protection to the cord. The technique has been discarded because of the time involved for cooling, added complications, including ventricular fibrillation, respiratory complications in the elderly patients, interference with blood-clotting mechanisms and resulting post-operative hemorrhage, questionable predictibility and control, and because it did not prevent proximal hypertension.

SHUNTS

Numerous shunts have been used during surgical repair. These shunts have ranged from simple lucite intraluminal tubes,[44] to polyethylene cannula, Tygon tubing, Teflon tubes, graphite-benzalkonium-heparin (GBH) coated polyvinyl catheters,[39, 42-48] which were used to bypass the occluded area. These shunts were inserted into the aorta proximally and the aorta distal to the distal clamp or the femoral artery (Figure 5). One technique reports utilizing a Teflon graft connected end-to-side to the left subclavain proximally and end-to-side to the femoral artery distally.[46]

The use of shunts, though simple and convenient, presents several drawbacks. First, the shunt is usually cluttering the operative field. Difficulty may be encountered in placing the proximal cannula and excessive manipulation be required. Adequate flow distribution is not assured with the shunt technique and proximal hypotension may ensue. In most instances systemic heparinization is not required. Investigators differ on this point. Crawford[39] and others[44] believe that general heparinization increases bleeding, potentiates postoperative hypotension, increases transfusion, and hemorrhage in the pulmonary parenchyma. On the other hand, Dawes et al.[49] has demonstrated a protective effect of heparin on the cord, probably by inhibiting the formation of microemboli.

When the left subclavian artery is the source of the shunt, ischemia of the spinal cord may occur due to an *iatrogenic "subclavian steal."*[50] Since the anterior spinal artery system is derived from the vertebral arteries, a "subclavian steal," which reverses blood flow in the vertebral arteries may also reverse blood flow in the anterior spinal artery, thus causing ischemia of the cord.

LEFT HEART BYPASS

The classical perfusion method is that of the left heart bypass.[51, 52] The left atrium is cannulated and blood is returned to a reservoir or directly through a roller pump to the femoral artery (Figure 6 & 7). Though the left atrium is easily cannulated, the tubing is still in the operative field. If a reservoir is interposed between the roller pump and the femoral cannula, some provision must be made to prohibit excess gravity drainage from the left atrium. On the other hand, if the tubing is not large enough, to permit adequate

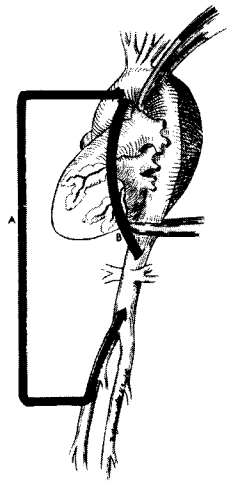


Figure 5.

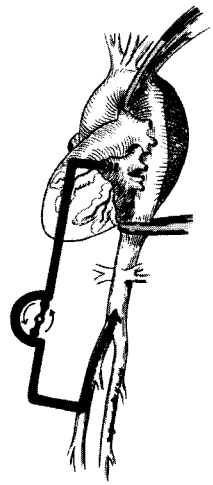


Figure 6.

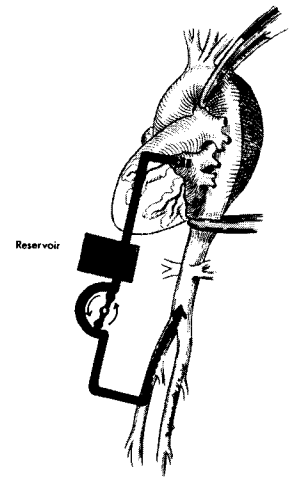


Figure 7.

5. Drawing of bypass shunts showing (a) shunt from the aorta to the femoral artery; and (b) aorta-aorta shunt which bypasses the operative area.

6. Drawing of the classical left heart bypass from left atrium to femoral artery.

7. Drawing of the left heart bypass set up incorporating a reservoir between the left atrium and the roller pump.

flow, left atrial hypertension and pulmonary edema may occur.[44]

If excessive negative pressure is generated, by either method, and if the seal about the atrial cannula is incomplete, small air bubbles may be aspirated into the left atrium and then through the roller pump into the femoral artery. It is unlikely that these small bubbles would be noticed unless they coalesce. These microemboli could cause retrograde ischemic damage to major organs or to the spinal cord. Cannulation may be carried out through the right atrium into the superior and inferior vena cavae thus providing means for total cardiopulmonary bypass (Figure 8). This technique involves extreme manipulation of the heart and leaves bypass tubing in the operative field.

FEMORAL—FEMORAL BYPASS

Another popular method of perfusion of the descending thoracic aneurysm is the femoral vein to femoral artery perfusion using a small oxygenator.[45, 53-55] This method offers several advantages over the other techniques reviewed. These advantages include: removal of tubing from the operative field, may be used fo resuscitation before thoracotomy, may be converted to total cardiopulmonary bypass if necessary, distal perfusion is independent of cardiac output, and since general heparinization and hemodilution are necessary with this technique, it minimizes microembolization and reduces blood sludging.

The technique is rather straightforward. The femoral artery is cannulated as is the femoral vein. The femoral vein cannula (French size #28-#32) is inserted and maneuvered high into the inferior vena cava to insure adequate return. The oxygenator is primed with 500 ml. to 1,000 ml. Ringer's lactate solution with 5% dextrose; the amount is of little consequence since conservation of fluid is not of prime importance. Flow rates of between 1,500 ml. per minute to 2,000 ml. per minute have been advocated and a minimum flow of 20 ml. per minute per kg. of body weight has been suggested (Figure 9).

It is entirely possible that these flow rates are higher than absolutely necessary to maintain renal function and prevent cord ischemia. A flow rate of 1,000 ml. per minute will produce a distal pressure between 40 and 60 mmHg. Investigators[56, 57] have demonstrated that a measurable glomerular function and urinary output may continue during the period of bypass if a minimal pressure of 30 mmHg is maintained. This is not to say that a lower perfusion rate is desirable, but rather acceptable if necessary (Figure 10).

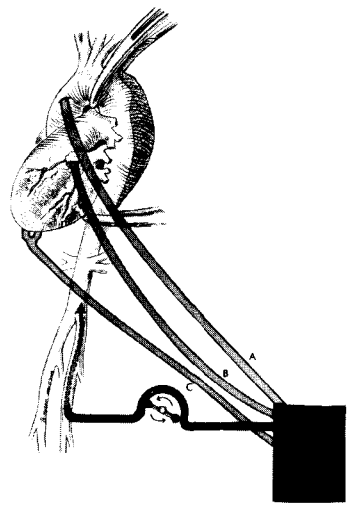
Of primary importance is the prevention of acute left ventricular strain and failure.

Some type of adjustable clamping device must be incorporated in the venous return line so that venous return may be regulated minute-to-minute in order to maintain a proximal systemic pressure at about 20 mmHg above pre cross-clamp pressure. Some patients are extremely brittle; the translocation of as little as 200 ml. is sufficient to change the proximal pressure from 80 mmHg to 150 mmHg. An alternative method is setting the arterial pump at the desired flow rate and regulating the systemic pressure by means of a second roller pump which controls the rate of venous return. In this technique, placement of the venous cannula is of extreme importance and caution must be used in manipulating the venous roller pump.

When no shunt or partial bypass is used, proximal hypertension and acute left ventricular failure become a medical problem. Proximal hypertension may be treated with narcotics, halothane, d-tubocurarine, sodium nitroprusside, or other adjuvants. Continuous proximal pressure monitoring is mandatory.

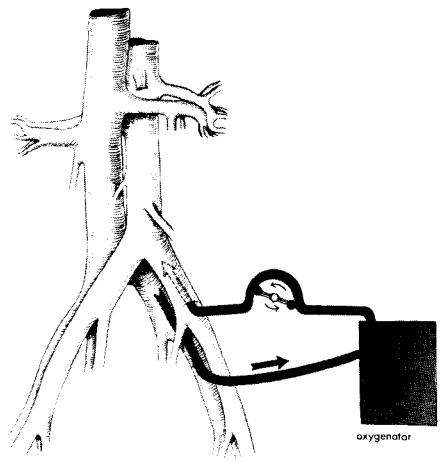
DECLAMPING SHOCK

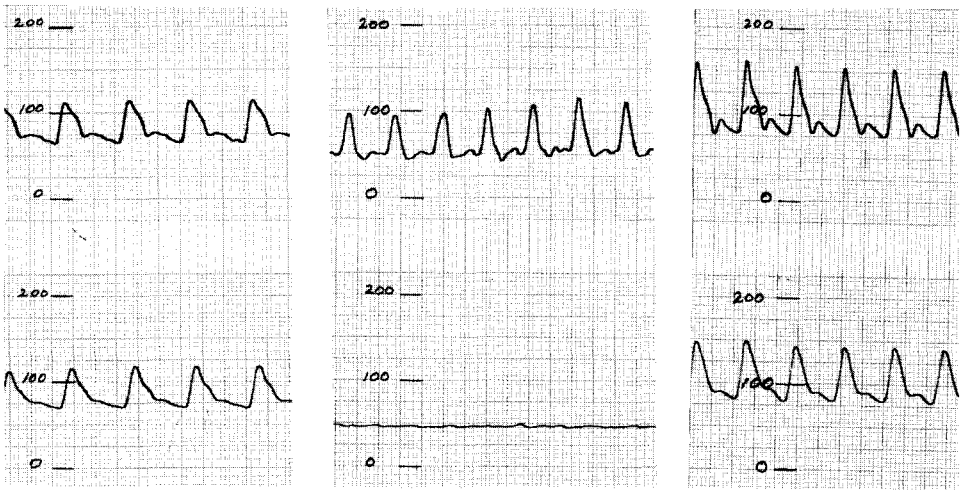
Irreversible hypotension and death following release of occlusion is not uncommon. This has been referred to as declamping shock. One popular hypothesis to explain



8. Drawing of bypass setup with reservoir showing cannulation sites: (a) ascending aorta, (b) left atrium, and (c) superior and inferior vena cavae via the right atrium.

9. Drawing of femoral-femoral bypass showing cannulation of the femoral vein, oxygenator, roller pump, and femoral artery.





10. Simultaneous proximal and distal arterial blood pressures. Indwelling catheters were placed in the radial artery and dorsal pedis artery. The pre cross-clamp tracing on the left shows the dorsal pedis artery (below) to be the same as the radial pressure (above). The center tracing, during cross-clamping and on femoral-femoral bypass at a flow rate of 1,200 ml. per minute, shows the distal pressure (below) to be about 45 mmHg. The tracing on the right was taken after surgical repair of the thoracic aneurysm and after removal of the cross-clamp.

this hypotension has been wash out of acid metabolities from ischemic extremities upon restoration of blood flow. The resulting acidosis was said to decrease cardiac output leading to myocardial, cerebral, and renal ischemia, all of which contributed to mortality. The other major hypothesis has been pooling of blood in the limbs immediately after declamping with decreased venous return, cardiac output and blood pressure. The use of cardiopulmonary bypass will usually prevent the acidosis and we have found it beneficial to overload the proximal circulation by about one liter of balanced electrolyte solution prior to declamping to prevent transient hypotension. This is of prime consideration since Crawford and others[39, 58] believe that hypotension is a principal factor in the development of the anterior spinal artery syndrome.

REFERENCES

1. Morgagni, G. B.: De Sedibus et Causis Morborum Per Anatomen Indagatis Libri Quinque, Venice, Ex Typog Remondiana, 1761, ep. 26, Art. 21: The Seats and Causes of Diseases Investigated by Anatomy. Translated by B. Alexander, London, A. Miller and T. Cadele, 1769, Cited by Sailer.

2. Laennec, R. T. H.: De l'auscultations mediate, ou traite due diagnostic des maladies des poumons et du coeur, fonde principalemente sur ce nouveau moyen d'exploration. Paris, J. A. Brosson & J. S. Chaude, 1819, Vol. 2, pg. 411.

3. Carrel, A.: On the Experimental Surgery of the Thoracic Aorta and Heart. *Ann. Surg.*, 52:83, 1910.

4. Gurin, D., Bulmer, J. W., and Derby, R.: Dissecting Aneurysm of the Aorta: Diagnosis and Operative Relief of Acute Arterial Obstruction Due to this Cause. *New York State J. Med.*, 35:1200, 1935.

5. Paullin, J. E. and James, D. F.: Dissecting Aneurysm of Aorta. *Postgrad. Med.*, 4:291, 1948.

6. Gross, R. E.: Coarctation of the Aorta. *Circulation*, 7:757, 1953.

7. DeBakey, M. E., Cooley, D. A., and Creech, O., Jr.: Surgical Consideration of Dissecting Aneurysm of the Aorta. *Ann. Surg.*, 142:586, 1955.

8. Warren, W. D., Beckwith, J., and Muller, W. H., Jr.: Problems in Surgical Management of Acute Dissecting Aneurysm of Aorta. *Ann. Surg.*, 144:530, 1956.

9. Cooley, D. A., Belmonte, B. A., DeBakey, M. E., Latson, J. R.: Temporary Extracorporeal Circulation in the Surgical Treatment of Cardiac and Aortic Disease. *Ann. Surg.*, 145:898, 1957.

10. Gerbode, F., Braimbridge, M., Osborn, J. J., Hood, M., and French, S.: Traumatic Thoracic Aneurysms: Treatment by Resection and Grafting With Use of an Extracorporeal Bypass. *Surgery,* 42:975, 1957.

11. Gore, I., and Seiwart, V. J.: Dissecting Aneurysm of the Aorta: Pathologic Aspects and Analysis of Eighty-Five Fatal Cases. *Arch. Path.,* 53:121, 1952.

12. Schattenberg, J. J., and Zisking, J.: Dissecting Aneurysm of Aorta. *J. Lab. & Clin. Med.,* 24:264, 1938.

13. Levinson, D. C., Edmeades, D. T., and Griffith, G. C.: Dissecting Aneurysm of the Aorta: Its Clinical, Electrocardiographic and Laboratory Features: Report of Fifty-Eight Autopsied Cases. *Circulation,* 1:360, 1950.

14. Tyson, M. D.: Dissecting Aneurysms. *Am. J. Path.,* 7:581, 1931.

15. Gore, I.: Pathogenesis of Dissecting Aneurysm of the Aorta. *Arch. Path.,* 53:142, 1952.

16. Whitman, R. G., and Stein, H. B.: Pathogenesis of Dissecting Aneurysms: Case of Dissecting Mesaortitis (Babes and Mironescu) Without Dissecting Aneurysm of Aorta. *Proc. Staff Meet., Mayo Clin.,* 16:41, 1941.

17. Krukenberg, E.: Eitrage que Frage des Aneurysma Dissecans. *Beitr. Path. Anat.,* 67:329, 1920.

18. Bauer, F. C., Jr., and Hirsch, E. F.: Spontaneous Longitudinal Cleavage of Wall of Aorta. *Arch. Path.,* 59:219, 1955.

19. McGeachy, T. E., and Paullin, J. E.: Dissecting Aneurysm of the Aorta. *JAMA,* 108:1690, 1937.

20. Schnitker, M. A., and Bayer, C. A.: Dissecting Aneurysm of the Aorta In Young Individuals, Particularly in Association with Pregnancy With Report of Case. *Ann. Int. Med.,* 20:486, 1944.

21. Mandel, W., Evans, E. W., and Walford, R. L.: Dissecting Aortic Aneurysm During Pregnancy. *New England J. Med.,* 251:1059, 1954.

22. Allison, P. R.: Successful Excision of a Dissecting Aneurysm of the Aorta Complicating Coarctation. *Proc. Roy. Soc. Med.,* 53:976, 1960.

23. Gore, I., and Seiwert, V. J.: Dissecting Aneurysm of the Aorta. Pathological Aspects. An Analysis of Eighty-Five Fatal Cases. *A.M.A. Arch. Path.,* 53:121, 1952.

24. Gore, I.: Dissecting Aneurysms of the Aorta in Persons Under Forty Years of Age. *A.M.A. Arch. Path.,* 55:1, 1953.

25. Hamilton, W. F., and Abbott, M. E.: Coarctation of the Aorta of Adult Type. *Am. Heart J.,* 3:381, 1928.

26. Hirst, E. E., Jr., Johns, V. J., Jr., and Kime, S. W., Jr.: Dissecting Aneurysm of the Aorta: A Review of 505 Cases. *Medicine,* 37:217, 1958.

27. Nielson, N. C.: Aneurysma Dissecans Aortae, 58 Tilfaelde i et Materiale Pa 33, 265 Autopsier. *Nord. Med.,* 65:778, 1961.

28. Blalock, A., and Park, E. A.: Surgical Treatment of Experimental Coarctation (Atresia) of Aorta. *Ann. Surg.,* 119:445, 1944.

29. Stenonis, Nicolai (Stensen, Niels): Elementorum Myologiae Specimen: Seu Musculi Descriptio Geometrica. Ferdinandum 11, Magnum Amstelodami Apud Johan, Janssonium a Waesbert and Viduam Elizei Weyerstraet, 1669.

30. Swammerdam, Jan: Tractatus Physico-an-atomico-medicus de Respiratione Usuque Pulmonum. Lugduni Batavorum, D. Abraham and A. a Gaasbeeck, 1667.

31. Preobrazhenskii, P. A.: Vascular System of the Human Spinal Cord. Pg. 224-254, 1904. Quoted by Levantovskii, M. I., In: Blood Supply of Central and Peripheral Nervous System of Man., Ed. by B. V. Ogney Moskow, 1950, Translated by P. V. DePorte at the National Institutes of Health, Bethesda, Md.

32. Williamson, R. T.: Spinal Thrombosis and Hemorrhage Due to Syphilitic Disease of the Vessels. *Lancet,* 2:14-16, 1894.

33. Spiller, W. G.: Thrombosis of the Cervical Anterior Median Spinal Artery; Syphilitic Acute Anterior Poliomyelitis. *Ibid.,* 36:601-613, 1909.

34. Cooley, D. A.: *Complications of Procedures on the Thoracic Aorta.* Artz, C. P., and Hardy, J. D.: *Complications in Surgery and Their Management.* W. B. Saunders Co., Chapter 31, Philadelphia, 1960.

35. Crafoord, C., Ejrup, B., and Gladnikoff, H.: Coarctation of Aorta. *Thorax.,* 2:121, 1947.

36. Dubost, Ch., and Dubost, Cl.: Traitement Chirurgical des Anevrysmes de l'aorte. Les Possibilites d'exerese. *J. Chir.* 69:581, 1953.

37. Eiseman, B., and Summers, W. B.: Factors Affecting Spinal Cord Ischemia During Aortic Occlusion. *Surg.,* 38:1063, 1955.

38. Adams, H. D., and van Geertruyden, H. H.: Neurologic Complications of Aortic Surgery. *Ann. of Surg.*, No. 4, 144:574, 1956.

39. Crawford, E. S., and Rubio, P. A.: Reappraisal of Adjuncts to Avoid Ischemia in the Treatment of Aneurysms of Descending Thoracic Aorta. *J. Thor. & Cardiovas. Surg.*, No. 5, 66:693, 1973.

40. Gillilan, L. A.: The Arterial Blood Supply of the Human Spinal Cord. *J. Comp. Neurol.*, 110:75, 1958.

41. Oudot, J.: La Greffe Vasculaire Dans Les Thromboses du Carrefour Aortique. *Presse Med.*, 59:234, 1951.

42. DeBakey, M. E., Henly, W. S., Cooley, D. A., Crawford, E. S., and Morris, G. C.: Surgical Treatment of Dissecting Aneurysm of the Aorta: Analysis of Seventy-Two Cases *Circulation*, No. 2, 24:290, 1961.

43. Hume, D. M., and Porter, R. R.: Acute Dissecting Aortic Aneurysms. *Surgery*, No. 1, 53:122, 1963.

44. Kahn, D. R., Vathayanon, S., and Sloan, H.: Resection of Descending Thoracic Aneurysms without Left Heart Bypass. *Arch. Surg.*, 97:336, 1968.

45. Dillon, M. L., Young, W. G., and Sealy, W. C.: Aneurysms of the Descending Thoracic Aorta. *Ann. of Thorac. Surg.*, No. 5, 3:430, 1967.

46. Inoue, T., Kawada, K., Tanaka, S., Sohma, Y., Fukuda, T., and Koyanagi, H.: Clinical Application of the Temporary Long External Bypass Method for Cross-Clamping of the Descending Thoracic Aorta. *J. of Thorac. & Cardiovas. Surg.*, No. 5, 63:787, 1972.

47. Nagel, C. B., and Williams, G. R.: Method of Repair in the Surgical Treatment of Aneurysms of the Descending Thoracic Aorta. *Am. J. Surg.*, 112:709, 1966.

48. Valiathan, M. S., Weldon, C. S., Bender, H. W., Jr., Topa, S. R., and Gott, V. L.: Resection of Aneurysms of the Descending Thoracic Aorta Using a GBH-Coated Shunt Bypass. *J. Surg. Res.*, 8:197, 1968.

49. Dawes, R. B., Lepley, D., Jr., Carey, L., and Ellison, E. H.: Heparin and Low Molecular Weight Dextran in Thoracic Aorta Occlusion. *Arch. Surg.*, 88:699, 1964.

50. Sabawala, P. B., Strong, M. J., and Keats, A. S.: Surgery of the Aorta and Its Branches. *Anesthes.*, No. 2, 33:229, 1970.

51. Cooley, D. A., DeBakey, M. D., and Morris, G. C., Jr.: Controlled Extra-Corporeal Circulation in Surgical Treatment of Aortic Aneurysm. *Ann. Surg.*, 146:473, 1957.

52. Gerbode, F., et al: Traumatic Thoracic Aneurysms: Treatment by Resection and Grafting with the Use of Extracorporeal Bypass. *Surgery*, 42:975, 1957.

53. Neville, W. E., Cox, W. D., Leininger, B., and Pifarre, R.: Resection of the Descending Thoracic Aorta with Femoral Vein to Femoral Artery Oxygenation Perfusion. *J. Thorac. Cardiovas. Surg.*, No. 1, 56:39, 1968.

54. Austen, W. G., and DeSanctis, R. W.: Dissecting Aneurysm. *Surg. Clin. N. Amer.*, 46:573, 1966.

55. Bloodwell, R. D., Hallman. G. L., and Cooley, D. A.: Partial Cardiopulmonary Bypass for Pericardiectomy and Resection of Descending Thoracic Aortic Aneurysms. *J. Am. Med. Assoc.*, 205:479, 1968.

56. Morris, G. C., Jr., Moyer, J. H., Cooley, D. A., and Brockman, H. L.: The Renal Hemodynamic Response to Hypothermia and to Clamping of the Thoracic Aorta with and without Hypothermia. In *Surgical Forum*, Philadelphia, W. B. Saunders Co., 5:219, 1954.

57. Morris, G. C., Jr., Witt, R. R., Cooley, D. A., Moyer, J. H., and DeBakey, M. E.: Alterations in Renal Hemodynamics During Controlled Extra-Corporeal Circulation in the Surgical Treatment of Aortic Aneurysms. *J. Thorac. Surg.*, 34:590, 1957.

58. Zuber, W. F., Gaspar, M. R., and Rothschild, P. D.: The Anterior Spinal Artery Syndrome —A Complication of Abdominal Aortic Surgery: Report of Five Cases and Review of the Literature. *Ann. Surg.*, No. 5, 175:909, 1970.