Neurologic Complications of Aortic Surgery *

HERBERT D. ADAMS, M.D.,

Department of Surgery,

HERMAN H. VAN GEERTRUYDEN, M.D.†

Former Fellow in Surgery, The Lahey Clinic, Boston, Massachusetts

IN THE LAST ten years operative procedures on the aorta have developed into a well established field in surgery. The different technical, physiologic and hemodynamic aspects are now fairly well defined. Surgical treatment of congenital and acquired lesions is now carried out from the ascending aorta to its bifurcation. The problems arising from the acute interruption of the circulation have been the most important factors in the relatively late development of this branch of surgery. It is amazing to consider how few difficulties were encountered by interrupting the circulation, and once undertaken, the solution of most aspects of the problem has been relatively easy.

The spinal cord, however, is the one organ on which the effects of vascular occlusion remain unsolved. This is partly because this tissue is the most sensitive to vascular occlusion but mainly because the exact mechanism of effects of the occlusion of the aorta on this organ is unknown. Although in those cases in which damage results, the complications are not lethal, they may lead to permanent and crippling infirmity which may be out of proportion to the seriousness of the disease for which operation was undertaken. Spinal complications have been reported following all types of aortal operations but a knowledge of their true incidence is still lacking and as in all new fields of surgery there has been a tendency to consider the outstanding successes rather than the few failures.

Although these complications are now fairly well known, the exact reason for their occurrence in individual cases is difficult to explain. In fact, when these accidents occur the operative conditions are identical to those in many similar but successful cases, and so a circulatory abnormality in the spinal cord or a predisposing or previously latent pathologic condition is thought responsible for these unexpected complications. The anatomic and physiologic background in these cases of spinal complications is complex. Understanding is difficult because of both the interaction of so many structural and dynamic variables in the circulation and incomplete knowledge of the reactions of nervous tissue to anoxia. As a result there seems to have been a tendency toward dogmatism in the consideration of the problem of postoperative neurologic deficit. Attention concerning its causation has been focused mainly on the act of occluding the aorta. Because of the protecting extensive collateral circulation, nervous tissue damage during operations for congenital aortic lesions is considered unlikely; also, in resectional surgical procedures with or without the use of a shunt, the effects of technically necessary occlusion of segmental vessels to the spinal cord are not fully appreciated.

Again the tendency has been to consider the neurologic deficit to originate only from damage to the spinal cord so that little at-

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† Present address, Antwerp, Belgium.

tention has been given to the possibility of involvement of the extraspinal nervous system. The increasing number of postoperative complications of operations for congenital and acquired lesions of the aorta reported in the more recent literature proves that our concepts have not kept pace with the progress made in this branch of surgery; the necessity for a rational explanation becomes apparent. It was felt, therefore, that it might be of interest to review the extensive literature related to this subject to endeavor to obtain a better understanding of anatomic and neurophysiologic factors causing these complications. As yet no specific description exists of the different aspects of the neurologic picture of medullary ischemia, while little is known about the picture of extraspinal neurologic complications. The possibility of similarity between the two which may contribute to an erroneous diagnosis of spinal etiology makes a description of both necessary. Many studies have been devoted to the prevention of these serious neurologic complications by searching for technics to prevent the effect of occlusion by shunt and injections distal to the occlusion to increase the oxygen level and pressure, and recently by lowering the metabolism of the nervous tissue by hypothermia. Successful application of local or general hypothermia is no longer in the experimental phase. As hypothermia has yet to prove itself completely effective in man and as experimental conditions are not always identical to those at operation, the possibility of neurologic damage occurring during certain types of procedures will remain until some means is found of giving complete and rationalized guarantee for its prevention on anatomic and physiologic grounds.

GENERAL CONSIDERATIONS

The ischemic type of neurologic phenomena under consideration may be caused by any occlusion of the circulation from the aorta to the nervous system resulting from aortic surgery. Procedures on the aorta are a recent field of surgical progress, but the neurologic complications of occlusion of the aorta are an old topic of investigation and discussion. The first mention of this phenomenon was made in 1667 when both Stenonis [103] and Swammerdam [107] observed paralysis of the hindquarter in the rabbit after occlusion of the abdominal aorta. This observation is known as Stenonis' experiment. The etiology of this paralysis remained obscure and was explained alternatively as ischemia of the medulla or of peripheral nerves until Schiffer [98] in 1869 proved both etiologic possibilities. He discovered that in the dog the determining factor for the central or peripheral origin was the difference between suprarenal or infrarenal levels of aortic occlusion. In 1899 Hoche [60] explained that Stenonis' experiment as seen in the rabbit was impossible in the dog because of the different spinal circulation.

At the end of the nineteenth century a method was found for producing elective destruction in the central nervous system in Stenonis' experiment for the purpose of studying the pathology of nerve cells. The neuropathologic findings of these studies form the basis of our present knowledge of ischemia of the spinal cord. In man, neurologic complications after aortal occlusion were an early subject of much discussion and controversy. In the older literature on the etiology of neurologic damage after saddle embolism many authors accepted a peripheral nerve ischemic origin but others were influenced by the experimental findings in the rabbit and defended a spinal origin in such cases. This may have delayed the development of abdominal aortic surgery. Occlusion of segmental vessels of the aorta by a dissecting aneurysm resulting in damage to the cord has kept up the early interest in this subject. With the discovery of the operability and possibility of cure of aortic embolism in 1913 by Bauer,[12] interest in this problem was lost until the advent,

in the last decade, of aortic surgery for congenital lesions. It has gained impetus recently with the development of surgery on acquired lesions of the abdominal and thoracic aorta, in 1951. Stenonis' observation, which for 300 years had been of experimental value and of scientific interest in aortic embolism and dissecting aneurysm, has now become of great practical importance in its application to man.

The part of the nervous system which in man is supplied by the aorta distal to the left carotid artery, and now limits occluding procedures of the aorta, includes the entire spinal cord except for part of the cervical cord, the nerve roots, dorsal ganlia and peripheral nerves. These parts of the nervous system can be divided into spinal and extraspinal parts which vary in their dependence upon the circulation by difference in sensibility to ischemia. The lower limit of the important aortic branches to the spinal cord varies from the level of the eighth thoracic to the fourth lumbar. With this in mind it is possible to divide the aortic blood flow into two parts: one part below the lower level of the spinal branches supplying only the peripheral nervous system and one part above that lower level supplying both the central nervous system (by the blood flow supplying the spinal branches) and the peripheral nervous system (by the blood flow going to the distal aorta) (Fig. 1). As aortic surgery is now carried out over the entire length of the aorta, the circulation to the nervous system may be interfered with in three ways depending on the site of occlusion: (1) to the central and peripheral nervous systems by occluding the aorta above the level of the lowest spinal branch, (2) to the central nervous system alone by occlusion of the spinal branches only, and (3) to the peripheral nervous system alone distal to that point. In the first eventuality the circulatory occlusion will cause a central lesion long before a peripheral nervous lesion develops because of the difference

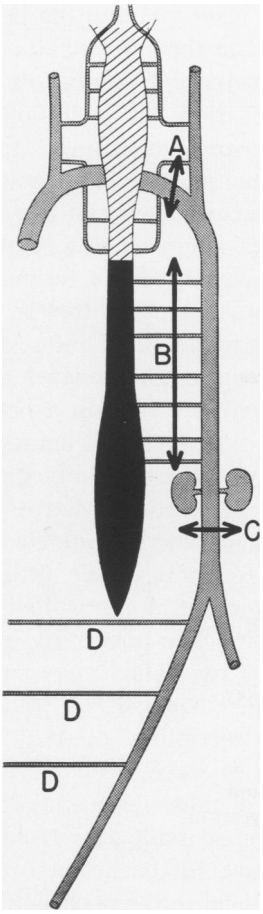


FIG. 1. Illustrating potential neurologic damage. *A*, Suprarenal occlusion of the aorta—interferes with the circulation of both the spinal cord and peripheral nerves. *B*, Occlusion of the intercostal arteries interferes with only the blood supply of the spinal cord. *C*, Infrarenal occlusion of the aorta interferes only with the circulation to the peripheral nerves through arteries *D*.

in sensibility to ischemia, while a peripheral lesion can become apparent only when the collateral circulation in the spinal cord prevents a central lesion. Therefore, occlusion of the aortic circulation as a result of operation may result in neurologic complications caused by spinal ischemia or in those caused by ischemia of peripheral nerves.

CLINICAL PICTURE OF SPINAL ISCHEMIC NECROSIS

Surgical occlusion as the cause of ischemic lesions of the spinal cord is a new entity in human pathology and little is known

TABLE 1. *Clinical Picture in the Complicated Cases*

Case	Postoperative Neurologic Disturbances	Evolution of Neurologic Disturbances
Case of Lam (Table 4)	Marked weakness in lower extremities; slight flexion of knees and dorsiflexion of left foot; hypoalgesic to L_1; urinary retention	After 3 weeks prompt partial recovery; spontaneous urination; after 12 weeks able to walk
Case of Cooley (Table 4)	General weakness of all muscles of lower extremities; diminished sensation in feet; no loss of function of sphincters of anus and bladder	After 2 months, ambulatory with cane support; after 11 months, unassisted walking; minimal residual weakness in legs
Case of Burgess (Table 2)	Almost complete flaccid paralysis of both lower extremities	After 3 weeks strength and active control of legs slowly returned; after 2 years had not regained use of all muscles of lower extremities; able to walk without assistance, with alteration of gait; marked atrophy of calves; legs tire readily
Case of Bahnson (Table 3)	Paraplegia	
Case of Bahnson (Table 4)	Weakness of lower extremities	
Case of DeBakey (Table 4)	Moderate manifestations of spinal cord ischemic damage; paresthesias of lower extremity	
Case of Crafoord Patient $8\frac{3}{4}$ years old (Table 2)	Flaccid paralysis and hypoesthesia both legs; vesicorectal incontinence	First day, improved sensibility and slow improvement of vesicorectal incontinence; 10th day, some motility of toes of right foot; died 14th day; autopsy showed necrosis of lumbosacral spinal cord
Case of Crafoord Patient 7 years old (Table 2)	Weakness and spasticity of both legs, right greater than left; pain in lower extremities	After 4 years diminishing of weakness; after 6 years atrophy of right calf muscles and bilateral anterior tibial region; spasticity of feet; absent plantar reflexes
Case of Crafoord Patient 34 years old (Table 2)	Urinary retention	After 1 year leg muscle rigidity and sudden attacks of complete loss of power; 5 years after operation; atrophy of adductors and dorsal muscles of thigh; cramps of calf muscles, absent plantar reflexes bilaterally
Case of Crafoord Patient 20 years old (Table 2)	Complete flaccid paralysis including hip muscles, bilaterally; severe pain in left leg	After 1 month, slight flexion of left hip and both knees; pain in thighs and groin; hypesthesia and hypalgesia to umbilicus; in 10 months, considerable loss of function of lower legs, ugly waddling walk, marked hypesthesia and hypalgesia of lower legs and feet; weakness of patellar reflexes, absent plantar and Achilles reflexes
Case of Eiseman (Table 4)	Severe motor deficit in both lower extremities with maximal damage below L_3	Permanent

about the various aspects of the clinical picture of postoperative ischemic necrosis. Pertinent data on these aspects as they were reported in the literature are given in the Tables. Neurologic data on these cases are limited because of the difficulties of a systematic neurologic examination after operation (Table 1).

The neurologic symptomatology is the manifestation of destruction of the lower motor neurons and the sensory structures in the lower spinal cord; therefore, as suggested by van Harreveld,[113] it is unlikely that a stage of spinal shock is present in these cases. The recognition of the symptoms as soon as the patient recovers from anesthesia is characteristic. Disturbances of motor function in the lower extremities are constantly present, and vary from absent or reduced to exaggerated. They may be localized to the sacral segments or they may reach as high as the eighth thoracic segment, and the upper level may be different on each side. Sensory disturbances are not constant and may vary from absent or reduced sensations to hypersensibility. Dissociation of various types of sensory function is often present, touch and deep sensibility being more frequently preserved. The upper level corresponds to the level of the motor disturbances. Reflexes are most often reduced or absent and depend on the magnitude and extent of the motor insult. Vesicorectal disturbances almost always are present. The motor and sensory symptoms may vary from very temporary to permanent, but recovery is most likely to occur in the sensory function and often does so in the first postoperative days. The different functions frequently recover in an irregular way and one side may show better recovery than the other. It seems that complete recovery is possible only during the immediate postoperative period and that otherwise variable sequelae will remain. In the apparent recovery in the motor system an important role is played by auxiliary muscle groups. The most serious permanent clinical picture is that of paraplegia.

NEUROLOGIC COMPLICATIONS CAUSED BY ISCHEMIA OF PERIPHERAL NERVES

Little attention has been paid to the possibility of neurologic complications after aortic surgery that are not caused by spinal ischemia, but result from ischemic damage to the peripheral nervous system of the lower extremities. These are important to consider because in this way a neurologic symptomatology may be caused which is similar to that caused by spinal ischemia. It results from prolonged interference with the circulation to the lower extremities which occurs during occlusion of the abdominal aorta. Prolonged occlusion of the abdominal aorta may be necessary during abdominal aortic surgery as a result of extension of disease beyond this field. It also may be caused by saddle embolism which is a potential hazard in any aortic procedure, and the more so in operation for aneurysm which, even without surgery, is one of the etiologic factors of saddle embolism. Prolonged occlusion of the abdominal aorta may also result from dissecting aneurysm which has been reported as resulting from aortic surgery (Clagett[41]).

The main diagnostic difficulty arises from the fact that the vascular symptoms of abdominal occlusion may be only temporary and not result in gangrene of the extremities, but enough damage can be caused to the nervous system to produce paraplegia with no apparent damage to other tissues because of the timely return of circulation. This return may occur either during operation with re-establishment of normal circulation or, in the case of saddle embolism, by establishment of sufficient collateral circulation. This is substantiated by a review of the literature from which it appears that many cases of untreated saddle embolism as well as of ligature of the aorta did not result in loss of tissue. Many of these patients were left with neurologic sequelae of

varying degree to the extent of long-lasting paraplegia.

A similarity of the neurologic picture caused by ischemia of peripheral nerves, and that caused by spinal ischemia is substantiated by the neurologic symptomatology of saddle embolism reported in the literature before the operative era. In more recent cases neurologic symptoms often do not have time to develop, whereas the vascular symptomatology is the predominant immediate problem requiring treatment.

Motor, sensory and reflex symptoms may resemble in their different variations, in their extent and in their evolution those resulting from spinal ischemic necrosis. In fact, a neurologic picture may be that of paraplegia and loss of sensation which extends as high as the umbilicus. Vesicorectal disturbances which have been a subject of much controversy in discussing the neurologic picture of ischemia of peripheral nerves are reported only in older descriptions and their occurrence after saddle embolism as reported in the more recent literature is not fully appreciated. In going over that older literature, vesicorectal disturbances as symptoms of abdominal aortic occlusion were found in 24 instances, each occurring in cases with high level of motor and sensory disturbances. Urinary or rectal incontinence, or both, were found in 21 cases and urinary retention in three. In no instance of a nonoperated embolism was their absence mentioned.

Clinical differentiation between a central or peripheral origin of neurologic complications associated with aortic surgery, may be aided by an aortogram. When this shows the presence of an embolus, operation may prevent lasting nerve damage.

ANALYSIS OF COMPLICATIONS OF AORTIC OPERATIONS

An attempt was made to review the incidence of lesions due to spinal ischemia associated with aortic surgery from cases in the literature and also from personal cases.

Cases of suprarenal aortic surgery were divided into three categories according to the manner of interference with the vascular supply of the spinal cord. This may occur (1) by occluding the aorta alone, (2) by occluding the intercostal arteries alone, as in aortic resection with shunt, and (3) by occluding both in cases of resection without a shunt. Fifty-one cases from the literature of occlusion of the suprarenal aorta alone, with details of occlusion as well as data on the presence or absence of neurologic complications, are listed in Table 2. Of these, six were found to have been accompanied by spinal lesions. Illustration of similar cases is given in the following two case reports:

Case 1. This patient gave a 6 month history of lower abdominal and back pain. Complete studies had been carried out elsewhere and were repeated at the Lahey Clinic. Exploratory laparotomy was performed on June 9. Chronic cholecystitis and cholelithiasis were found and cholecystectomy and appendectomy were performed. A mediastinal mass was demonstrated on a chest roentgenogram. Bronchoscopy, angiocardiography and myelography were carried out. Aneurysmorrhaphy was performed on July 3. The day after operation he was paraplegic and an epidural block was performed. On July 13 an aortogram showed obstruction of the aorta at the third lumbar segment. This patient has permanent paraplegia to the fourth lumbar segment on the left and the first lumbar segment on the right.

Case 2.* A 25-year-old woman had ligation of a patent ductus arteriosus in 1950. In November 1952 she was readmitted to the hospital with vague complaints in the upper thorax and symptoms of compression of the left recurrent nerve. On November 7, during reintervention a large aneurysmal mass was found between the left pulmonary artery, the upper part of the descending aorta and the aortic arch. This mass had originated at the site of the former operation on a fistulous tract between the aorta and the left pulmonary artery. It was necessary to liberate the aortic arch and the first portion of the descending aorta, and the three pairs of upper intercostal arteries had to be divided. Crafoord clamps were placed on the aorta proximal and distal to the aortic end of the fistulous tract and distal to the left subclavian artery. The

* Case 2 is reported with the permission of Dr. R. Sweet.[109]

TABLE 2. *Occlusion of Aorta Alone*

Author	Operation, year	No. of Cases	Age, yrs.	Operative Indications	Aortic Occlusion: Duration	Aortic Occlusion: Level	Complications	Comment
33 Czerny, 1879	1870	1		Hemorrhage of aorta, right kidney torn off aorta	Permanent	Between 2 renal arteries	Paralysis and anesthesia, lower extremities +	Patient died 10 hours after operation; gradual onset of paraplegia 2 hours after operation (probably caused by peripheral nervous ischemia)
117 Villar, 1892	1892	1	46	Aneurysm of aortic arch	Temporary	Distal to aortic arch	No neurologic complications −	
50 Guinard, 1909	1909	1		Aneurysm, upper thoracic aorta	Permanent	At D_9	No neurologic complications −	Died 2nd postoperative day of anuria
75 Kümmel, 1914	1914	1	45	Aneurysm low thoracic aorta	12 min.	10 cm. above diaphragm		Died during operation
87 Reid, 1926	1921	1	36	Abdominal aneurysm	Permanent	Above celiac axis	No neurologic complications −	Died 1 month after operation
7 Andrus, 1931	1931	1	57	Syphilitic aneurysm, upper abdominal aorta	Permanent	Ligation and division of middle descending thoracic aorta	No neurologic complications −	Died 1½ hours after operation
88 Reid, 1931	1931	1	35	Syphilitic aneurysm, upper abdominal aorta	Temporary (time to fill aneurysm with ball plug)	Middle descending thoracic aorta	No neurologic complications −	Died 10 hours after operation
30 Crafoord, 1952	1945	1	7	Patent ductus arteriosus	18 min. 50 sec.	Below left subclavian artery	Permanent spastic paraplegia +	Alive 6 years after operation
30 Crafoord, 1952	1945	1	34	Patent ductus arteriosus	24 min.	Below left subclavian artery	Permanent spastic paraplegia +	Alive 5 years after operation
26 Conklin, 1950	1946	1	9	Hemorrhage of aorta, patent ductus arteriosus; tear posterior aorta	22 min.; interval 25 min. then 13 min.	Below left subclavian artery	No neurologic complications	
30 Crafoord, 1952	1946	1	8¾	Patent ductus arteriosus	49 min.	Below left subclavian artery	Flaccid paraplegia +	Died 14th day after operation
4 Adams	1947	1	4	Patent ductus arteriosus; aortic hemorrhage	15 min.	Below left subclavian artery	No neurologic complications −	
109 Sweet	1950	1	20	Aneurysm on recurrent patent ductus arteriosus	38 min.	Below left subclavian artery	Flaccid paraplegia +	Plus ligation of upper 3 and 4 pairs of intercostal arteries
64 Holman, 1953	1950	1	11	Patent ductus arteriosus; aortic hemorrhage	Temporary (time to repair tear)	Below left subclavian artery	No neurologic complications −	
95 Sandblom and Ekström, 1951	1950	1	8	Wide patent ductus arteriosus	Temporary	Distal left subclavian artery		Died during operation
28 Cooley and DeBakey, 1952	1951	1	41	Syphilitic aneurysm of descending aorta; lateral excision	22 min.	Above D_9	No neurologic complications −	During lateral excisional therapy

TABLE 2. *Continued*

Author	Operation, year	No. of Cases	Age, yrs.	Operative Indications	Aortic Occlusion: Duration	Aortic Occlusion: Level	Complications	Comment
30 Crafoord, 1952	1951	1	20	Patent ductus arteriosus	27 min.	Below left subclavian artery	Flaccid paraplegia +	Alive 10 months after operation
30 Crafoord, 1952	1951	2	Not stated	Patent ductus arteriosus	10–15 min.	Distal left subclavian artery	No neurologic complications	
		5	Not stated	Patent ductus arteriosus	15–19 min.	Distal left subclavian artery	No neurologic complications	
		10	Not stated	Patent ductus arteriosus	20–24 min.	Distal left subclavian artery	No neurologic complications	
		1	Not stated	Patent ductus arteriosus	25–29 min.	Distal left subclavian artery	No neurologic complications	
		3	Not stated	Patent ductus arteriosus		Distal left subclavian artery	No neurologic complications	
9 Bahnson, 1953	1952	1	45	Syphilitic aneurysm of upper abdominal aorta	37 min.	Between diaphragm and celiac axis	No neurologic complications	Lateral excision therapy
9 Bahnson, 1953	1952	1	41	Syphilitic aneurysm of upper thoracic aorta	20 min.	Distal to left subclavian artery, and 10 cm. below left subclavian artery	No neurologic complications	Lateral excision therapy
19 Burch *et al.*, 1953	1952	1	67	Liver metastasis	10 min.	Just above diaphragm	No neurologic complications	Partial resection of liver
9 Bahnson, 1953	1952	1	49	Traumatic aneurysm of upper thoracic aorta	7 min.	Proximal to left carotid	No neurologic complications	
19 Burch *et al.*, 1953	1952	1	70	Liver metastasis	10 min.	Proximal to celiac axis	No neurologic complications	Partial resection of liver
20 Burgess, 1955	1953	1	32	Patent ductus arteriosus	More than 60 min.	Distal to left subclavian artery	Flaccid paraplegia +	Tear of posterior aortic wall; operative cardiac arrest
67 Javid *et al.*, 1955	1954	1	61	Ruptured aneurysm of abdominal aorta	Temporary	Above diaphragm	Not mentioned	Died 2 hours after operation
67 Javid *et al.*, 1955	1954	1	64	Ruptured aneurysm of abdominal aorta	50 min.	Above diaphragm	Postoperative movement of legs normal	Died 4 hours after operation
65 Hughes, 1954	1954	1	Not stated	Traumatic hemorrhaging abdominal aorta	15 min. 10 min.	At diaphragm	?	Died during operation, intra-aortic balloon catheter tamponade
36 DeBakey *et al.*, 1955	1955	1	72	Dissecting aneurysm of thoracic and abdominal aorta	Temporary	Upper thoracic aorta	No neurologic complications	Obliteration of false aortic passage
36 DeBakey *et al.*, 1955	1955	1	43	Dissecting aneurysm of thoracic and abdominal aorta	18 min.	Low thoracic aorta	No neurologic complications	Obliteration of false aortic passage
38 Dripps, 1955	1955	1	Not stated		More than 2 hours	At diaphragm	No neurologic complications	Under hypothermia
69 Julian *et al.*, 1955	1955	1	23	Patent ductus arteriosus	38 min.	Below subclavian artery	No neurologic complications	Accidental tear of aorta

TABLE 3. *Occlusion of Intercostal Arteries Alone*

Authors and Reference	Operation, year	Age, years	Operative Indication	Extent of Occlusion of Intercostal Arteries		Result and Comment
97 Schafer and Hardin, 1952	1951	60	Syphilitic aneurysm of ascending, arch and descending aorta	Upper half of thoracic intercostal arteries	Shunt aorta	Died 1 hour after operation
105 Stranahan *et al.*, 1955	1953	53	Syphilitic aneurysm of arch and descending thoracic aorta	All thoracic intercostal arteries	Shunt aorta	No neurologic complication
68 Johnson *et al.*, 1955	1953	24	Syphilitic aneurysm of upper thoracic aorta	Upper half of thoracic intercostal arteries	Shunt aorta	No neurologic complication
105 Stranahan *et al.*, 1955	1953	25	Traumatic aortic aneurysm	Upper half of thoracic intercostal arteries	Shunt aorta	No neurologic complication
10 Bahnson, 1953	1953		Aneurysm of thoracic and suprarenal abdominal aorta	All thoracic intercostal arteries	Shunt aorta	Paraplegia, lower extremities
3 Adams, 1955	1954	58	Syphilitic aneurysm of upper thoracic aorta	Upper half of thoracic intercostal arteries	Shunt aorta	No neurologic complication
68 Johnson *et al.*, 1955	1954	24	Congenital aneurysm of upper thoracic aorta	Upper half of thoracic intercostal arteries	Shunt aorta	No neurologic complication
3 Adams, 1955	1955	30	Traumatic aneurysm of upper thoracic aorta	Upper half of thoracic intercostal arteries	Shunt aorta	No neurologic complication
22 Chamberlain, 1955	1955	64	Multiple aneurysms of thoracic aorta	All thoracic intercostal arteries	Shunt aorta	No neurologic complication

aneurysm was then excised at its aortic end, the fistulous opening in the aorta closed and the aortic clamps removed. The aneurysm was freed from the left pulmonary artery and resection of the aneurysm completed. The aorta had been occluded for 38 minutes. The blood pressure level remained stable during the procedure. As soon as the patient awakened after operation it was noted that she had marked pain in both legs and it was impossible for her to move her legs. Sensation was absent in the lower part of the body up to a level between pubis and umbilicus. There was urinary and rectal incontinence. During the first postoperative week, however, there was gradual, slow and partial return of motor power in all muscle groups of the lower extremities. Deep sensibility returned completely, pain sensibility returnd to the level of the groin and touch sensibility to the midcalf. One and a half months after operation the patient was able to control urinary and bowel function but no further return of motor and sensory function occurred.

Data on interference with the intercostal circulation alone were found in nine cases, with one spinal complication, and are listed in Table 3. Data on occlusion of the aortic circulation and of the intercostal arteries were found in 18 cases and are listed in Table 4. Of these, five had resultant spinal lesions. Recently DeBakey [35] reported two cases of manifestation of ischemia of the spinal cord not mentioned in Table 1; they were described as mild and transient and occurred after resection which included the proximal half of the thoracic aorta.

Ten cases of repair of isthmic coarctation followed by spinal neurologic deficit were gathered from the already extensive series of resections reported in the literature. Pertinent operative and neurologic data on these cases are listed in Table 5. To these we add the following case report.

Case 3. During routine physical examination a 20-year-old healthy man was found to have hypertension (220/110). A left transthoracic sympathectomy from the fourth thoracic to the first lumbar segment was performed on October 7, 1948, and a right transthoracic sympathectomy from the fourth thoracic to the first lumbar segment on

TABLE 4. *Occlusion of Both Aorta and Intercostal Arteries*

Authors and Reference	Operation, year	Age, years	Operative Indication	Extent of Occlusion of Intercostal Arteries	Aortic Occlusion		Result and Comment
					Duration, minutes	Level	
108 Swan *et al.*, 1950	1949	16	Postcoarctation aneurysm	Upper half of thoracic intercostal arteries			No neurologic complication; graft 8 cm.
76 Lam and Aram, 1951	1950	56	Syphilitic aneurysm from left subclavian artery to diaphragm	All thoracic intercostal arteries	24	Below left subclavian	Flaccid paraplegia of lower extremities; insertion of graft, aneurysm left in place
34 DeBakey and Cooley, 1953	1953	46	Syphilitic aneurysm from low thoracic aorta to upper abdominal aorta	Intercostal and lumbar arteries from D_9 to superior mesenteric artery	45	Midthoracic aorta	No neurologic complication; graft 8 cm.
29 Cooley and DeBakey, 1955	1953	50	Syphilitic aneurysm from left subclavian to 5 cm. above hiatus of diaphragm	All thoracic intercostal arteries	45	Below left subclavian	Paralysis of lower extremities; graft
Idem	1953	58	Aneurysm from left subclavian artery to diaphragm	All thoracic intercostal arteries	77	Below left subclavian	Died 12 hours after operation; graft 30 cm.
Idem	1953	50	Syphilitic aneurysm of lower half of thoracic aorta	Lower half of thoracic intercostal arteries	38	Midthoracic aorta	Graft 12 cm.
Idem	1953	17	Congenital aneurysm of aortic arch and upper thoracic aorta	Upper half of thoracic intercostal arteries (plus occlusion of left subclavian artery)	53	Below left carotid	No neurologic complication; under hypothermia
Idem	1954		Traumatic aneurysm of upper thoracic aorta	Upper thoracic intercostal arteries (plus occlusion of left subclavian artery)	58	Proximal to left subclavian	No neurologic complication; under hypothermia
Idem	1954			Upper thoracic intercostal arteries (plus occlusion of left subclavian artery)	50	Proximal to left subclavian	No neurologic complication; under hypothermia
Idem	1954	31	Traumatic aneurysm of aortic arch and upper thoracic aorta	Upper half of thoracic intercostal arteries (occlusion of left subclavian artery)	62	Below left carotid	No neurologic complication; under hypothermia
Idem	1954	66	Syphilitic aneurysm of upper two-thirds of thoracic aorta	Upper two-thirds of thoracic intercostal arteries	53	Proximal to left subclavian	
51 Gwathmey and Thompson, 1955	1954	60	Aneurysm of thoracic aorta	All thoracic intercostal arteries	40 plus 20		No neurologic complication
69 Julian *et al.*, 1955	1954	22	Traumatic aneurysm of upper thoracic aorta	Upper half of thoracic intercostal arteries	30	Below left subclavian	No neurologic complication; under hypothermia
42 Eiseman and Summers, 1955	1954	32	Traumatic aneurysm of lower thoracic aorta, D_{10} to D_{11}	D_9 to L_1	63	At D_8	Paraplegia of lower extremities; under hypothermia (28° to 30° C.)
36 DeBakey *et al.*, 1955	1954	58	Dissecting aneurysm of lower two-thirds of thoracic aorta	3 midthoracic intercostal arteries	44	Midthoracic aorta	No neurologic complication; under hypothermia
Idem	1955	40	Thoracic and abdominal dissecting aneurysm	Upper two-thirds of thoracic intercostal arteries	34		Manifestations of spinal cord ischemia in lower extremities
11 Bahnson, 1955	1955		Aneurysm of thoracic aorta	? thoracic intercostal arteries	45		Weakness of lower extremities
69 Julian *et al.*, 1955	1955	51	Aneurysm of upper thoracic aorta	Upper half of thoracic intercostal arteries	68	Below subclavian	No neurologic complication; under hypothermia

TABLE 5. *Complicated Cases after Operation for Coarctation of Aorta*

Authors and Reference	Operation, year	Age, years	Technical Details	Neurologic Findings	Evaluation of Neurologic Findings	Comment
16 Blalock (Bing *et al.*), 1948	1948		Proximal segment of aorta too short, left subclavian used as bypass; operative shock	Partial paralysis of legs; sensory probably normal		Diagnosed as occlusion of anterior spinal artery
13 Beattie *et al.*, 1953	1951	29	No distinct rib notching; 2 highest intercostals below coarctation not dilated; occlusion of subclavian artery; normal circulation restored after 38 min.	On 1st postoperative day paralysis areflexia; intact tactile and position sense; catheter necessary	Died 5th postoperative day; statu quo	Autopsy: Pathologic changes L_2 to S_2 anterior gray matter; posterior horns and white matter free
48 Gross, 1953	1952	31	Ligation of 4 sets of intercostals which were not large and graft 4 to 5 cm.; nothing abnormal	During first 24 hours unable to move lower extremities; loss of sensation	After 4 to 5 days sensory function returned and able to move big toes of each foot in 1 month; ± return in thighs; pain in lower extremities 3 months; in 1 year good return of thigh muscle function; uses braces	
48 Gross, 1953	1952	50	Not large collaterals; nothing abnormal; no shock; no division of collaterals	Complete flaccid paraplegia; rectovesical incontinence	In 4 months patchy return of motor function, left better than right; return of sensation to knees; in 2 years pain in legs; no spontaneous defecation, automatic bladder; little movement in lower extremities	Diagnosed as caused by presence of small anterior spinal artery and arteriosclerotic changes
44 Finnerty, 1955	1952		Occlusion of right subclavian artery originating distal to coarctation	Paraplegia	Persistent	
96 Santy, 1953	1952	36	Huge collateral intercostal torn off aorta below stenosis; clamping of aorta proximal to stenosis and proximal and distal to torn intercostal; clamping of left subclavian artery; severe blood loss	Paraplegia with loss of sensation up to sternum	Died 3rd postoperative day; clinical picture unchanged	
15 Beck, 1953	1953	19	Occlusion of left subclavian artery originating distal to coarctation; normal circulation in 45 min.; intercostals less hypertrophied than expected	Mild lower leg weakness requiring use of braces	Permanent	
80 Mustard *et al.*, 1955	1953	13	1949 ligation of patent ductus arteriosus; ligation of 2 pairs of intercostal arteries 3 mm. in diameter; normal circulation restored in 60 min.	Bilateral lower limb flaccid paralysis and areflexia; sensory normal except hypoalgesia D_8; urinary retention	1 month postoperative motor statu quo; hypothermoalgesic to L_2 to L_5	Diagnosed as injury or thrombosis of anterior spinal artery
79 Mustard	1954	8	Plus ligation of very large ductus; moderate coarctation; normal circulation restored after 60 min.; exploratory thoracotomy during previous year	Some movement possible in both legs	In 14 days motor function normal to thigh; sensory normal except hypoalgesia T_{11} to T_{12}; absent reflexes	Diagnosed as thrombosis of anterior spinal artery
83 Swan (Owens *et al.*), 1955	1955	18	Plus ligation of patent ductus arteriosus in preductal type; poor collaterals; normal circulation restored in 62 min.; no shock	Bilateral symmetrical paraplegia and vesicorectal disturbances	Return of sphincter function; partial return of leg function; uses crutches	

December 4, 1948. At the time of the left thoracotomy an isthmic coarctation was found to be present. Resection of the coarctation was advised and on May 20, 1949, the patient again underwent operation.

A high degree coarctation was found to be present at the usual site. The left subclavian artery was huge, approximating the size of the aorta. The collateral circulation was developed to a high degree and the fourth and fifth intercostal arteries were exceedingly large and tortuous, and disappeared almost together. A very small prestenotic ductus arteriosus which appeared and felt patent was ligated and divided. One pair of intercostal arteries was ligated and divided above the coarctation and two pairs of intercostal arteries were similarly treated below the coarctation. Modified Blalock clamps were placed on the left subclavian artery, on the aortic arch distal to the left carotid artery and on the aorta distal to the coarctation. The coarctation was then resected and repair was completed. The blood pressure level throughout the procedure remained stable. The morning following operation flaccid paralysis and areflexia in the lower trunk and lower extremities was noted, with loss of all modalities of sensation up to the level of the eighth thoracic segment. There was urinary and rectal incontinence. Arterial pulsations were normal in the lower extremities. The neurologic picture has remained unchanged since then until March 1955 when he was last seen.

In infrarenal aortic surgery both by ligature and resection no instance of a spinal ischemic lesion has been found although the cases published and our own series are now extensive.

From this review it appears that spinal complications are reported only after suprarenal aortic surgery and even then occur very irregularly. This has happened despite the same type of procedure and similar operative circumstances and in patients of all ages. The patients were above the minimum age of seven years in the instance of occlusion of the aorta and eight years in coarctation. Occlusion for less than 18 minutes 40 seconds produced no complications. In the complicated cases occlusion was of variable duration above this time. From clinical observations and from pathologic data it would appear that the lumbosacral localization of lesions of the spinal cord was a constant feature.

An attempt will be made to analyze the local and general factors which play a role in this erratic occurrence of complications after suprarenal aortic surgery and also in the absence of lesions of the spinal cord after infrarenal aortic surgery.

I. The Occurrence of Spinal Lesions after Suprarenal Aortic Surgery. From the anatomic distribution of the important aortic vessels to the spinal cord above the level of the eighth thoracic to the fourth lumbar it may be said that, except in rare cases, suprarenal aortic surgery is constantly accompanied by varying degrees of interference with the spinal blood supply. Yet neurologic lesions occurred in only a small percentage of cases, as may be seen from Tables 3 to 5. In this small percentage all were occluded for longer than 18 minutes 40 seconds. The absence of a lesion with occlusion below this length of time may be explained by the physiologic resistance of the structures of the spinal cord to anoxia.

The results of the experiments on resistance of nervous tissue to anoxia were reviewed by Heymans.[57] This resistance varies in most tissues and nervous tissue is most sensitive. Even here the sensitivity varies from one structure to another, with the highest sensitivity in the brain cortex decreasing toward the periphery and lowest in the distal end of the peripheral nerves. Thus, the minimal survival time when one part of the nervous tissue is ischemic seems to be determined by the resistance offered by the structure most sensitive to anoxia. This same difference is encountered in the spinal cord where the gray matter is most sensitive. It is probable that in the entire spinal cord the resistance of the various structures is comparable to that of similar structures at other levels.

Considerable difference of opinion exists as to the minimal survival time of the spinal cord experimentally because of the technical difficulty of obtaining the necessary absolute ischemia of the spinal cord. Dif-

ferent methods have been used, all aiming at the exclusion of the collateral circulation. In the rabbit most experimenters used modifications of Stenonis' experiment. The results of earlier experiments were revised by De Buck and De Moor.[37] In these experiments, times of vasular occlusion necessary to cause damage varied from 30 minutes to 60 minutes. More recently Tureen [112] obtained ischemic spinal lesions in cats after occlusion of the aorta for 15 minutes. The shortest time of aortic occlusion followed by spinal lesions in different kinds of animals was 10 minutes (Spronck,[102] Carrel,[21] Gross,[49] Rexed [90]). Krogh,[73] however, proved that in similar experiments the ischemia had been incomplete, so that the minimal survival time is likely to be even shorter than indicated by these experiments. In man the duration of resistance to anoxia of the most sensitive structure of the spinal cord is unknown. Cooley [27] stated that 20 to 30 minutes of occlusion of the aorta is tolerable in man. Crafoord [31] stated that 27 minutes and Dubost [39] 30 minutes of aortic occlusion was permissible. Eiseman [42] thought that 52 to 60 minutes of aortic occlusion was safe. The occurrence of ischemic lesions after 18 minutes 40 seconds would indicate that in man the survival time is closely related to that in animals. As in aortic occlusion there always exists a theoretical possibility of a complete ischemia in part of the spinal cord, it is quite possible that the safe time of occlusion is shorter than that generally accepted. The cytochemical mechanisms which influence the maximal duration of the anaerobic life of the nervous cell fall beyond the scope of this article and the reader is referred to the works of Himwich [58] and Gerard.[46] Also, for the histopathologic phenomena which characterize the reversibility and irreversibility of the ischemic cell, the reader is referred to the works of Tureen [111] and Gildea and Cobb.[47]

Having first considered this resistance to anoxia and the absence of lesions resulting from the physiologic resistance of the spinal structures most sensitive to anoxia, we will now consider the cases with occlusion of longer duration. Even in these cases there are relatively few complications and they occurred after variable times of occlusion.

In discussing these constants it may be said that the duration of vascular occlusion necessary to cause spinal damage depends on the degree of the resulting anoxia in the spinal cord. This time will be minimal when the degree of anoxia is maximal, but this probably never occurs, even experimentally. In fact, when the normal blood flow to a part of the spinal cord is interrupted, the collateral circulation becomes operative and prevents complete ischemia by causing a reduction in the degree of anoxia so that the structures concerned still receive some oxygen. Under these conditions the interval of time necessary for their destruction becomes longer. In fact, it seems likely that there is a certain level of anoxia at which cellular damage is impossible, where there is a relation between degree and duration of unsafe anoxia, in the sense that for a given level of anoxia there may be a requisite duration to cause damage. It should be noted, however, that very little is known about this relation and about the effects of stagnant anoxia on the nervous tissue in general. Thus, if damage results during suprarenal aortic surgery, this indicates that during the occlusion the collateral circulation did not develop fast enough in the deprived area. Therefore, damage should be considered as resulting from the interaction of (1) the interference with the normal blood supply and (2) the speed of establishment of the collateral circulation. It is the dependence on the interaction of such variables that has made the explanation for the occurrence of lesions of the spinal cord complex. But one of the reasons for this difficulty is also that little is known about how aortic surgery interferes with normal spinal circulation and also about the

establishment of collateral circulation. A discussion of the interference with normal circulation and of the establishment of collateral circulation will therefore follow.

A. Interference with Normal Spinal Circulation. Before discussing the interference, certain anatomic features on the circulation to the spinal cord will be considered solely from the viewpoint of aortic surgery.

Anatomic Considerations. The circulation of the spinal cord is not exactly defined, although there are many excellent descriptions of its vascular system. The modern principle of this circulation based on the plurality in origin of blood vessels each supplying different parts of the spinal cord dates from Adamkiewicz [2] in 1882. He refuted the older concept that the supply to the spinal cord was from the anterior and posterior branches of the vertebral arteries running on the respective surfaces of the cord and giving branches to the segmental nerve roots, and therefore called anterior and posterior spinal arteries. Instead, he described these arteries as anastomosing channels, each being formed from anastomosing branches of segmental arteries from the aorta. Kadyi [70] called them tractus arteriosus anterior and posterior. In spite of 70 years' knowledge of this principle, the older concept is still described in some anatomy books, while in the 1936 nomenclature [81] the older terms of arteriae spinales were maintained. Many studies in neurology and other fields have been based on the older anatomic concept. Present knowledge about the human spinal circulation is derived from studies of arterial and venous injection carried out by Adamkiewicz,[2] Kadyi,[70] Tanon,[110] Suh [106] and Bolton,[17] who altogether have examined 108 spinal cords. In their interpretation of the circulation from these studies, these authors depended on the direction taken by the injected product and by the appearance and dimensions of the injected vessels. No attempt has yet been made to study the regional blood supply *in vivo* by injection of radiopaque products. Most current data on the spinal vascular system have been reviewed by Tureen [111] in 1936. Essentially, most descriptions are concordant. In the extrinsic circulatory apparatus the only supplying branches which are constant are the most cranially situated two anterior and two posterior segmental branches from the vertebral arteries. These are the most cranial anterior and posterior radicular arteries of the spinal cord. A variable number of anterior radicular arteries (Adamkiewicz, three to ten; Suh, six to eight; Kadyi, five to ten) and of posterior radicular arteries (Kadyi, ten to 24) originate from the vertebral arteries, the upper two intercostals from the truncus costocervicalis, the aortic intercostals and the lumbar arteries. All these radicular arteries running on the ventral or dorsal, right or left nerve roots, join the midline. All adjacent cranial and caudal branches of the ventral radicular arteries join to form the continuous tractus arteriosus anterior, while dorsally, too, the tracti posteriores, similarly formed, are continuous only in the cervical and lumbosacral region.

Only minimal variations were found in the anterior and posterior intrinsic circulatory apparatus, and so it is possible to schematize the intrinsic blood supply in different individuals. The anterior tractus arteriosus gives off transverse branches which supply nearly the entire cross-sectional area of the spinal cord. Transverse branches from the tracti posteriores supply only the posterior horns of the gray matter and the dorsal white matter (Fig. 2).

In the extrinsic supply over the posterior spinal surface schematization to one type in the manner of distribution of the blood supply is again possible because of the great number of posterior radicular arteries which arborate into an extensive posterior network with better development of the tracti arteriosi only in the lumbosacral and cervical regions.

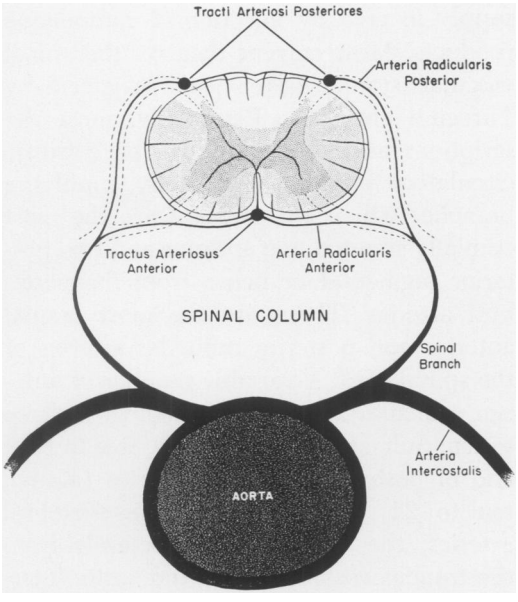


FIG. 2. Cross-sectional vascular supply to the spinal cord demonstrating the large anterior area (shaded) of the spinal cord receiving its blood supply via the arteria radicularis anterior and the tractus arteriosus anterior.

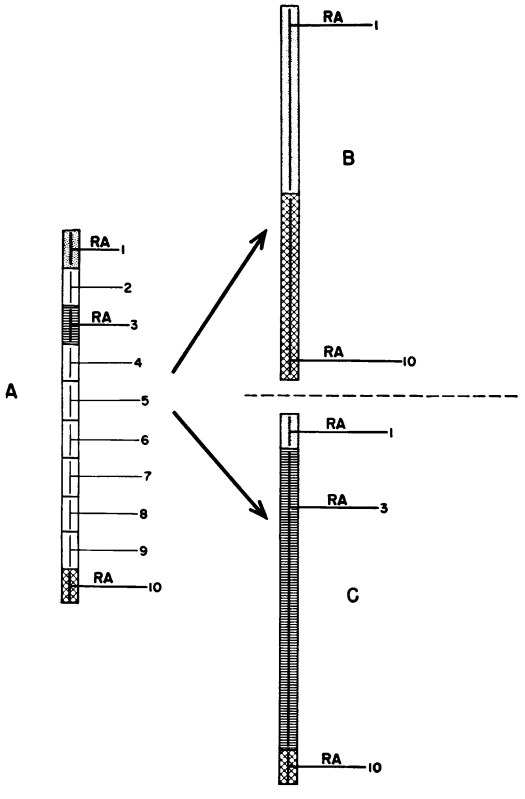


FIG. 3. Two different patterns of desegmentation are shown. *A*, Purely segmental vascular supply, radicular arteries 1, 3 and 10 each supplying one segment. *B*, Desegmentation pattern with radicular arteries 1 and 10, each supplying five segments via the tractus arteriosus anterior. *C*, Desegmentation pattern with radicular arteries 1 and 10 supplying only one segment, and radicular artery 3 supplying eight segments.

A similar schematization into one type in the extrinsic distribution of the blood from the anterior radicular arteries is possible only in lower Vertebrata [62, 104] and in the human fetus [59] (His). In man after fetal life, schematization is difficult because of great variations in the anterior segmental pattern and in the number of radicular arteries, both of which result from developmental evolutional desegmentation. In spite of these variations many authors were able to find in their interpretations of the regional spinal circulation some similarity among several spinal cords in the anterior blood supply. In such studies more attention is paid to factors which stress the individual similarities instead of differences and therefore are less helpful for this study.

Kadyi's investigations are especially interesting to this study because he examined the individual differences in the anterior circulatory pattern. After an exhaustive research, this author concluded that a schematization in the anterior circulation is impossible, that the blood supply is equal at any level of the spinal cord and that the apparent relative poverty of the thoracic regional supply recognized by other authors is caused merely by the greater length of the thoracic segments in which the central arteries are separated farther from each other than in the other parts of the spinal cord. In his interpretation of the spinal circulation he stated that in the process of evolution the tractus anterior is primarily formed by fusion of adjacent branches of the purely segmental primitive radicular arteries. By evolutional changes the area formerly supplied by regressed radicular arteries is taken over by the remaining

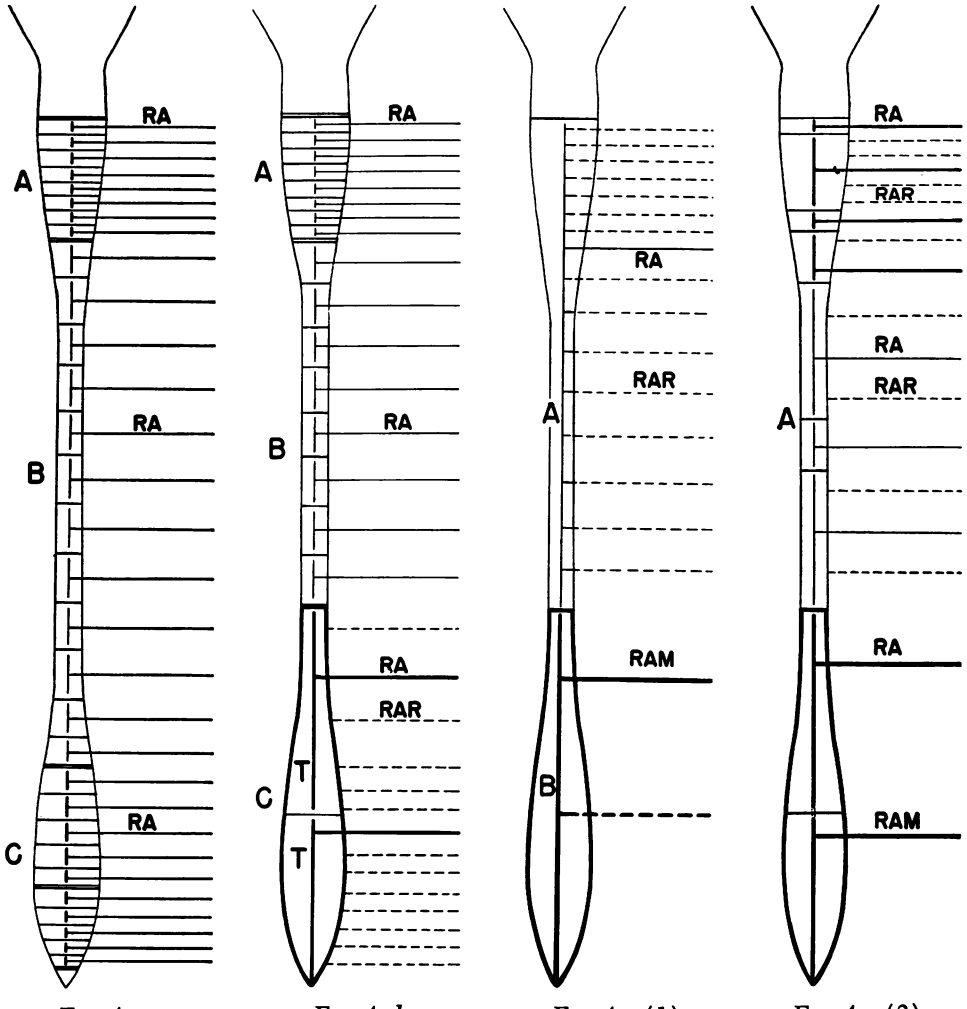


FIG. 4. *a* FIG. 4. *b* FIG. 4. *c*(1) FIG. 4. *c*(2)

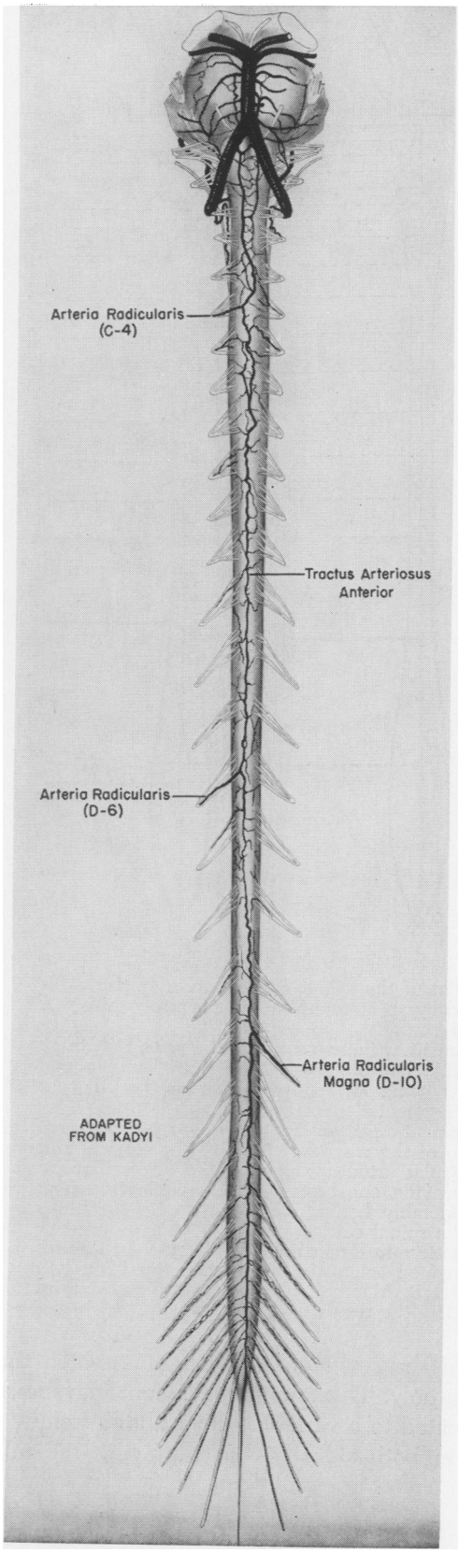
FIG. 4. *a*, Stages in the phylogenetic development of the spinal cord vascular system. This is the initial stage in which each segment is supplied by an individual radicular artery (rabbit). *A*, Cervical cord; *B*, thoracic cord; *C*, lumbosacral cord.

b, A succeeding stage of phylogenetic development with two radicular arteries remaining after regression of most of the segmental arteries (RAR) in the lower thoracic and lumbosacral spinal cord. The tractus arteriosus anterior (T) is being formed by fusion of the adjacent ascending and descending branches of the purely segmental primitive radicular arteries.

c(1), A further stage in phylogenetic development resulting in two important patterns: Type 1 (here illustrated), complete desegmentation of the vascular supply of the cervical and upper thoracic region. The lower of the two radicular arteries supplying the lower thoracic and lumbosacral spinal cord has also regressed, leaving one large arteria radicularis magna (RAM) in the lower thoracic region and a considerably better developed tractus arteriosus anterior (*B*) supplying the entire lower third of the spinal cord.

c(2), Type 2, illustrating a second important pattern, a larger number of radicular arteries (RA) persists at all levels with a less well developed tractus arteriosus anterior. The two important lower radicular arteries remain (RA and RAM), one to the lower thoracic level of the spinal cord and the other from the aorta below the renals.

radicular arteries. As a result the number and diameter of the radicular arteries do not depend on a greater or smaller need of a particular region, but rather on the length of the segment of the tractus which they supply. This supplied segment may be situated to a variable extent above, below, or on both sides of the supplying radicular



artery (Fig. 3). This "phenomenon of regression and progression" (Fig. 4*a*) was found most constant and evolved farthest in the anterior lower third of the thoracic and the entire lumbosacral spinal cord. At a certain point in development there were two radicular arteries, one in the lower thoracic and one in the lumbar region (Fig. 4 *b*). (An example of this happens to exist in the anterior venous pattern of the lower spinal cord.) If subsequently the superior artery becomes the more important one, the inferior artery regresses (Fig. 4 *c*-1). This process has occurred in approximately 50 per cent of Kadyi's cases. In contrast, if the inferior artery becomes more important, the superior one persists (Fig. 4 *c*-2). This occurs in the remaining approximately 50 per cent of his cases. As a result the lowest radicular artery was found to be always the best developed and has therefore been called the arteria radicularis magna; in it the blood flow is consistently in a craniocaudal direction (Kadyi found the latter artery from the ninth thoracic to the fourth lumbar; Suh found it as high as the eighth thoracic). The lower one fourth of the spinal cord is always supplied by this artery. The lumbosacral pattern was found to influence the cervical and upper thoracic pattern. With a high type of arteria radicularis magna the cervical and upper thoracic radicular arteries are usually better developed and in these cases the whole spinal cord is supplied by a smaller number of larger radicular arteries. It thus becomes possible to distinguish two extreme degrees of anatomical pattern with all variations between (1) the type with high arteria radicularis magna, important cervical and upper thoracic vessels and a small number of important radicular arteries, and (2) the type with low magna with numerous but small radicular arteries. Between these two all transitions are possible (Fig. 5). Indication of the variable region supplied by the arteria radicularis magna as suggested by

Fig. 5 .The human spinal cord demonstrating the extreme desegmentation of the vascular pattern.

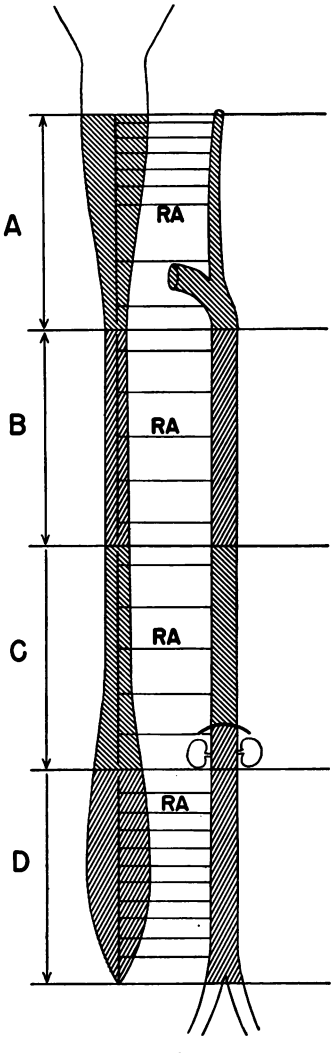


FIG. 6. *a*

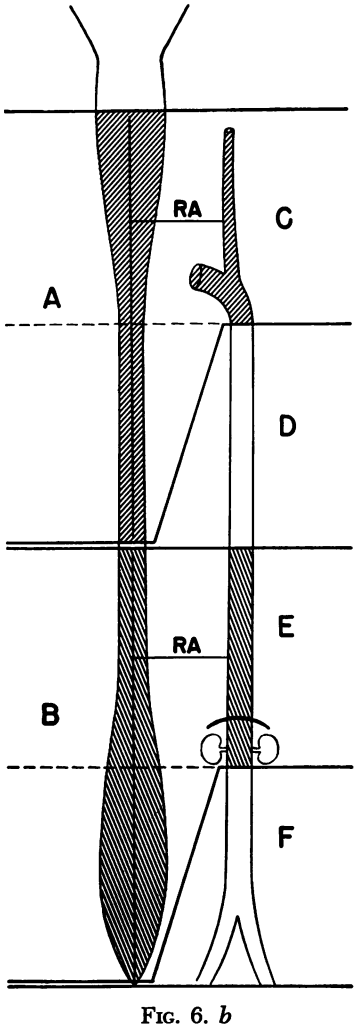


FIG. 6. *b*

FIG. 6. *a*, Purely segmental, primitive, vascular supply demonstrating the corresponding levels of origin of the segmental radicular arteries from the aorta (rabbit).
b, Desegmentation of the vascular supply of the spinal cord demonstrating the considerably higher aortal origin of the blood supply of the spinal cord for each level and all of the blood supply arising from the aorta above the renals.

Kadyi's findings can be shown from findings in cases of paraplegia after lumbar aortography as reported by Antoni,[8] Boyarsky [18] and Evans.[43] Antoni's case showed permanent flaccid paraplegia with anesthesia to the twelfth thoracic segment, and autopsy showed necrosis of the lumbosacral region. Boyarsky's patient developed permanent complete paraplegia with temporary anesthesia up to the eighth thoracic segment. Although Antoni explained that his case resulted from compression of the aorta for 30 minutes by the use of a pillow under the epigastrium, another explanation of this phenomenon suggested by Boyarsky and by Hol [63] was the toxicity of the contrast medium to the cord. The latter author proved this possibility in rabbits. The localization of the lesions in both human cases would indicate that the injection

occurred at the level of the arteria radicularis magna which in one case extended to the twelfth thoracic and in the other to the eighth thoracic segment.

The divisions of the spinal cord, each supplied by the subclavian arteries, by the proximal half of the thoracic aorta, by the distal half of the thoracic aorta and by the infrarenal aorta, would be equal to each other in a purely segmentally supplied spinal cord such as occurs in the rabbit (Fig. 6 *a*). In man, however, individual variations in the manner of desegmentation do not allow such divisions; individual differences occur in the extent and localization of the area of the spinal cord supplied by each considered aortic segment. This is caused, first, by the variation in the anterior pattern with differences in number and importance of radicular arteries and, second, by the erratic manner in which radicular arteries supply segments located above, below or on both sides. This difference is most obvious in the variations of the area supplied by the arteria radicularis magna. In approximately 50 per cent of the cases the lower thoracic aorta supplies one fourth of the spinal cord. In the other 50 per cent it supplies approximately one half of the spinal cord (Fig. 6 *b*). This difference also becomes apparent in the variation in the lower level of the area supplied by the subclavian arteries: the subclavian arteries may supply half of the spinal cord if the upper thoracic aorta does not give rise to a radicular artery.

These anatomic and circulatory findings applied to aortic operations suggest that great individual variations may occur in the extent of interference with the spinal blood supply. In the degree of interference with the normal blood supply the type of aortic procedure is important: (a) the extent of interference in cases of occlusion of the aorta alone will increase proportionately to a rise in the level of occlusion, (b) the degree of the interference will also increase when segmental branches of the aorta are occluded, proportionately to the number of occluded intercostal and lumbar vessels, (c) when both are occluded the degree again will increase proportionately to the level of occlusion as in (a). This difference in the degree of interference is caused by the difference in number of radicular arteries occluded from one type to another which is the result of the segmental pattern of spinal supply. As a result of the segmental character of the spinal blood supply it becomes apparent that interference with blood supply of the cervical and uppermost part of the thoracic spinal cord can be produced only by occlusion of the left subclavian artery. When this artery is occluded, only occlusion of the left truncus costocervicalis with terminal spinal branches is of concern, as the left vertebral artery has a good collateral circulation via the basilar artery.

Even in a single type of procedure the extent of the area to which there is interference of the blood supply may show individual differences. These differences will result from individual variations in the extent of the area supplied from one segment of the aorta, which is the result of the variation in the anterior spinal circulatory pattern and the variable manner in which blood is divided over the anterior tractus from the anterior radicular arteries. It should be noted that the type of aortic procedure is also important in the degree of interference with normal circulation: (a) The degree of interference is incomplete and temporary when the aorta alone is occluded because partial circulation exists in the aorta distal to the site of occlusion due to the collateral circulation around the occlusion. It has been shown by different authors that during experimental occlusion of the aorta, blood pressure in the aorta distal to the site of occlusion is maintained at one fourth to one third of its normal value.[6, 14, 54, 86, 116, 119] (b) The degree of in-

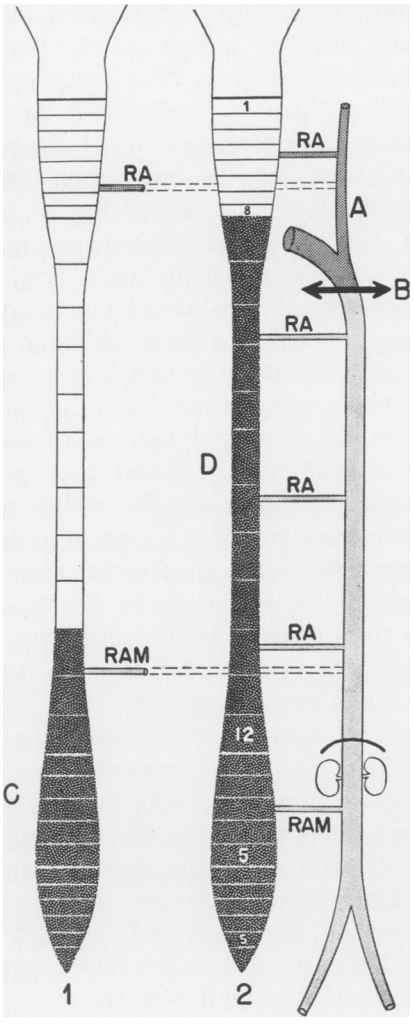


FIG. 7. *a*

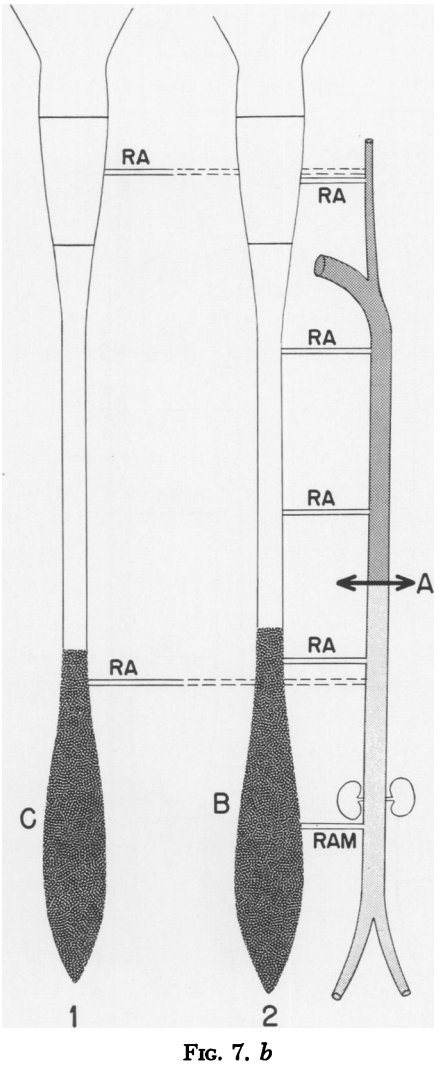


FIG. 7. *b*

FIG. 7. The effect of occlusion of the aorta alone. *a*, Effect of occlusion of the aorta distal to the left subclavian artery at *B*. Incomplete interference with the blood supply to the cord (stippled areas) *C* in Type 1 and *D* in Type 2 patterns of vascular supply. The incomplete degree of interference is due to the fact that the aorta distal to the site of occlusion still receives blood via collaterals.

b, Effect of occlusion of the aorta in the lower thoracic region. Incomplete degree of interference with the blood supply to the lumbosacral cord (stippled areas) *C* in Type 1 and *B* in Type 2 patterns of vascular supply.

terference is complete and permanent when the occlusion is only of intercostal and lumbar arteries (resection with shunt). (c) Both the above possibilities are combined in resectional therapy without shunt.

These differences in degree and extent of interference as they occur in different individuals and in different types of aortic procedures are illustrated by the theoretical manner in which the blood supply to the spinal cord would suffer interference in two types of circulatory pattern (Figs. 7, 8 and 9).

B. The Role of Establishment of the Spinal Collateral Circulation. No study dealing with the problem of the collateral

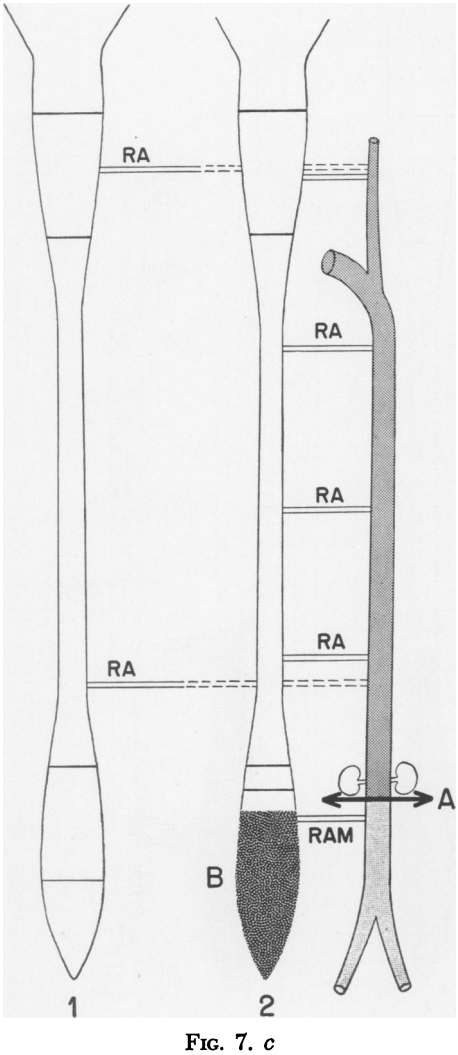


FIG. 7. *c*

FIG. 7. *c*, The effect of occlusion of the aorta below the renals at *A*. Incomplete interference with blood supply in Type 2 only (stippled area *B*).

circulation in the spinal cord is available. Most authors concluded from the extensive network in and around the spinal cord that the collateral circulation was potentially good. This should protect the cord against limited pathologic conditions in the blood vessels and against physiologic changes in the blood flow due to changes in pressure during movement. Kadyi compared the anastomotic network of the spinal cord with the circle of Willis. He stated that whereas in the brain there was one anastomotic network formed by four arteries, the spinal cord possessed a series of circular anastomoses formed from 20 or more arteries. In spite of this the spinal cord is the only organ in aortic surgery in which the collateral circulation may be insufficient. This paradox is apparently caused by the fact that in man the spinal anastomotic network is not always sufficiently well developed to allow the collateral circulation to come into play rapidly in a large ischemic area. A similar situation may occur in cases of dissecting aneurysm but data from such cases have not increased the understanding of how collateral circulation develops as information concerning the occlusion of the intercostal vessels is rarely reported. Observations from experimental aortic occlusion in animals cannot be directly applied to man because of the difference in the circulatory pattern.[61, 62, 104]

Conditions common to cases with and without spinal complications after aortic surgery, and some cases of dissecting aneurysm, provide an opportunity for studying some of the factors playing a role in the speed with which the spinal collateral circulation comes into use. In both complicated and uncomplicated cases after aortic occlusion just below the subclavian artery, the normal blood flow to the spinal cord is interfered with partially but to the same degree, and in both the same source of collateral blood supply from the cervical spinal cord is available. This fact and the constant lumbosacral localization of the spinal lesions suggest that a deciding factor in the causation of spinal damage may be the difference in the speed with which the collateral blood supply comes from the cervical to the lumbosacral spinal cord. This difference may be explained by a difference in capacity of the anterior spinal tractus. Actually it has been shown by injection studies of the intrinsic and extrinsic vascular system that the anterior tractus is the only continuous arterial channel from the cervical to the lumbosacral spinal cord.[17]

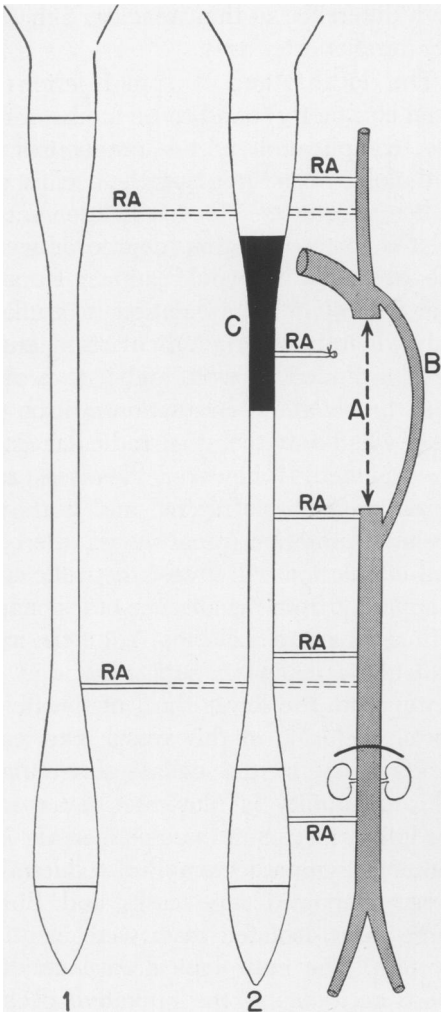


FIG. 8. *a*

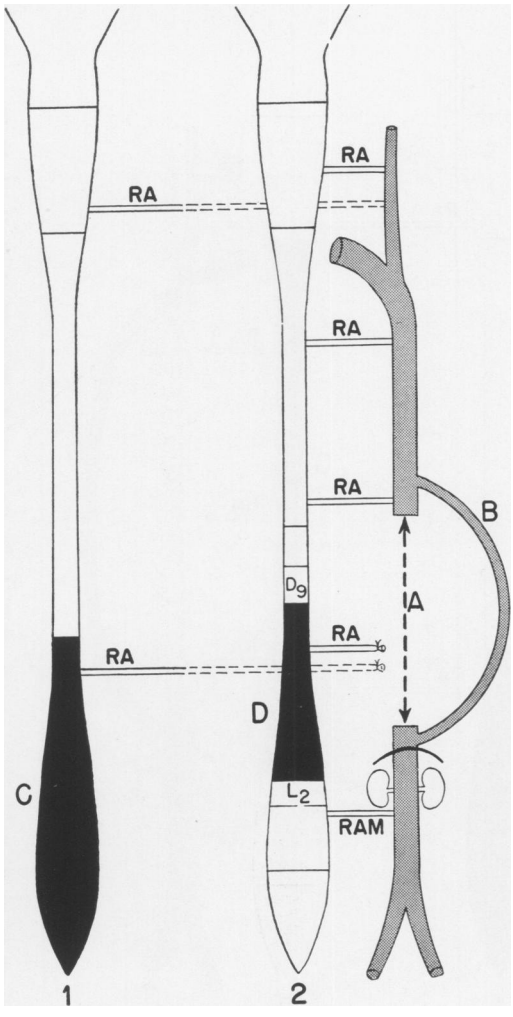


FIG. 8. *b*

FIG. 8. Interference with circulation of the spinal cord in suprarenal aortic resection utilizing a shunt. *a*, The effect of resection of the upper thoracic aorta (*A*) utilizing a shunt (*B*). Interference with the blood supply to the upper thoracic spinal cord in Type 2 only (*C*).

b, The effect of resection of the lower half of the thoracic aorta (*A*) utilizing a shunt (*B*). Interference with the blood supply of the lumbosacral spinal cord in Type 1 (*C*) and in the lumbar cord in Type 2 (*D*).

In animals it has been shown [61] that the presence of ischemic lesions in the rabbit and their absence in the dog following abdominal aortic occlusion are due to a better development of the dog's anterior tractus in the lower thoracic and lumbosacral regions (Fig. 10 *a* and *b*). This better development is caused by desegmentation in the lumbosacral vascular pattern. In man it is possible to find this difference in the extent of desegmentation resulting in a difference in development of the tractus and therefore it seems likely that these variations may become so important in some cases that, other factors being equal, great differences in the availability of the collateral blood supply occur (Fig. 11 *a* and *b*). When there are only two segmental arteries, one cervical and the arteria radicularis magna, the blood will be more read-

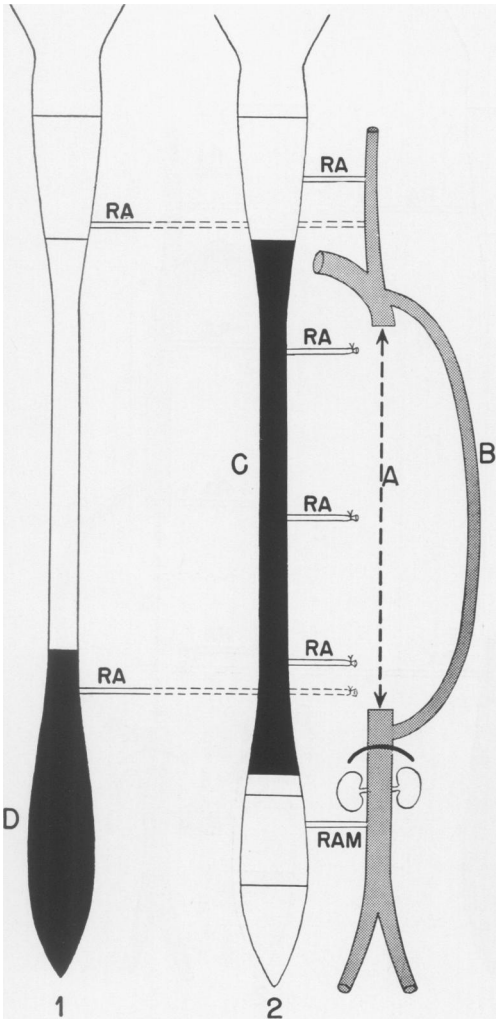


FIG. 8. *c*

FIG. 8. *c*, The effect of resection of the entire thoracic aorta distal to the left subclavian (*A*) utilizing a shunt (B). Interference with the blood supply to the spinal cord in Type 1 (*D*) and Type 2 (*C*).

ily available from the cervical sources within the lumbosacral region than when there is a small tractus with many segmental arteries. A similar comparison may be drawn between cases of dissecting aneurysm of Hamburger [53] and Moersch [77] in both of which complete occlusion of all aortic intercostal and lumbar arteries occurred. The complete necrosis up to the ninth thoracic in the first and the absence of lesions in the second may be explained by a difference in the capacity of the anterior tractus arteriosus.

The localization of spinal lesions has been constantly found to be lumbosacral in the complicated aortic procedures and pathologic conditions with occlusion of radicular arteries. This localization was unrelated to the duration, degree or level of the occlusion. It would appear from this that the collateral circulation is insufficient only when the arteria radicularis magna has been occluded as well, and it is probable that the collateral circulation will be sufficient when only the other radicular arteries are occluded. If, however, the arteria radicularis magna is infrarenal and is the only occluded anterior spinal vessel, the collateral circulation will always be sufficient, as is apparent from the absence of lesions after infrarenal aortic occlusion. Yet if the arteria radicularis magna is suprarenal and originates from the lower third of the thoracic aorta, occlusion of this vessel may lead to insufficiency in the collateral circulation. This possibility is illustrated by cases of medullary ischemic necrosis after lower thoracic sympathectomy, of which Mosberg [78] reported nine cases and Rubio [93] three cases. Isolated cases were mentioned by Suh. The most logical explanation for these accidents is the operative occlusion of a low thoracic arteria radicularis magna.

In contrast with occlusion of the aorta below the subclavian artery, the number of cases with occlusion of the midthoracic aorta alone is very small, but the absence of lesions indicates that sufficient collateral circulation is present in these cases.

In thoracic aortic resection, the spinal collateral circulation may become insufficient from one of the forms of occlusion mentioned above: (1) When lesions occur after resection of the proximal half of the thoracic aorta, insufficiency of the collateral circulation will have been caused by the temporary but too prolonged occlusion of the aorta below the subclavian artery but not by the ligation of the upper thoracic

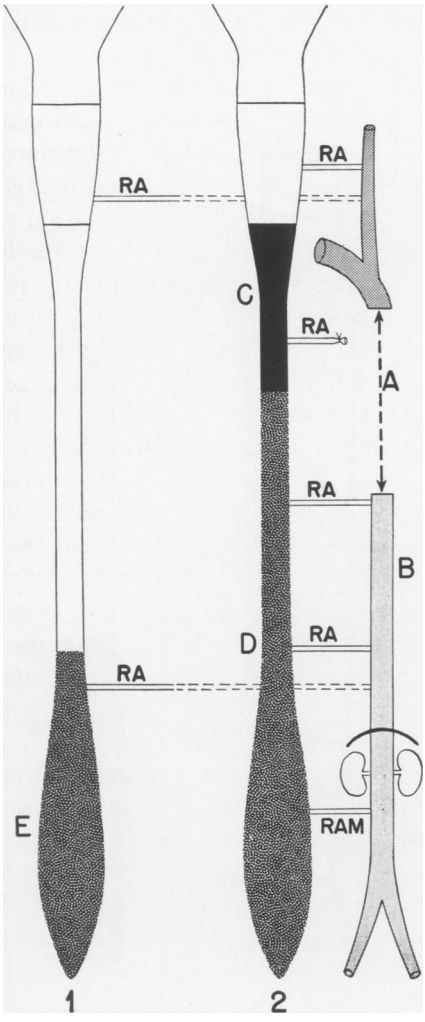


FIG. 9. *a*

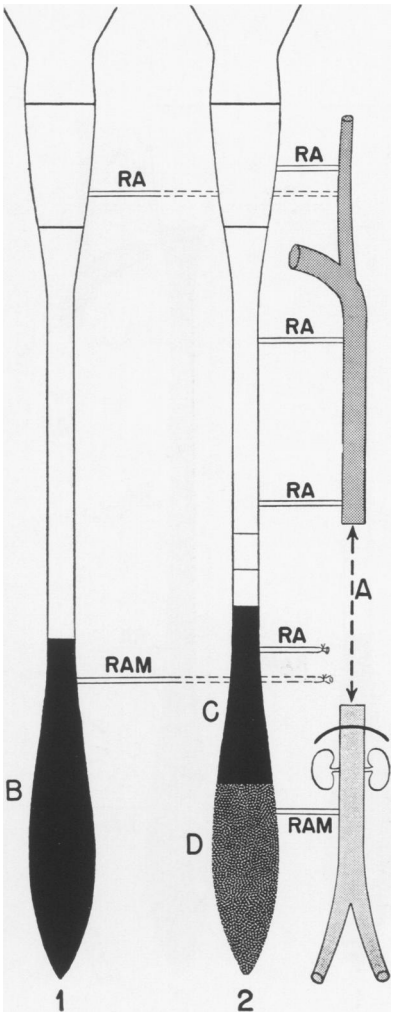


FIG. 9. *b*

FIG. 9. Interference with circulation of the spinal cord in suprarenal aortic resection without utilizing a shunt. *a*, Effect of resection of the proximal half of the thoracic aorta (*A*) without utilizing a shunt. Interference with the blood supply incomplete in both Type 1 and Type 2 in areas *E* and *D* (stippled) and complete in area *C* Type 2 where the upper spinal cord has a greater number of radicular arteries and consequently a less well developed tractus arteriosus anterior which does not adequately supply this region from above as in Type 1.

b, The effect of resection of the distal half of the thoracic aorta (*A*) without utilizing a shunt. Complete interference with the blood supply in the lumbosacral spinal cord in Type 1 (*B*) and complete interference in the distal thoracic and lumbar spinal cord in Type 2 (*C*) and incomplete interference in the lumbosacral supply in Type 2 (*D*).

intercostals. (2) When lesions occur after resection of the lower half of the thoracic aorta insufficiency of the collateral circulation will probably not have been caused by the temporary midthoracic aortic occlusion but by the occlusion of a low thoracic type of arteria radicularis magna. (3) In resection of the entire thoracic aorta the collateral circulation may be insufficient both as a result of the temporary aortic occlusion and by occlusion of a low thoracic type of arteria radicularis magna.

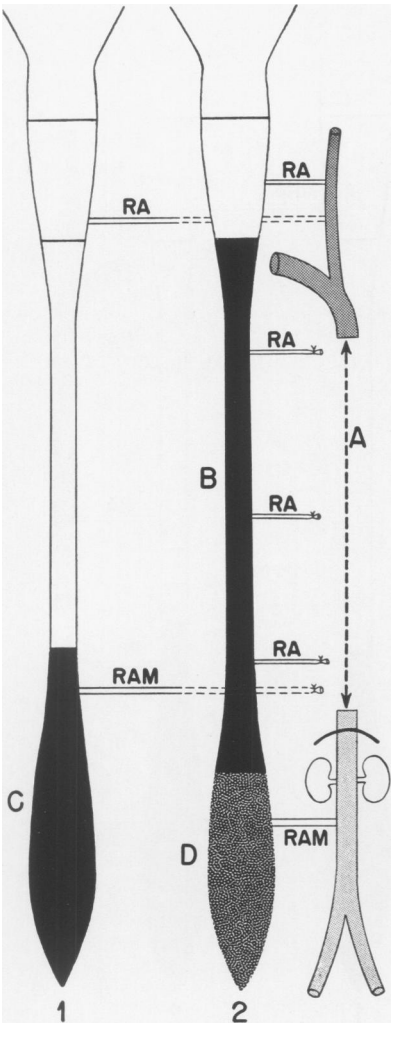


FIG. 9. *c*

FIG. 9. *c*, The effect of resection of the entire thoracic aorta distal to the left subclavian artery (*A*) without utilizing a shunt. Complete interference with the blood supply to the lumbosacral spinal cord in Type 1 (*C*) and complete interference in the thoracolumbar spinal cord in Type 2 (*B*) and incomplete interference in the blood supply in the lumbosacral spinal cord in Type 2 (*D*).

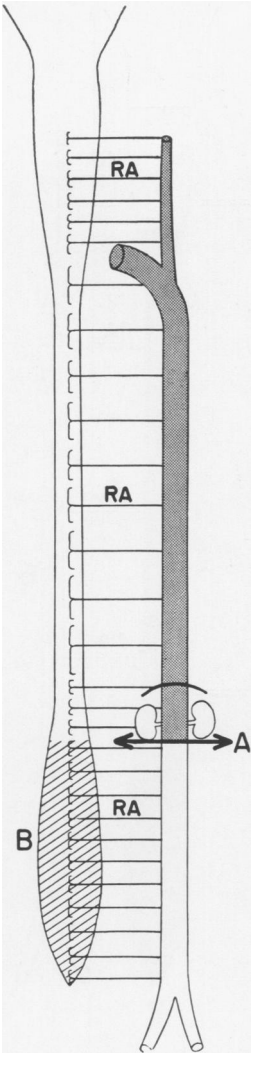


FIG. 10. *a*

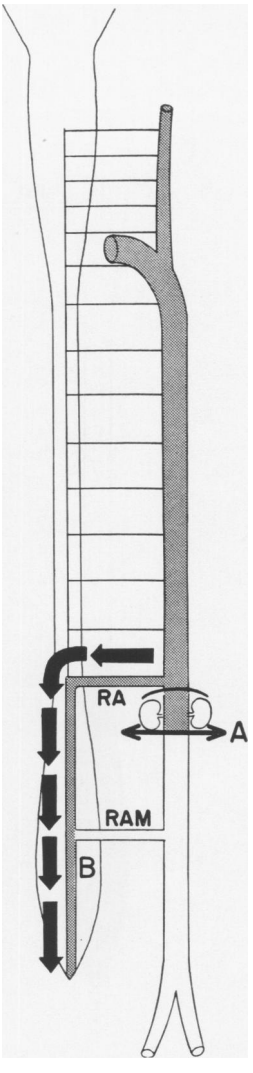


FIG. 10. *b*

FIG. 10. *a*, Steno's experiment in the rabbit. Occlusion of the abdominal aorta at *A* is always followed by ischemic necrosis of the lumbosacral spinal cord (*B*). This results from the purely segmental vascular supply and the resulting lack of development of collateral or anastomotic channels running on the anterior surface of the spinal cord.

b, Steno's experiment in the dog does not produce ischemic necrosis as in the rabbit due to the intermediate stage of desegmentation and collateral development approaching these developmental changes in man.

Considering these concepts of the collateral circulation with those on the differences in interference with normal spinal circulation it seems that in most cases the interference is such that the tractus arteriosus anterior will be able to supply sufficient collateral circulation: (1) The collateral circulation will be sufficient in cases of occlusion of any thoracic intercostals when the arteria radicularis magna is infrarenal because the tractus arteriosus anterior in the ischemic area is supplied from above and below by collateral blood. (2) In mid-

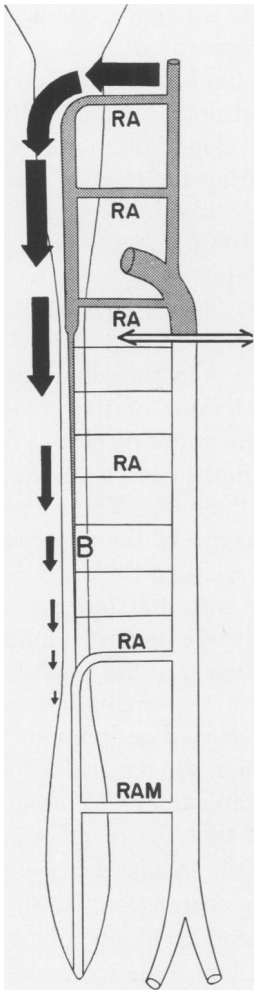


FIG. 11. *a*

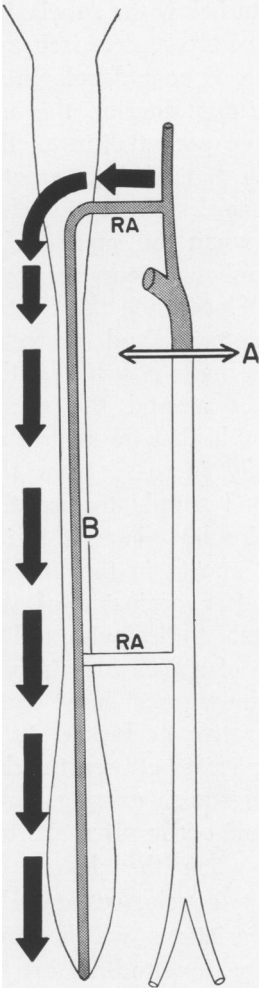


FIG. 11. *b*

FIG. 11. *a*, The effect of occlusion of the aorta distal to the left subclavian artery at *A* in a Subtype 2 with many persistent segmental vessels (RA) in the thoracic region and a resulting less well developed tractus arteriosus anterior (*B*) causing a very poor blood supply to the lower spinal cord during the occlusion, and ischemic damage may occur.

b, The effect of occlusion of the aorta distal to the left subclavian artery at *A* in a Subtype 1 with only two radicular arteries remaining and a very well developed tractus arteriosus anterior (*B*). Excellent blood supply to the lower spinal cord during occlusion, and ischemic damage is unlikely to occur.

thoracic aortic occlusion, it is the incomplete degree of interference with the blood supply which will enable the tractus to complete the necessary supply. (3) In occlusion of an infrarenal type of arteria radicularis magna sufficient collateral circulation will be given from the important and constantly present low thoracic radicular artery.

In two instances, however, the interference may be such that the tractus may be unable to supply sufficient collateral circulation: (1) in occlusion of a suprarenal type of arteria radicularis magna in which case such a large and completely ischemic area may be caused that in some cases a sufficiently rapid development of collateral circulation cannot be provided, and (2) in

occlusion of the aorta below the subclavian artery a large area of incomplete ischemia is caused and if there is poor development of the tractus arteriosus anterior, this not only may lead to poor potentialities of the collateral circulation but also to greater length of the spinal cord suffering interference, for example, when the supra-occlusional aorta only supplies the cervical cord. In this latter form of occlusion the tractus would carry the anterior and posterior blood supply. This is made possible by the extensive anastomosis around the spinal cord. It is also probable that the degree of anoxia will gradually increase from the level of normal blood supply toward the conus medullaris. Krogh [73] found this increase in degree of anoxia in the lumbosacral spinal cord after occlusion of the aorta in experimental studies with radiosodium. This degree of anoxia may in one individual be so profound as to cause damage during the operative occlusion. As a relation is thought to exist between the degree and duration of anoxia causing damage, this damage will occur after shorter times of occlusion when the degree of anoxia is greater. These conclusions are substantiated by the variations which were found to occur in the longitudinal extent of the ischemic lesions in the reported cases. In fact all were found to reach as low as the conus medullaris but they varied in their upper level of extent from the level of the lower lumbar to the level of the eighth thoracic. These variations can be explained as resulting from individual differences in the caudal extension of sufficient collateral supply to prevent ischemic damage for a given duration of occlusion. These variations can also be explained by the fact that the lesion will increase in length with longer duration of anoxia, as a result of both the gradual caudal increase of the degree of anoxia and the accepted relation between degree and duration of anoxia sufficient to cause a lesion.

In summary, damage seems to result after a type of procedure that causes ischemia of the lower spinal cord of sufficient extent and degree in an individual with a poorly developed tractus arteriosus anterior, which during the time of operative ischemia does not provide adequate caudal collateral circulation for the protection of the nervous tissue.

C. Spinal Ischemic Necrosis after Operation for Coarctation of the Aorta. A special aspect in this discussion of the occurrence of lesions of the spinal cord after suprarenal aortic surgery is the origin of ischemic lesions in cases of operation for coarctation of the aorta. These form a special problem because of their increased aortic collateral circulation. Occurrence and clinical similarity with the other cases of postoperative ischemic necrosis indicate a preoperative ischemia of the caudal spinal cord. In these cases the tractus anterior is always better developed and may even produce compression in the cervical spinal cord with ensuing paraplegia.[23, 52] This might lead to the belief that the distal spinal cord is provided mainly by blood coming from sources above the coarctation. With this belief the reported accidents might be explained by occlusion of the intercostals at the coarctation level or by occlusion of the left subclavian artery (Fig. 12 *a*). However, Maude Abbott [1] stated that in isthmic coarctation changes in the vascular system only occur in the neighborhood of the coarctation itself and that from the midthoracic aorta onward a normal circulatory pattern is preserved. This is substantiated in the spinal cord by autopsy findings of cases of coarctation from which it appears that dilatation of the tracti arteriosi only occurs in the cervical and upper third of the thoracic spinal cord.[41, 120] From this it appears likely that the lumbosacral spinal cord is supplied in a normal way through the arteria radicularis magna. Therefore, it seems more likely that ischemic lesions after operation

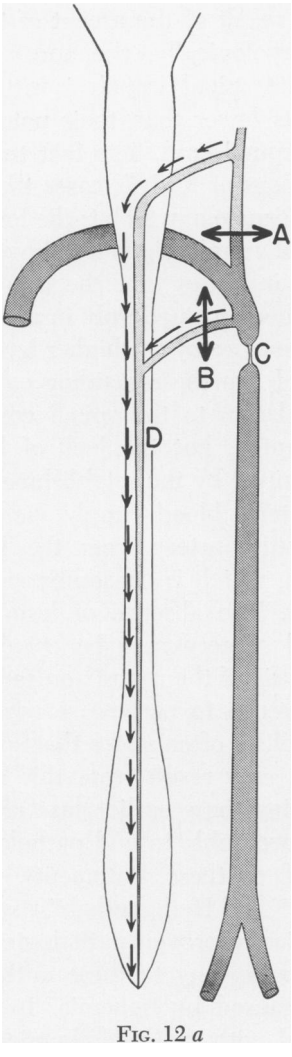


FIG. 12 *a*

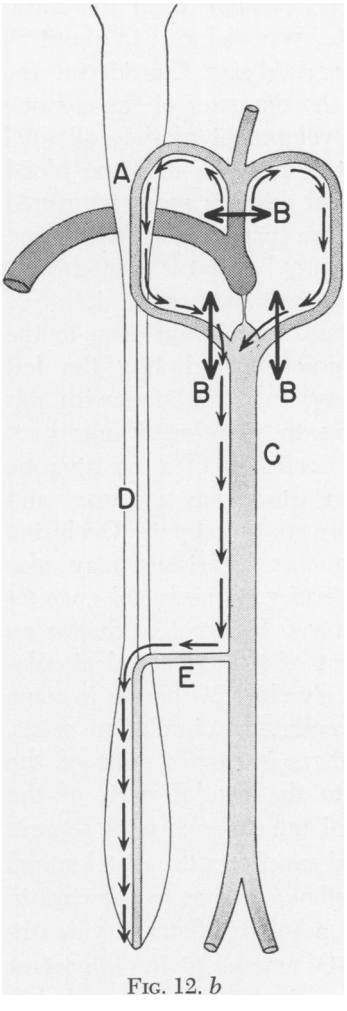


FIG. 12. *b*

FIG. 12. *a*, The theoretical, present concept of the circulation to the lower spinal cord in isthmic coarctation of the aorta. This supply is from the vertebrals and prestenotic intercostals via the "arteria spinalis anterior" (*D*). According to this concept, lower spinal cord ischemia can be caused by occlusion of the sources of supply to the anterior spinal artery at *A* and *B*.

b, Our new concept of the vascular supply of the lower spinal cord in isthmic coarctation is based on the excellent collateral circulation to the aorta distal to the stenosis and the presence of a normal pattern of aortal sources of blood supply to the lower spinal cord via the arteria radicularis magna (*E*). Marked interference with the collateral circulation to the aorta distal to the stenosis by barriers B,B,B can produce ischemic damage to the lumbosacral spinal cord.

for isthmic coarctation are produced similar to those in cases with normal aortic circulation and by interference with the circulation in the distal aorta (Fig. 12 *b*). It has been difficult to conceive how this can occur as it is generally agreed that only minor interference may be caused to the distal aorta by the operative occlusion in these cases. This may be due to the fact that it is often difficult to evaluate the diameter of the stenosis and that more blood may be passing than is suspected. This difficulty is partly caused by the fact that there is no direct relation between the amount of blood passing through a stenotic area and its diameter. In experimental

aortic stenosis Clatworthy [25] found little influence on blood passage until the intraluminal diameter was reduced to about 50 per cent of its normal size. Considering the importance of the diameter of the stenotic area on the development of the collateral circulation [89] it is possible that the blood flow to the distal aorta is more interfered with than has been suspected in some cases. Another factor may be that it is often necessary for technical reasons to interfere with the collateral circulation even to the extent of temporarily occluding the left subclavian artery. In connection with this it is interesting to mention Finnerty's [44] case in which occlusion of a poststenotic right subclavian artery was necessary and paraplegia followed the repair. Occlusion of a patent ductus arteriosus may also play a role. In cases of preductal coarctation the part played by a patent ductus on the development of the collateral circulation is not entirely clear. Although in some of them good collateral circulation exists, probably in others it carries most of the blood supply to the caudal parts of the body. Incidental interference with sources of normal blood supply to the distal spinal cord may also play a role as in our case in which during a previous bilateral sympathectomy intercostal arteries giving important branches to the spinal cord were probably occluded and although this produced no lesion at that time, it is probable that it played a role in causing damage during operation for the coarctation.

II. Infrarenal Aortic Surgery. In their anatomical explanation for the absence of spinal complications after infrarenal surgery, many authors concluded that interference with the blood supply to the spinal cord was impossible because in man the spinal cord reaches only to the first lumbar segment. This explanation is based on the concept that the spinal branches originate from the aorta at the same level as that at which they reach the spinal cord. In reality, as a result of the ascent of the spinal cord embryologically, the aortic origin of the lowest spinal vessels is situated five segments lower than their point of arrival in the spinal cord. The fact that in about 50 per cent of Kadyi's cases the arteria radicularis magna, which is the lowest important spinal vessel, originates above the renal vessels, indicates that the absence of lesions can be explained only in about 50 per cent of the cases by the higher level of the spinal vessels. In the remaining cases the normal circulation to the spinal cord may be interrupted, but the lack of lesions can be explained by the establishment of sufficient collateral blood supply via the important radicular artery from the lower thoracic aorta, which is constantly present in these cases. The absence of lesions after infrarenal surgery may be correlated with an analysis of the reports on saddle embolism. Reference to early case reports of saddle embolism often states that spinal complications may result from this disease. In reviewing these earlier case reports we have not been able to find pathologic or clinical proof of these statements. Only in Helbing [56] and Heiligenthal [55] cases were microscopically proven pathologic lesions found and according to these authors they were not caused by ischemia. In the only case found with typical ischemic lesions of the spinal cord, Alexander's [5] case, the embolus reached high above the renal vessels and produced renal and splenic infarction.

GENERAL FACTORS

Influence of the Blood Pressure. The importance of the blood pressure at the time of aortic occlusion has been mentioned by different authors in connection with neurologic deficit.[45, 99, 101, 118] Although the presence of nerve fibers in the blood vessels of the spinal cord was demonstrated by Clark [24] and by Penfield,[85] there has been no study on the influence of nervous control of the systemic blood pressure and of the

action of chemicals and drugs on the spinal circulation. The presence of a safety mechanism against fall in pressure similar to that in the brain has not been demonstrated.

Age. Of the patients with spinal complications reported in the literature the youngest with occlusion of a normal aorta was seven years, and of those who were operated upon for isthmic coarctation the youngest was eight years. Many observers, however, have shown the difficulty of producing experimental spinal ischemic damage to young animals.[74, 94, 102, 114] This was explained by the greater resistance of the spinal structures to anoxia,[57, 71, 115] although it has been proved experimentally that the oxygen requirement in the spinal cord of young animals is relatively greater.[115] It is very likely that during operation on the aorta the young individual is protected also against spinal damage by better potentialities of collateral circulation from the cranial spinal cord. In fact in the child Suh [106] demonstrated remakably pronounced anterior spinal vessels and on measurement the diameter of the tractus arteriosus anterior was almost equal to that of the adult. This may be related to the greater oxygen requirement. In isthmic coarctation it is possible that, besides these factors, the lack of localization of collateral circulation around the aortic stenosis in the young patient provides more protection against technical manipulations than does localization of collateral circulation in the older patient. In fact, collateral circulation usually becomes apparent only after five years. With increasing age and the development of arteriosclerotic changes the possibility of development of collateral circulation dwindles.

Hypothermia. In the spinal cord, van Harreveld [115] showed that the temperature coefficient of the processes ultimately resulting in destruction of nerve cells was consistently higher than that of the processes involved in the oxygen uptake of the spinal cord. Thus, as the temperature is lowered, destruction of nerve cells is decreased to a greater extent than is the oxygen uptake. As a result, hypothermia has been reported to be effective in the experimental prevention of paraplegia following temporary high occlusion of the aorta,[14, 40, 83, 84, 86] although experimental and clinical cases are reported in which paralysis occurred in spite of general and local hypothermia.[14, 42, 49, 84] Since first used by Oudot [82] in aortic surgery it has now been employed by many surgeons. One of the reasons for the clinical value of hypothermia in temporary occlusion is that in these cases circulation in the ischemic areas returns to normal when the temperature returns to normal. The question may be raised as to whether in cases of permanent occlusion of spinal vessels sufficient collateral circulation is actually established during the hypothermia to prevent spinal damage when the temperature is returned to normal. In brain tissue it was shown [72, 92] that the blood flow varied to the same extent as the oxygen consumption under hypothermia, and so far there is no consistent evidence concerning the vascular status during recovery from hypothermia. Imig [66] has shown that in cases in which the temperature is brought down to 27° C., blood flow is within normal values after hypothermia but it has been found [32, 100] that recovery to normal blood flow is often slow and incomplete. Experiments by Rosomoff [91] indicate that with permanent occlusion of the arteria cerebri media in the dog under hypothermia, the incidence of ischemic damage is much lower, but the incidence in normothermic dogs is indefinite. With this question in the balance, it is still possible that in aortic resection under hypothermia permanent occlusion of a thoracic type of arteria radicularis magna might cause ischemic damage. Eiseman [42] has recently reported a case of permanent damage to the spinal cord associated with resection of a lower thoracic aneurysm under hypothermia with the aorta clamped at the level of the sixth thoracic for 63 minutes. This well docu-

mented case and Eiseman's careful evaluation of the factors involved are an important contribution to this subject.

CONCLUSIONS

Twenty-four instances of ischemia of the spinal cord associated with aortic surgery have been gathered from reported and personal cases, 11 after operation for coarctation, seven after aortic resection, and six after temporary occlusion of the aorta. By comparing the procedures that cause these complications with the aortic procedures without complications as they have been reported in the literature and from consideration of the circulation of the spinal cord in relation to aortic surgery, the following conclusions were drawn:

1. The spinal cord is protected against damage during infrarenal aortic surgery either by the absence of infrarenal blood supply to the cord, or when present, by collateral supply from a constant and important low thoracic spinal vessel.

2. From all types of suprarenal aortic procedures, only temporary occlusion of the aorta below the subclavian artery and procedures including ligature of the lower thoracic intercostal vessels were found likely to cause spinal damage. The reason temporary occlusion of the aorta below the subclavian artery is followed by ischemic damage in one individual and not in another is explained by the extreme differences that are possible in the segmental pattern of the spinal cord supply. In the multisegmental type, occlusion of the aorta results in a greater interference with normal supply and poor potentialities of collateral circulation from the aorta above the occlusion via the tractus arteriosus anterior.

Ligation of the lower thoracic intercostals causes damage when it results in occlusion of a low thoracic type of arteria radicularis magna, supplying the major part of the lower spinal cord. Therefore, if the radicular arteries from the eighth to the twelfth thoracic are sacrificed there is a definite possibility of spinal damage.

For the same reason even when hypothermia or a shunt is used it becomes necessary to maintain adequate circulation to the lower spinal cord in some cases.

Therefore, a lesion in the lower thoracic aorta can be resected safely only if the arteria radicularis magna arises from the abdominal aorta, "roughly about 50 per cent" of Kadyi's series of cases.

In all other types of procedures either the interference with the normal blood supply is small or incomplete or else adequate sources of collateral blood supply are present.

In spite of hypothermia or shunt, aortic surgery may still cause spinal damage when the lower intercostals are occluded. Failure of hypothermia to protect the spinal cord from injury can be explained by the fact that nerve tissue may survive during the lowered metabolism and decreased circulation if the occlusion is temporary. If the occlusion is permanent, such as during resection, these same tissues may not survive when the normal temperature is resumed owing to the fact that collateral circulation has not developed adequately.

After surgical procedures for isthmic coarctation, damage to the spinal cord is caused by interference with the circulation to the distal aorta in the same manner as that produced in the presence of normal circulation. In addition, there is danger to the cord if any congenital shunts are present around the coarctation, such as a poststenotic ductus arteriosus or a poststenotic subclavian, either right or left, or if the degree of stenosis is relatively incomplete, "less than 50 per cent," since under these conditions adequate collateral circulation may not be established in the sense of the usual coarctation, and prolonged occlusion of the aorta cannot be tolerated.

The minimum safe time of occlusion of circulation of the spinal cord is still un-

known but it is shorter than is generally accepted and is probably under 18 minutes.

The young individual is protected against spinal damage by greater resistance to anoxemia of the spinal cord and better potentialities of collateral circulation resulting from relatively larger spinal vessels. The youngest individual with spinal damage was seven years old.

Lesions of the spinal cord are always localized in the lower spinal cord and variations of their clinical manifestations result from the differences in extent and degree of the ischemia.

Neurologic complications after aortic surgery may be caused by ischemia of peripheral nerves to the lower extremities as well, resulting from prolonged occlusion of the infrarenal aorta. In such instances the symptomatology may be similar to that of spinal ischemia but usually there is evidence of peripheral circulatory deficiency as well.

BIBLIOGRAPHY

1. Abbott, M. E.: Coarctation of Aorta of Adult Type; Statistical Study and Historical Retrospect of 200 Recorded Cases with Autopsy, of Stenosis or Obliteration of Descending Arch in Subjects above Age of 2 Years. Am. Heart J., **3**: 574, 1928.
2. Adamkiewicz, A.: Die Blutgefässe des menschlichen Rückenmarkesoberfläche. Sitz. d. akad. d. Wiss. in Wien. Math. Natur. Klass., **85**: 101, 1882.
3. Adams, H. D.: Shunt Graft with Resection for Aneurysm of Arch of Aorta. J. A. M. A., **159**: 1195, 1955.
4. Adams, R.: Unpublished data.
5. Alexander, A.: Zur Kenntnis der Rückenmarksveränderungen nach Verschluss der Aorta abdominalis. Ztschr. f. Klinische Med., **58**: 247, 1906.
6. Allbaugh, E. and S. M. Horvath: Effect of Total Occlusion of Thoracic Aorta on Blood Pressure, Splanchnic Blood Flow and Metabolic State in Dogs. Am. J. Physiol., **180**: 451, 1955.
7. Andrus, W. DeW.: Quoted by Reid,[88] M. R.
8. Antoni, N. and E. Lindgren: Steno's Experiment in Man as Complication in Lumbar Aortography. Acta chir. scandinav., **98**: 230, 1949.
9. Bahnson, H. T.: Definitive Treatment of Saccular Aneurysms of Aorta with Excision of Sac and Aortic Suture. Surg., Gynec. & Obst., **96**: 383, 1953.
10. Bahnson, H. T.: Considerations in Excision of Aortic Aneurysms. Ann. Surg., **138**: 377, 1953.
11. Bahnson, H. T.: Surgical Treatment of Thoracic Aneurysms. In Henry Ford Hospital International Symposium on Cardiovascular Surgery. Philadelphia, Saunders, 1955, pp. 434–447.
12. Bauer, Fritz: Fall von Embolus aortae abdominalis, Operation, Heilung. Zentralblatt für Chirurgie, **40**: 1945, 1913.
13. Beattie, E. J., Jr., J. J. Nolan and J. S. Howe: Paralysis Following Surgical Correction of Coarctation of Aorta; Case Report with Autopsy Findings. Surg., **33**: 754, 1953.
14. Beattie, E. J., Jr., D. Adovasio, J. M. Keshishian and B. Blades: Refrigeration in Experimental Surgery of Aorta. Surg., Gynec. & Obst., **96**: 711, 1953.
15. Beck, C. S.: Quoted by Izant, R. J., C. A. Hubay and W. D. Holden: Nonsuture Aortic Shunt—Experimental Study. Surg., **33**: 233, 1953.
16. Bing, R. J., J. C. Handelsman, J. A. Campbell, H. E. Griswold and A. Blalock: Surgical Treatment and Physiopathology of Coarctation of Aorta. Ann. Surg., **128**: 803, 1948.
17. Bolton, B.: Blood Supply of Human Spinal Cord. J. Neurol. & Psychiat., **2**: 137, 1939.
18. Boyarsky, S.: Paraplegia Following Translumbar Aortography. J. A. M. A., **156**: 599, 1954.
19. Burch, B. H., D. W. Traphagen and M. J. Folkman: Use of Aortic Occlusion in Abdominal Surgery with Report of 2 Human Cases; Preliminary Report. Surg., **34**: 672, 1953.
20. Burgess, C. M., G. C. Freeman, J. W. Cherry and A. S. Hartwell: The Surgical Treatment of Patent Ductus Arteriosus; Report of 14 Consecutive Cases. Hawaii M. J., **14**: 393, 1955.
21. Carrel, A.: On the Experimental Surgery of the Thoracic Aorta and the Heart. Ann. Surg., **52**: 83, 1910.
22. Chamberlain, J. M.: In discussion of J. D. Fryfogle, *et al.*: Rapid Vessel Anastomoses with Transient Arterial Interruption Employing Suture Methods over a Removable Bivalve Prosthesis. In Henry Ford Hospital International Symposium on Cardiovascular

Surgery. Philadelphia, Saunders, 1955, pp. 476–495.

23. Christian, P. and W. Noder: Akute Rückenmarkssyndrome bei Isthmusstenose der Aorta als Folge eines pathologischen Kollateralkreislaufs über die A. spinalis ant. Ztschr. für Kreislaufforsch., **43**: 125, 1954.
24. Clark, S. L.: Innervation of Blood Vessels of Medulla and Spinal Cord. J. Comp. Neurol., **48**: 247, 1929.
25. Clatworthy, H. W., Y. Sako, T. C. Chisholm, C. Culmer and R. L. Varco: Thoracic Aortic Coarctation: Its Experimental Production in Dogs, with Special Reference to Technical Methods Capable of Inducing Significant Intraluminal Stenosis. Surg., **28**: 245, 1950.
26. Conklin, W. S. and E. Watkins, Jr.: Use of Potts-Smith-Gibson Clamp for Division of Patent Ductus Arteriosus. J. Thorac. Surg., **19**: 361, 1950.
27. Cooley, D. A.: In: Panel Discussion on Advantages and Disadvantages of Various Methods of Inducing Hypothermia. In Henry Ford Hospital International Symposium on Cardiovascular Surgery. Philadelphia, Saunders, 1955, pp. 408–431.
28. Cooley, D. A. and M. E. DeBakey: Surgical Considerations of Intrathoracic Aneurysms of Aorta and Great Vessels. Ann. Surg., **135**: 660, 1952.
29. Cooley, D. A. and M. E. DeBakey: Resection of the Thoracic Aorta with Replacement by Homograft for Aneurysms and Constrictive Lesions. J. Thorac. Surg., **29**: 66, 1955.
30. Crafoord, C.: In: Ekström, G.: The Surgical Treatment of Patent Ductus Arteriosus. Acta chir. scandinav., Supplement **169**: 1 and 109, 1952.
31. Crafoord, C., B. Ejrup and H. Gladnikoff: Coarctation of Aorta. Thorax, **2**: 121, 1947.
32. Crismon, J. M. and F. A. Fuhrman: Studies on Gangrene Following Cold Injury: Capillary Blood Flow After Cold Injury, Effects of Rapid Warming and Sympathetic Block. J. Clin. Investigation, **26**: 468, 1947.
33. Czerny: Ueber Nierenexstirpation. Centralblatt f. Chir., **6**: 737, 1879.
34. DeBakey, M. E. and D. A. Cooley: Successful Resection of Aneurysm of Thoracic Aorta and Replacements by Graft. J. A. M. A., **152**: 673, 1953.
35. DeBakey, M. E., D. A. Cooley and O. Creech, Jr.: Aneurysms and Occlusive Diseases of the Aorta. Analysis of 203 Cases Treated by Resection and Homograft Replacement. In Henry Ford Hospital International Symposium on Cardiovascular Surgery. Philadelphia, Saunders, 1955, pp. 468–475.
36. DeBakey, M. E., D. A. Cooley and O. Creech, Jr.: Surgical Considerations of Dissecting Aneurysm of the Aorta. Ann. Surg., **142**: 586, 1955.
37. De Buck, D. and L. De Moor: Lésions des cellules nerveuses sous l'influence de l'anémie aiguë. Le Névraxe, **2**: 3, 1901.
38. Dripps, R. D.: Quoted by Johnson, J. *et al.*[68]
39. Dubost, Ch. and Cl. Dubost: Traitement chirurgical des anévrysmes de l'aorte. Les possibilités d'exérèse. J. chir., **69**: 581, 1953.
40. Edwards, W. S., P. P. Salter, Jr. and V. A. Carnaggio: Intraluminal Aortic Occlusion as a Possible Mechanism for Controlling Massive Intra-Abdominal Hemorrhage. Surgical Forum, Philadelphia, Saunders, Vol. 4, 1953, pp. 496–499.
41. Edwards, J. E., O. T. Clagett, R. L. Drake and N. A. Christensen: Collateral Circulation in Coarctation of Aorta. Proc. Staff Meet., Mayo Clinic, **23**: 333, 1948.
42. Eiseman, B. and W. B. Summers: Factors Affecting Spinal Cord Ischemia During Aortic Occlusion. Surg., **38**: 1063, 1955.
43. Evans, A. T.: Quoted by Boyarsky, S.[18]
44. Finnerty, J.: Discussion. J. Thorac. Surg., **29**: 1955.
45. Gelfan, S. and I. M. Tarlov: Differential Vulnerability of Spinal Cord Structures to Anoxia. J. Neurophysiol., **18**: 170, 1955.
46. Gerard, R. W.: Brain Metabolism and Circulation. The Circulation of the Brain and Spinal Cord: A Symposium on Blood Supply. Assoc. Res. Nerv. & Ment. Dis., Baltimore, Williams & Wilkins Co., Vol. 18, 1938, pp. 316–345.
47. Gildea, E. F. and S. Cobb: Effects of Anemia on Cerebral Cortex of Cat. Arch. Neurol. & Psychiat., **23**: 876, 1930.
48. Gross, R. E.: Coarctation of Aorta. Circulation, **7**: 757, 1953.
49. Gross, R. E. and C. A. Hufnagel: Coarctation of Aorta: Experimental Studies Regarding its Surgical Correction. New England J. Med., **233**: 287, 1945.
50. Guinard, A.: Traitement des anévrysmes de la base du cou par la ligature simultanée de la carotide primitive et de la sous-clavière droite ligature de l'aorte thoracique. Rev. d. chir., **39**: 229, 1909.
51. Gwathmey, O. and C. W. Thompson: Aneurysm Formation in a Homologous Aortic Graft in a Human; Report of a Case. J. Thorac. Surg., **30**: 218, 1955.

52. Haberer, H.: Ein Fall von seltenem Collateralkreislauf bei angeborener Obliteration der Aorta und dessen Folgen. Ztschr. f. Heilk., **24**: 26, 1903.
53. Hamburger, M., Jr. and E. B. Ferris, Jr.: Dissecting Aneurysm; Study of 6 Recent Cases. Am. Heart J., **16**: 1, 1938.
54. Hardin, C. A., K. R. Reismann and E. G. Dimond: The Use of Hypothermia in the Resection and Homologous Graft Replacement of the Thoracic Aorta. Ann. Surg., **140**: 720, 1954.
55. Heiligenthal: Embolie der Aorta abdominalis. Deutsche med. Wchnschr., **24**: 519, 1898.
56. Helbing, C.: Zur Kenntniss der Rückenmarksveränderungen beim Menschen nach Thrombose der Aorta abdominalis. Deutsche med. Wchnschr., **22**: 672, 1896.
57. Heymans, C.: Survival and Revival of Nervous Tissues after Arrest of Circulation. Physiol. Rev., **30**: 375, 1950.
58. Himwich, H. E.: Brain Metabolism and Cerebral Disorders. Baltimore, The Williams & Wilkins Co., 1951, 451 pp.
59. His, W.: Zur Geschichte des menschlichen Rückenmarkes und der Nervenwurzeln. Abhandl. der K. Sachs. Gesellschaft der Wissenschaften. 22. Mathem. Phys. Classe 13, Leipzig, 1887.
60. Hoche, A.: Experimentelle Beiträge zur Pathologie des Rückenmarkes. I: Die Veränderungen im Rückenmarke nach aseptischer Embolie. Arch. f. Psychiat. u. Nerven., **32**: 209, 1899.
61. Hoche, A.: Vergleichend-Anatomisches über die Blutversorgung der Rückenmarksubstanz. Ztschr. f. Morphol. u. Anthrop., **1**: 241, 1899.
62. Hofmann, M.: Zur vergleichenden Anatomie der Gehirn und Rückenmarksarterien der Vertebraten. Ztschr. f. Morphol. u. Anthrop., **2**: 247, 1900.
63. Hol, R. and O. Skjerven: Spinal Cord Damage in Abdominal Aortography. Acta radiol., **42**: 276, 1954.
64. Holman, E., F. Gerbode and A. Purdy: Patent Ductus; Review of 75 Cases with Surgical Treatment Including Aneurysm of Ductus and One of Pulmonary Artery. J. Thorac. Surg., **25**: 111, 1953.
65. Hughes, C. W.: Use of an Intra-Aortic Balloon Catheter Tamponade for Controlling Intra-Abdominal Hemorrhage in Man. Surg., **36**: 65, 1954.
66. Imig, C. J., W. J. Roberson, M. Gault and H. M. Hines: Blood Flow in the Hind Legs of Dogs after Exposure to Cold. Am. J. Physiol., **181**: 395, 1955.
67. Javid, H., W. S. Dye, W. J. Grove and O. C. Julian: Resection of Ruptured Aneurysms of the Abdominal Aorta. Ann. Surg., **142**: 613, 1955.
68. Johnson, J., C. K. Kirby and H. B. Lehr: A Method of Maintaining Adequate Blood Flow through the Thoracic Aorta while Inserting an Aorta Graft to Replace an Aortic Aneurysm. Surg., **37**: 54, 1955.
69. Julian, O. C., W. J. Grove, W. S. Dye, M. S. Sadove, H. Javid and R. F. Rose: Hypotension and Hypothermia in Surgery of the Thoracic Aorta. Arch. Surg., **70**: 729, 1955.
70. Kadyi, H.: Ueber die Blutgefässe des menschlichen Rückenmarkes, nach einer im XV. Bande der Denkschriften der Math. Natur. Classe der Akad. d. Wissenschaften in Krakau erschienenin Monographie. Aus dem Polnischen überzetzt vom Verfasser. Gubrynowicz and Schmidt, Lemberg, 1889.
71. Kety, S. S.: Changes in Cerebral Circulation and Oxygen Consumption Which Accompany Aging and Maturition: In Waelsch, H.: Biochemistry of the Developing Nervous System. New York, Academic Press, 1955.
72. Kleinerman, J. and A. L. Hopkins: Effects of Hypothermia on Cerebral Blood Flow and Metabolism in Dogs. Federation Proc., **14**: 410 (Mar.) (Pt. 1), 1955.
73. Krogh, E.: Studies on Blood Supply to Certain Regions in Lumbar Part of Spinal Cord. Acta physiol. scandinav., **10**: 271, 1945.
74. Krogh, E.: Effect of Acute Hypoxia on Motor Cells of Spinal Cord. Acta physiol. scandinav., **20**: 263, 1950.
75. Kümmel: Chirurgie des Aortenaneurysmas. Deutsche med. Wchnschr., **40**: 731, 1914.
76. Lam, C. R. and H. H. Aram: Resection of Descending Thoracic Aorta for Aneurysm; Report of Use of Homograft in Case and Experimental Study. Ann. Surg., **134**: 743, 1951.
77. Moersch, F. P. and G. P. Sayre: Neurologic Manifestations Associated with Dissecting Aneurysm of Aorta. J. A. M. A., **144**: 1141, 1950.
78. Mosberg, W. H., Jr., H. C. Voris and J. Duffy: Paraplegia as a Complication of Sympathectomy for Hypertension. Ann. Surg., **139**: 330, 1954.
79. Mustard, W. T.: Personal communication.
80. Mustard, W. T., R. D. Rowe, J. D. Keith and A. Sirek: Coarctation of the Aorta with

Special Reference to the First Year of Life. Ann. Surg., **141**: 429, 1955.

81. Nomina Anatomica: In: Verhandlungen der Anatomischen Gesellschaft, **43**: 323, 1936.
82. Oudot, J.: La greffe vasculaire dans les thromboses du carrefour aortique. Presse méd., **59**: 234, 1951.
83. Owens, J. C., A. E. Prevedel and H. Swan: Prolonged Experimental Occlusion of the Thoracic Aorta During Hypothermia. Arch. Surg., **70**: 95, 1955.
84. Parkins, W. M., M. Ben and H. M. Vars: Tolerance of Temporary Occlusion of the Thoracic Aorta in Normothermic and Hypothermic Dogs. Surg., **38**: 38, 1955.
85. Penfield, W.: Intracerebral Vascular Nerves. Arch. Neurol. & Psychiat., **27**: 30, 1932.
86. Pontius, R. G., H. L. Brockman, E. G. Hardy, D. A. Cooley and M. E. DeBakey: The Use of Hypothermia in the Prevention of Paraplegia Following Temporary Aortic Occlusion: Experimental Observations. Surg., **36**: 33, 1954.
87. Reid, M. R.: Aneurysms in Johns Hopkins Hospital; All Cases Treated in Surgical Service from Opening of Hospital to Jan. 1922. Arch. Surg., **12**: 1 (Pt. 1), 1926.
88. Reid, M. R.: A Report of Vascular Lesions. Am. J. Surg., **14**: 17, 1931.
89. Reifenstein, G. H., S. A. Levine and R. E. Gross: Coarctation of Aorta; Review of 104 Autopsied Cases of "Adult Type," 2 Years of Age or Older. Am. Heart J., **33**: 146, 1947.
90. Rexed, B.: Some Observations on Effect of Compression of Short Duration of Abdominal Aorta in Rabbit. Acta psychiat. et neurol., **15**: 365, 1940.
91. Rosomoff, H. L.: Occlusion of the Middle Cerebral Artery under Hypothermia. Federation Proc., **14**: 125 (Pt. 1), 1955.
92. Rosomoff, H. L. and D. A. Holaday: Cerebral Blood Flow and Cerebral Oxygen Consumption during Hypothermia. Am. J. Physiol., **179**: 85, 1954.
93. Rubio, R.: Complicaciones neurológicas en la cirugía de la hipertensión arterial. Bol. Soc. cir. Uruguay, **23**: 439, 1952.
94. Sako, Y., T. C. Chisholm, K. A. Merindino and R. L. Varco: Experimental Evaluation of Certain Methods of Suturing Thoracic Aorta. Ann. Surg., **130**: 363, 1949.
95. Sandblom, P. and G. Ekström: Surgical Treatment of Wide Patent Ductus Arteriosus. Acta chir. scandinav., **102**: 167, 1951.
96. Santy, P.: Les formes anatomiques des sténoses de l'isthme de l'aorte. Acta chir. belg., **52**: 551, 1953.
97. Schafer, P. W. and C. A. Hardin: Use of Temporary Polythene Shunts to Permit Occlusion, Resection, and Frozen Homologous Graft Replacement of Vital Vessel Segments; Laboratory and Clinical Study. Surg., **31**: 186, 1952.
98. Schiffer, J.: Ueber die Bedeutung des Stenson'schen Versuchs. Centralblatt f. die medicinischen Wissenschaften, **7**: 593, 1869.
99. Shumacker, H. B., Jr.: Use of Subclavian Artery in Surgical Treatment of Coarctation of Aorta. Surg., Gynec. & Obst., **93**: 491, 1951.
100. Shumacker, H. B., Jr.: Sympathectomy in Treatment of Frostbite. Surg., Gynec. & Obst., **93**: 727, 1951.
101. Sealy, W. C. and G. H. McSwain: Method for Producing Coarctation of Thoracic Aorta in Dogs. Surg., **25**: 451, 1949.
102. Spronck, C. H. H.: Contribution à l'étude expérimentale des lésions de la moelle épinière déterminées par l'anémie passagère de cet organe. Arch. physiol. norm. et pathol., 4th Series, Vol. I, pp. 1–31, 1888.
103. Stenonis, Nicolai (Stensen, Niels): Elementorum myologiae specimen: seu Musculi descriptio Geometrica. Ferdinandum 11. Magnum Amstelodami Apud Johan. Janssonium à Waesberg, & Viduam Elizei Weyerstraet, 1669.
104. Sterzi, G.: Die Blutgefässe des Rückenmarks. Untersuchungen über ihre Vergleichende Anatomie und Entwickelungsgeschichte. Anatomische Hefte, **24**: 1, 1904.
105. Stranahan, A., R. D. Alley, W. H. Sewell and H. W. Kausel: Aortic Arch Resection and Grafting for Aneurysm Employing an External Shunt. J. Thorac. Surg., **29**: 54, 1955.
106. Suh, T. H. and L. Alexander: Vascular System of Human Spinal Cord. Arch. Neurol. & Psychiat., **41**: 659, 1939.
107. Swammerdam, Jan: Tractatus physico-anatomico-medicus de respiratione usuque pulmonum. Lugduni Batavorum. D. Abraham and A a Gaasbeeck, 1667.
108. Swan, H., C. Maaske, M. E. Johnson and R. Grover: Arterial Homografts; Resection of Thoracic Aortic Aneurysm Using Stored Human Arterial Transplant. Arch. Surg., **61**: 732, 1950.
109. Sweet, R.: Unpublished data.

110. Tanon, L.: Les arteres de la moelle dorsolombaire. Considérations anatomiques et cliniques. These de Paris, Vigot, Paris, 1908, 75 pp.
111. Tureen, L. L.: Effect of Experimental Temporary Vascular Occlusion on Spinal Cord; Correlation Between Structural and Functional Changes. Arch. Neurol. & Psychiat., **35**: 789, 1936.
112. Tureen, L. L.: Circulation of the Spinal Cord and the Effect of Vascular Occlusion. In: The Circulation of the Brain and Spinal Cord: A Symposium on Blood Supply. Assoc. Res. Nerv. & Ment. Dis., Baltimore, Williams & Wilkins Co., Vol. 18, 1938, pp. 394–437.
113. van Harreveld, A.: On Spinal Shock. Am. J. Physiol., **129**: 515, 1940; Abstr., Proc. Nat. Acad. Sc., **26**: 65, 1940.
114. van Harreveld, A. and D. B. Tyler: Metabolism of Asphyxiated Spinal Cord. Am. J. Physiol., **138**: 140, 1942.
115. van Harreveld, A. and D. B. Tyler: Influence of Temperature on Spinal Cord Damage Caused by Asphyxiation. Am. J. Physiol., **142**: 32, 1944.
116. van Harreveld, A., G. A. Feigen and L. S. Lerman: Hemodynamics of Aortic Occlusion. Am. J. Physiol., **157**: 168, 1949.
117. Villar, F.: Anévrysme de la crosse de l'aorte—De la ligature temporaire de l'aorte thoracique—Voie de pénétration chirurgicale dans le médiastin postérieur. J. de Med. de Bordeaux, **22**: 317, 1892.
118. Wagner, J. A. and J. Alvarez de Choudens: Unpublished Experiments. Quoted by Mosberg, W. H. *et al.*[78]
119. Watkins, E., Jr.: Circulatory Changes Produced by Clamping of Thoracic Aorta. Surg., **22**: 530, 1947.
120. Wyburn-Mason, R.: The Vascular Abnormalities and Tumours of the Spinal Cord and Its Membranes. St. Louis, Mosby, 1944.

DISCUSSION.—DR. WILLIAM S. MCCUNE, Washington, D. C.: I wish to congratulate Dr. Adams on this very thorough and excellent discussion of a highly important subject. We have had the misfortune of having two patients who have developed varying degrees of motor and sensory paralysis following operations on the abdominal aorta. In both of these instances the paralysis appeared immediately after the patient came out of the anesthetic. In both cases also there was some bilateral extremity cyanosis indicating perhaps that the paralysis may have been due to thrombosis of peripheral vessels with ischemia of the corresponding nerves.

In the first patient the paralysis was partial, of a flaccid type, and was accompanied by a corresponding degree of sensory loss. This patient otherwise progressed fairly well for three days, but died suddenly of coronary occlusion. At the post mortem examination there was a partial occlusion of one femoral artery.

The second patient was found to have bilateral spastic paralysis with sensory loss extending above the crests of the ilium on each side immediately aftter operation. In this particular case the aorta had been occluded temporarily in the thoracic region during the operation because of severe hemorrhage. Another interesting finding was the presence of a very large lumbar vessel, almost as large as the hypogastric artery, which extended directly backward from the abdominal aorta.

These two cases had several points in common which may be of interest from the point of view of etiology of the paralysis. In both instances the resections were for ruptured aneurysms of the abdominal aorta. In both instances the patients were in fairly severe shock at the time of operation, in spite of rapid blood replacement.

In both instances when the abdomen was opened the hemorrhage was found to be severe, and it was necessary to clamp off the aorta almost immediately below the renal vessels. In one instance it temporarily was occluded in the thoracic region. This prolonged the length of clamping of the aorta.

In both instances the aneurysms were very large and extended down to involve not only the common iliac but part of the external iliac vessels, and when an orlon prosthesis was sutured in place it was impossible to reestablish the hypogastric circulation.

In reviewing these findings it was our impression that the large lumbar vessel may have been carrying a great deal of the circulation to the spinal cord. Furthermore, it has seemed to us that the hypogastric vessels may supply some circulation to the cord, although this has never been demonstrated satisfactorily.

At the present time we are injecting the hypogastric vessels of autopsy specimens in an endeavor to find out whether or not any circulation reaches the spinal cord from them.

Shock is also of importance. In an already impaired circulation severe hypotension must add to the ischemia of the chord.

I congratulate Dr. Adams again. Thank you.