Part X

Factors influencing results

Perioperative Specific Complications in Aortoiliofemoral Reconstruction

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All lower-extremity revascularisation procedures have a rate of general and specific complications related to patient's status and to surgical action.

Acute general complications comprise cardiac complications, deep vein thrombosis and wound infection. The cardiac complications, especially myocardial ischemia, remain the most common cause of morbidity and mortality after surgical revascularization procedures for peripheral arterial diseases. A high rate of these deaths are related to atherosclerotic heart disease (1) and the incidence of myocardial infarction after aortoiliofemoral reconstruction is 0,8 - 5.2 % (2,3).

In 1997 a randomised prospective trial on deep venous thrombosis prophylaxis in aortic surgery (4) did not show any increased incidence of deep vein thrombosis after aortic surgery in the control group compared with the patients which had received prophylaxis therapy.

In the same year, another study on deep vein thrombosis following vascular surgery (5) reported an incidence of 9.8% after aortic or distal revascularization. The patients undergoing amputation showed the highest incidence of deep vein thrombosis (5).

The incidence of wound infection in aortoiliofemoral reconstruction is often related to groin infection after aortofemoral bypass. The incidence of laparotomic midline wound infection is very low. The incidence of wound infection is higher when aortofemoral reconstruction is associated to a procedure for inguinocrural disease.

The incidence of death after aortofemoral reconstruction is reported from 0 to 3.3% and it is usually related in the clinical series to myocardial complications (6,7,8).

In this chapter, we present acute specific complications after aortofemoral reconstruction (Table I).

TABLE I

Perioperative specific complications in aortoiliofemoral reconstruction
Acute graft limb thrombosis
Spinal cord ischemia
Acute renal failure
Ureteral injury
Groin lymphatic complications (lymphatic fistula/lymphocele)
Bowel ischemia

Acute graft limb thrombosis

Acute graft limb thrombosis occurs rarely after aortic surgery for occlusive disease. When this complication occurs it is related to acute thrombosis of the graft or, more frequently, of its limb and the reported rates are 1 - 3% (9).

The causes are usually twisting or kinging of the graft limb or technical problems at the distal anastomosis site, particularly the femoral anastomosis site.
In other cases, acute graft occlusion and acute leg ischemia can occur as result of intraoperative or perioperative thromboembolism.

Spinal cord ischemia

Since the first clinical report by McCune (10) in 1956, the cases reported in the literature are 111, seventhy (63%) for abdominal aortic disease (39 ruptured abdominal aortic aneurysm) and fourthy- one (37%) for aortoiliac occlusive disease (11).

The spinal cord ischemia most frequently occurs after emergency operation for ruptured abdominal aortic aneurysm.

Spinal cord ischemia after abdominal aortic surgery is a rare but devasting event that continues to haunt the vascular surgery. Szilagyi in 1978 (12) noted only a 0.25% of incidence of paraplegia or parapareris after 3614 abdominal aortic procedures. The same author reported that incidence of paraplegia after abdominal aortic aneurysm is 1 : 400 and after aortoiliac reconstruction for occlusive disease is 1 : 5000 (13).

The symptoms of spinal cord ischemia of the lower extremities include sensory and motor deficits associated with bladder/rectal incontinence with conservation of vibratory and propriocettive sensation, thus reflecting damage to the anterior horn of the spinal cord.

The etiology of spinal cord ischemia includes interruption of the arteria radicularis magna, prolonged aor-

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tic occlusion, intraoperative hypotension, atheromatous embolization and interruption of the internal iliac artery circulation (14,15,16) (Table II).

TABLE II

Etiology of spinal cord ischemia after abdominal aortic surgery (13)
Interruption of the arteria radicularis magna (Adamkiewicz's artery)
Prolonged aortic occlusion
Intraoperative hypotension
Atheromatous embolization
Interruption of the internal iliac artery circulation

Older studies (17,18) indicated that the arteria radicularis magna was the major source of blood supply to the anterior spinal artery in the distal thoracolumbar spinal cord region. Later studies (19,20) on the contrary have demonstred the importance of the blood supply from the lumbar aorta and hypogastric vessels to the spinal cord.

The blood supply to the cord consists of an intrinsic and an extrinsic network of arteries (14,15,21).

The intrinsic blood supply is composed of three longitudinal systems: one anterior and two posterior spinal arteries.

Intramedullary anastomoses between the anterior and the posterior spinal systems are nonexistent along the circumference, where the arterial vasa coronae coalesces. The anterior spinal artery extends the full 40cm to 45cm length of the spinal cord, but it can be so narrow at the distal thoracic level that it cannot compesate for an interrupted radicular artery, thus increasing the risk of infarction after loss of a single radicular artery (22).

The anterior spinal artery is, therefore, the major indipendent source of spinal cord perfusion and inadvertent interruption of the largest of its segmental radicular arteries has been implicated as the principal cause of spinal cord ischemia (14).

The extrinsic blood supply to the spinal cord is a segmental medullary artery that supply the anterior median spinal artery and the spinal arteries, which are present at each vertebral level.

The anterior spinal artery is fed by these segmental medullary arteries, which originate from the subclavian, intercostals and lumbar arteries. Perfusion of the distal spinal cord is derived from the lumbar, ileolumbar and lateral sacral arteries that anastomose with intrinsic spinal arteries at the level of the conus medullaris.

The arteria radicularis magna is located between T9 and T12 in 75% of the cases, but if it originates abnormally high, or is compromised, these lower lumbar arteries that anastomose with the intrinsic network may contribute significantly to the blood supply of the terminal spinal cord (15).

In general, when the arteria radicularis magna is compromised, the pelvic blood supply may be critically important.

Spinal cord ischemia after abdominal aortic reconstructive procedures is multifactorial and it remains unpredictable, random and unpreventivable. However, the avoidance of profound hypotension, prolonged clamping (especially supraceliac clamping), embolization and revascularization of internal iliac artery in selected case, may minimize the possibility for spinal cord injury (Table III,IV,V).

TABLE III

Types of ischemic spinal cord injury–Pathological findings (15)	
Type I:	cord injury associated with global ischemia to the distal cord and the conus
Type II:	anterior spinal column lesion
Type III and IV:	nerve roots and lumbosacral plexus lesion
Type V:	segmental spinal cord infarction
Type VI:	posterior spinal column ischemia

TABLE IV

Types of ischemic spinal cord injury – Clinical manifestations (15)	
Type I:	flaccid paraplegia
Type II:	anterior spinal artery syndrome – flaccid paraplegia, loss of pain and temperature sensation with preservation of proprioception and vibration sensation.
Type III and IV:	asymmetric motor and sensitivity deficits with or without bladder and bowel incontinence
Type V:	lesion to the cord at the specific level caused by embolization or hypoperfusion
Type VI:	loss of proprioception and vibratory sensitivity alone

TABLE V

Types of ischemic spinal cord injury – Prognosis (11)	
Type I:	poor long term recovery
Type II:	frequently seen after thoracic or thoracoabdominal reconstruction (minimal long term improvement)
Type III e IV:	favourable prognosis because the lesions are at the axonal level
Type V:	favorable
Type VI:	favorable

Acute renal failure

Acute renal failure remains a significant complication of aortic reconstructive procedures (23,24).

The reported incidence in aortoiliac bypass is 0 to 4.6%, with previously renal dysfunction that increases risk (25,26). One important etiologic factor is reduction and redistribution of renal artery blood flow with cross clamping application (27).

In 1959 Powers (28) found that the aortic cross clamp application in healthy dogs caused significant reduction in renal artery blood flow and this was associated with oliguria and histologic features of acute tubular necrosis.

Nanson (29), in the same year, obtained similar results in terms of renal function and histologic features, but did not observe significant reduction of renal artery blood flow. Others works (30,31,32) found only a transient reduction in renal artery blood flow with minimal effects on renal function.

Abbot in 1973 (33), using a 133 xenon washout technique to measure the renal artery blood flow, described the phenomenon of corticomedullary shunting. He noted an immediate reduction in total renal artery blood flow on cross-clamp application and a return to normal after 90 minutes of occlusion. There was a significant reduction in flow to the renal cortex, that paralleled the effect on total renal artery blood flow whereas there was an accompaning increase in medullary flow. These changes were progressive with time throughout the occlusion period and persisted for at least one hour after clamp removal.

In 1977 Cronewett (34) using different radioactive substance could demonstrate neither a decrease in renal artery blood flow nor corticomedullary shunting.

In retrospective clinical studies the acute renal failure is related with peripoperative hypotension (35), preoperative renal impairment (23), renal vein ligation (36,37), renal artery atheroembolism (38), increased intrabdominal pressure after operation (39) (Table VI).

In 1984 Gamulin (40) reported significant reduction in renal artery blood flow and corticomedullary shunting associated with cross clamping in human beings.

TABLE VI

Historically advocated factors of acute renal failure in aortofemoral reconstructions
Cross-clamping
Perioperative hypontension
Preoperative renal impairment
Renal vein ligation
Renal artery atheroembolism
Increased abdominal post-operative pressure

Although sympathetic – mediated vasocostriction has been implicated (41), pharmacologic blockade did not abolish the response (42). Other factors, such as increased renin release (43), renal prostaglandins (44) and kallikrein (45) may also be involved (Table VII).

TABLE VII

Factors involved in renal failure during aortofemoral surgery (41-45)
Sympathetic-mediated vascocostrction
Renin release
Renal prostaglandins
Kallicrein

However, cross-clamping has an important role. This is confirmed in Welch's study (46), that reported a significant reduction in renal artery blood flow immediately after cross-clamp application with resolution at the removal (46).

Three major class of clinical patterns of post-ischemic acute renal failure may be identificated: isolated renal ischemia in protected kidneys as seen during resection of thoracoabdominal aneurysms (47), partial (or intermittent) renal ischemia as seen in patients with poor left ventricular function following cardiac surgery or ruptured abdominal aortic aneurysms (48) and severe form that tends to evolve into oliguric acute renal failure, frequently fatal (49).

The renal protection during aortic operation is essential and it is obtained through different types of methods (Table VIII).

TABLE VIII

Methods of renal protection
Intravenous renal expansion
Isovolemic hemodiluition (Welch)
Renal hypotermia
Intravenous sodium nitroprusside
Mannitol
Furosemide, cathecolamines, beta adrenergic blockers

Intravenous renal expansion, with the possibility to monitor and optimize systemic hemodynamic carefully in the operating room and intensive care unit, has great clinical impact in terms of minimizing ischemic renal injury (47). It is important to avoid episodes of hypovolemia and hypotension, since the autoregulation behavior of the renal circulation is lost in experimental post-ischemic acute renal failure (50). Thus, renal blood flow becomes pressure dependent, transient hypotension produces recurrent renal ischemia that can induce new episode of tubular necrosis.

The acute isovolemic hemodiluition is a process whereby acute blood loss is replaced immediately with cell-free plasmalike fluids, with circulating blood volume being mantained in its normal range (51). The resultant reduction in hematocrit decreases blood viscosity, diminishing the ability of red blood cells to form rouleaux and cell aggregates, and increases blood flow to most organs. A consequence of reduced hematocrit is decreased oxygen content per unit volume but this is compensated by increased flow velocity or shear rate (51), with improved flow in the microcirculation and normal or enhanced oxygen extraction by tissues. Isovolemic hemodiluition also maintains CO during aortic operation (46).

Cold (4°C or 5°C) hyperosmolar crystalloid solution (with or without mannitol, heparin, methyl prednisolone) can be infused into the renal ostia (52,53).

Because oxygen consumption falls by 7% for each Celsius degree that temperature is reduced, the metabolic needs of the tubular cells are reduced by almost 50% at 30° C.

The sodium nitroprusside is used frequently during aortic cross-clamping to control proximal hypertension and unload the left ventricle, but caution is necessary.

The administration of sodium nitroprusside, in experimental dog model, has demonstrated that the proximal hemodynamic pressure is normalized at the expense of reducing distal mean aortic pressure, cortical renal and iuxtamedullary renal blood flow (54). Distal spinal cord flow also fell.

Mannitol is purposed to attenuate the reduction of renal cortical blood flow that occurs before, during and after renal ischemia, while increasing glomerular ultrafiltration pressure (47-49,51). Mannitol also increases the velocity of proximal tubular fluid (filtrate) flow, thereby flushing necrotic tubular debris nephrons, preventing relieving of tubular obstruction (55). Its free radical scavanger effects may also attenuate post-ischemic acute renal failure.

The effect of furosemide (55), cathecolamines (dopamine HCl) (56) and beta adrenergic blockers (57) is mediated by reducing vascular resistance in the renal microvascular system and by increasing the rate of tubular fluid flow, thereby preventing tubular obstruction and increasing ultrafiltration pressure.

Ureteral injury

Ureteral lesions occur infrequently during an aortoiliofemoral reconstruction. The incidence is higher in inflammatory aneurysms and in redo surgery.

Groin lymphatic complications

The first report on groin lymphatic complication after vascular surgical procedures appeared in 1940 and described lymphorrhea after venous ligation (58).

Croft (59) reported the first groin lymphatic complications after an arterial reconstructive procedure in a patient in whom lymphorrhea developed after a femoro-femoral bypass.

Such problem is a well-recognized on most vascular surgery department. In the aorto-ilio-femoral bypass graft the incidence is reported from 1.5 to 3.5 % (60).

The etiology of groin lymphatic complications is not clearly know but presumably is due to disruption of lymphatic channels or lymph nodes during dissection (60).

Risk factors for groin lymphatic complication include poor operative technique, inguinal adenopaty, scar tissue and repeated groin operation, extensive groin dissection, surgery for limb salvage, distal leg infection.

Lymphorrhea (lymphatic fistula) is frequently diagnosed during the peripoperative period, whereas the lymphoceles are recognised later, usually during the first postoperative control.

The superficial cutaneous lymphatics drain the skin and superficial fascia into the inferomedial and inferolateral superficial inguinal nodes. The deep lymphatics of the legs follow along the branches of the femoral artery.

The treatment of lymphorrhea includes implantation of the damaged lymphatic channel into adjacent vein, marsupialization and open packing, application of thrombotic agents, thigh closure of the overlying tissues with pressure dressing, bed rest with leg elevation, observation and ligation of the leaking lymphatic (61,62,63).

Resolution of lymphorrhea has been reported in most patients who undergo conservative nonoperative therapy, but protracted lymphatic drainage may occur especially in the presence of prosthetic grafts (64,65).

Kwaan (66) was the first to document the improved outcome treated with early operation.

Early surgical intervention for lymphorrhea and lymphoceles shortens hospital length of stay and lowers the incidence of graft and wound infection (66,67).

Treatment of lymphoceles is also controversial and includes immobilization, repetitive aspirations, pressure dressing, percutaneous catheter drainage, radiation therapy, sclerosis and operative excision of the cyst wall with ligation of the leak site (68,69).

Because lymphoceles are associated with a closed wound, the risk of infection seems less. Because of limited experience with reexploration, most surgeons prefer conservative treatment except for large, progressive and persistent lymphoceles.

An argument against early surgical treatment for groin lymphatic complication is the fear of exposing the graft and subsequent graft infection.

The technical challenge in the operative repair of such complications is the identification of the open lymphatic channels.

Bowel ischemia

Intestinal infarction after reconstructive surgery of the abdominal aorta was mentioned for the first time by Moore in 1954 (70) and has been recognized as an infrequent but dramatic complication of this type of surgery.

Several retrospective studies have shown its occurrence to be 1% to 2% with an associated mortality of 40% to 100% (71,72,73,74).

These studies report the incidence of the intestinal ischemia to be higher after operation for aortic aneurysm, especially in ruptured cases, than after operation for aortoiliac occlusive disease.

The patent inferior mesenteric artery (IMA) is usually ligated or its orifice sutured during aortic reconstruction. Because of the many collateral routes in this area, this procedure is usually well tolerated unless the colonic circulation is dependent on a contribution solely from the IMA (74).

The connections between the superior mesenteric artery (SMA) and IMA, and which one contributes the most to the collateral circulation when either major artery is occluded, are both sources of controversy (75).

Much of the confusion arises from terminology, as there is general agreement on the precise arterial anatomy. There are typically three paths of communication between the two circulations:

a) the most peripheral artery, which is closest to the wall of the intestine and which supplies vasa recta to it (marginal artery of Drummond);
b) a more centrally placed artery of larger calibre, which usually represents a direct anastomosis of the middle colic and the left colic arteries (central anastomotic artery);
c) an artery that runs in the base of mesentery of the colon and joins the proximal portion of the middle colic artery to the point where the middle colic artery and ascending branch of the left colic artery join (arc or artery of Riolan).

In addition to communications with the SMA, the IMA has collateral pathways with the celiac axis via aberrantly derived middle colic arteries, the internal iliac branches, the femoral artery via the external iliac system and branches of the abdominal aorta.

The severity of ischemia-induced intestinal injury is inversely related to blood flow (76).

Reduction of blood flow to the intestine may be a reflection of generalized poor systemic perfusion, as in shock or with a failing heart, or a result of local morphologic or functional changes of the splanchnic vasculature.

Whatever the cause, the results of intestinal ischemia are the same: a spectrum of injury ranging from completely reversible functional alterations to transmural hemorrhagic necrosis of portions or all of the bowel. Colonic ischemia is the most common form of ischemic injury to the intestinal tract (77).

Today, colonic ischemia is recognized to manifest a spectrum of injury including: 1) reversible colopathy (submucosal or intramural hemorrhage); 2) transient colitis; 3) chronic ulcerating colitis; 4) stricture; 5) gangrene and 6) fulminant universal colitis.

The role of oxygen-derived free radicals has been demonstrated (78,79,80).

These molecules are generally produced in small quantities by enzymes such as xanthine oxidase in the small intestine or aldehyde oxidase in the colon. During intestinal ischemia, enzymes capable of synthesizing free radicals derived from oxygen and their substrata are produced in large quantities. These molecules possess major cellular toxicity and are responsible for tissular lesions (80).

It has been shown experimentally that inhibitors of xanthine oxidase reduce the permeability of the intestinal wall and attenuate the intensity of histological lesions, the risk of necrosis and perforation, and mortality (81).

Overall, factors that contribute to post-operative colonic ischemia include rupture of the aneurysm, hypotension, operative trauma to the colon, hypoxemia, dysrhythmias, long cross-clamp time and improper management of the IMA during aneurysmectomy.

Collateral blood flow to the left side of the colon after occlusion of the IMA comes from SMA through the Arc of Riolan, the central anastomotic artery or the marginal artery of Drummond, and from the internal iliac arteries through the middle and inferior hemorrhoidal arteries.

If these collateral pathways are intact, postoperative colonic ischemia can be minimized. Aortography to determine the patency of the celiac axis, SMA, IMA and internal iliac artery is advisable. The presence of a meandering artery does not allow safe ligation of the IMA, because the blood flow in the meandering artery frequently originates from the IMA and reconstitutes an obstructed SMA (75).

Ligation of the IMA is safe only when angiography confirms that the blood flows in the meandering artery from the SMA to IMA (75).

Reimplantation of the IMA and revascularization of the SMA are required when the SMA is occluded or tightly stenosed and the IMA provides inflow to the meandering artery.

Occlusion of both hypogastric arteries on the preoperative arteriogram indicates that the rectal blood flow depends on collateral flow from the IMA or from the SMA through the meandering artery. In this circumstance, reconstitution of flow to one or both hypogastric arteries is desiderable at the operative time.

During surgery, cross-clamp time should be minimized and hypotension must be avoided. If a meandering artery is identified, it should be carefully preserved.

Because the serosal appearance of the colon is not a reliable indicator of collateral blood flow, several methods have been suggested (75) to determine the need for IMA reimplantation (Table IX).

When IMA reimplantation is deemed necessary, the artery should be excised with a patch of aortic wall and the patch sutured into the side of the aortic prosthesis.

TABLE IX

Methods for determine the need of IMA reimplantation
- stump pressure < 40 mmHg in transected IMA
- mean IMA stump pressure / mean systemic blood pressure < 0.40
- absence of doppler ultrasonographic flow signals at the base of mesentery during temporary IMA inflow occlusion
- tonometric determination of intramural pH of the sigmoid colon

The difficulty in accurately assessing colonic ischemia postoperatively and the significant mortality rate associated with its occurrence mandate postoperative colonoscopy in high-risk patients.

In these cases, colonoscopy is routinely performed within 2 to 3 days of the operation.

All of the ischemic colon must be resected. Primary anastomosis after resection is contraindicated because of the potential danger of a leak and contamination of the aortic prosthesis.

REFERENCES

(1) MARTINEZ BD, HERTZER NR, BEVEN EG. *Influence of distal arterial occlusive disease on prognosis following aortobifemoral bypass.* Surgery 1980; 88: 795-805.

(2) PLECHA FR, BERTIN VJ, PLECHA EJ, AVELLONE JC, FARRELL CJ, HERTZER NR. *The early results of vascular surgery in patients 75 years of age and older: an analysis of 3259 cases.* J Vasc Surg 1985; 2: 769-774.

(3) BUNT TJ. *The role of a defined protocol for cardiac risk assessment in decreasing peripoerative myocardial infarction in vascular surgery.* J Vasc Surg 1992; 15: 626-634.

(4) KILLEWICH LA, ASWARD MA, SANDAGER GP, LILLY MP, FLINN WR. *A randomized, prospective trial of deep venous thrombosis prophylaxis in aortic surgery.* Arch Surg 1997; 132: 499- 504.

(5) FLETCHER JP, BATISTE P. *Incidence of deep vein thrombosis following vascular surgery.* Int Angiol 1997; 16: 65-68.

(6) SCHNEIDER JR, BESSO SR, WALSH DB, ZWOLAK RM, CRONENWETT JL. *Femorofemoral versus aortobifemoral bypass: outcome and hemodynamic results.* J Vasc Surg 1994; 19: 43-45.

(7) FRIEDMAN SG, LAZZARO RS, SPIER LN, MOCCIO C, TORTOLANI AJ. *A prospective randomized comparison of dacron and polytetrafluoroethylene aortic bifurcation grafts.* Surgery 1995; 117: 7-10.

(8) PRENDIVILLE EJ, BURKE PE, COLGAN MP, WEE BL, MOORE DJ, SHANIK DG. *The profunda femoris: a durable outflow vessel in aortofemoral surgery.* J Vasc Surg 1992; 16: 23-29.

(9) TRANS ATLANTIC INTER SOCIETY CONSENSUS MANAGEMENT OF PERIPHERAL ARTERIAL DISESASE. J Vasc Surg 2000; 31: 1-288.

(10) MCCUNE WS. DISCUSSION. In: Adams HD, Geetruyden HH (eds) *"Neurologic complications of aortic surgery".* Ann Surg 1956; 144: 610-616.

(11) ROSENTHAL D. *Spinal cord ischemia after abdominal aortic operation: it is preventable?* J Vasc Surg 1999; 30.

(12) SZILAGY DE, HAGEMAN JH, SMITH RF, ILLIOTT JP. *Spinal cord damage in surgery of the abdominal aorta.* Surgery 1978; 83: 38-56.

(13) SZILAGY DE. *A second look at the etiology of spinal cord damage in surgery of the abdominal aorta.* J Vasc Surg 1993; 17: 1111-1113.

(14) PICONE AL, GREEN RM, RICOTTA JR, MAY AG, DEWEESE JA. *Spinal cord ischemia following operations on the abdominal aorta.* J Vasc Surg 1986; 3: 94-103.

(15) GLOVCIZKI P, CROSS SA, STANSON AW, ET AL. *Ischemic injury to the spinal cord or lumbosacral plexus after aortoiliac reconstruction.* Am J Surg 1991; 162: 131- 136.

(16) DIMAKAKOS P, ARAPOGLOU B, KATSENIS K, ET AL. *Ischemia of the spinal cord following elective operative procedures of the infrarenal aorta.* J Cardiovasc Surg 1996; 37: 243-247.

(17) SUH TH, ALEXANDER L. *Vascular system of the human spinal cord.* Arch Neurol Psychiatry 1939; 41: 659-677.

(18) GILLIAN LA. *The arterial blood supply of the human spinal cord.* J Comp Neurol 1958; 110: 75- 103.

(19) LAZORTHES G, GOUAZE A, ZADEH JO, SANTINI JJ, LAZORTHES Y, BURDIN P. *Arterial vascularization of the spinal cord. Recent studies of the anastomotic substitution pathways.* J Neurosurg 1971; 35: 253-262.

(20) DOMMISE GF. *The blood supply of the spinal cord. A critical vascular zone in spinal surgery.* J Bone Joint Surg.

(21) PLECHA EJ, SEABROOK GR, FREISCHLAG JA. *Neurological complications of reoperative and emergent abdominal aortic reconstruction.* Ann Vasc Surg 1995; 9: 95-101.

(22) DEFRAIGNE JO, OTTO B, SAKALIHASAN N, LIMET R. *Spinal ischemia after surgery for abdominal infrarenal aortic aneurysm: diagnosis with nuclear magnetic resonance.* Acta Chir Belg 1997; 97: 250-256.

(23) ABURAHMA AF, ROBINSON PA, BOLAND JP. *Elective resection of 332 abdominal aortic aneurysms in a southern West Virginia community during a recent five-year period.* Surgery 1991; 109: 244-251.

(24) OLSEN PS, SCHROEDER T, PERKO M ET AL. *Renal failure after operation for abdominal aortic aneurysm.* Ann Vasc Surg 1990; 4: 580-592.

(25) DIEHL JT, CALI RF, HERTZER NR, BEVEN EG. *Complications of abdominal aortic reconstruction.* Ann Surg 1983; 197: 49-56.

(26) LIGUSH J, CRIADO E, BURNHAM SJ, JOHNSON G, KEAGY BA. *Management and outcome of chronic atherosclerotic infrarenal aortic occlusion.* J Vasc Surg 1996; 24: 394-405.

(27) GEWLMAN S, PATEL K, BISHOP SP, FOWLER KL, SMITH LR. *Renal and splanchnic circulation during infrarenal aortic cross-clamping.* Arch Surg 1984; 119: 1394-1399.

(28) POWERS SR, BOBA A, STEIN A. *The mechanism and prevention of distal tubular necrosis following aneurysmectomy.* Surgery 1957; 24: 20-34.

(29) NANSON EM, NOBLE JG. *The effects on the kidneys of cross clamping the abdominal aorta distal to the renal arteries.* Surgery 1959; 46: 156-162.

(30) FOSTER JH, ADKINS RB, CHAMBERLAIN NO, SYMABAS PN, HARRIS AP. *The renal effects of lower abdominal cross clamping.* JAMA 1963; 183: 451-454.

(31) MOWLEM A, MCCLINTOK JT, CAMPBELL GS. *The effect on renal function of occlusion of the aorta inferior to the renal vessels.* Surg Gynecol Obstet 1960; 111: 423-450.

(32) SLATER PV, HAYES MA. *Renal function during infrarenal cross-clamping of the aorta.* Rocky Mt. Med. J. 1969; 66: 50-52.

(33) ABBOTT WM, COOPER JD, AUSTEN WG. *The effect of aortic clamping and declamping on renal blood flow distribution.* J Surg Res 1973; 14: 385- 392.

(34) CRONENWETT JL, LINDENAUER SM. *Distribution of intrarenal blood flow following aortic clamping and declamping.* J Surg Res 1977; 22: 469-482.

(35) OSTRI P, MOURITSEN L, JORGENSEN B, FRIMODT–MOLLER C. *Renal function following aneurysmectomy of the abdominal aorta.* J Cardiovasc Surg 1986; 27: 714- 718.

(36) CALLIGARO KD, SAVARERSE RP, MCCOMBS PR, DELAURENTIS DEA. *Division of the left renal vein during aortic surgery.* Am J Surg 1990; 160: 192-196.

(37) DEARING PD, JAMES EC, SIEGEL MB, SWENSON VM, SLOTNICK HB, SEMEDT MJ. *Further experience with division of the left renal vein.* Surgery 1990; 107: 105-109.

(38) HERMRECK AS. *Prevention and management of surgical complications during repair of abdominal aortic aneurysms.* Surg Clin North Am 1989; 69: 869-894.

(39) PLATELL CF, HALL J, CLARKE G, LAWRENCE – BROWN M. *Intraabdominal pressure and renal fuction after surgery to the abdominal aorta.* Austr N ZJ Surg 1990; 60: 213-216.

(40) GAMULIN Z, FORSTER A, MOREL D, SIMONET F, AYMON E, FAVRE H. *Effects of infrarenal aortic cross-clamping on renal hemodynamics in humans.* Anesthesiology 1984; 61: 394-399.

(41) RATCLIFFE PJ, ENDRE ZH, TANGE JD, LEDINGHAM JGG. *Ischaemic acute renal failure: why does it occur?* Nephron 1989; 52: 1-5.

(42) GAMULIN Z, FORSTER A, SIMONET F, AYMON E, FAVRE H. *Effect of renal sympathetic blockade on renal hemodynamics in patients undergoing major aortic abdominal surgery.* Anesthesiology 1986; 65: 688-692.

(43) GELMAN S, PATEL K, BISHOP SP, FOWLER KL, SMITH LR. *Renal and splanchnic circulation during infrarenal aortic cross clamping.* Arch Surg 1984; 119: 1394-1399.

(44) DUNN MJ, HOOD VL. *Prostaglendins and the kidney.* Am J Physiol 1977; 233: 169-184.

(45) MARGOLIUS HS. *The callicrein –kinin system and the kidney.* Ann Rev Physiol 1984; 46: 309- 326.

(46) WELCH M, KNIGHT DG, CARR HMH, SMYTH JV, WALKER MG. *The preservation of renal function by isovolemic hemodiluition during aortic operations.* J Vasc Surg 1993; 18.

(47) MYERS BD, MILLER DC, MEHIGAN JT ET AL. *Nature of the renal injury following total renal ischemia in man.* J Clin Invest 1984; 73: 329-341.

(48) MEYRS BD, CARRIE BJ, YEE RR HILBERMAN M, MICHAELS AS. *Pathophysiology of hemodinamically mediated acute renal failure in man.* Kidney Int 1980; 18: 495- 504.

(49) MORAN SM, MEYERS BD. *Pathophysiology of protracted acute renal failure in man.* J Clin Invest 1985; 76: 1440-1448.

(50) KELLHER SP, ROBINETTE JB, CONGER JD. *Sympathetic nervous system in the loss of autoregulation in acute renal failure.* Am J Physiol 1984; 246: 379-386.

(51) MESSMER K. *Hemodiluiton.* Surg Clin North Am1975; 55: 659-678.

(52) OCHSNER JL, MILLS NL, GARDNER PA. *A technique for renal preservation during suprarenal abdominal aortic operations.* Surg Gynecol Obstet 1984; 159: 388-390.

(53) CRAWFORD ES, CRAWFORD JL, SAFI HJ, ET AL. *Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients.* J Vasc Surg 1986; 3: 389-404.

(54) GELMAN S, REVES JG, FOWLER K, SAMUELSON PN, LELL WA, SMITH LR. *Regional blood flow during cross-clamping of the thoracic aorta and infusion of nitroprusside.* J Thorac Cardiovasc Surg 1983; 85: 287- 291.

(55) HANLEY MJ, DAVIDSON K. *Prior mannitol and furosemide infusion in a model of ischemic acute renal failure.* Am J Physiol 1981; 241: 556-564.

(56) HILBERMAN M, MASEDA J, STINSON EB., ET AL. *The diuretic properties of dopamine in patients following open heart operations.* Anesthesiology 1984; 61: 489-499.

(57) SOLEZ K, D'AGOSTINO RJ, STAWOWY L, ET AL. *Beneficial effect of propanolol in a histologically appropriate model of postischemic acute renal failure.* Am J Physiol 1977; 88: 163-192.

(58) FISHBACK FC. *Lymph leakage (lymphorrea): a complication of saphenous vein ligation, with suggestions for treatment.* Surgery 1947; 22: 834-836.

(59) CROFT RJ. *Lymphatic fistula: a complication of arterial surgery (letter).* Br Med J 1978; 2: 205.

(60) TYNDALL SH, SHEPARD AD, WILCZEWSKI JM, REDDY DJ, ELLIOTT JP JR, ERNST CB. *Groin lymphatic complications after arterial reconstructions.* J Vasc Surg 1994; 19: 858-864.

(61) WOLFE JHN. *Treatment of lymphedema.* In: Rutherford RB ed. *"Vascular surgery 3rd edition"* Philadelphia, WB Saunders, 1989: 1668-1678.

(62) JOHNSTON KW. *Nonvascular complications of vascular surgery.* In Rutherford RB ed. *"Vascular Surgery 3rd edition"* Philadelphia, WB Saunders, 1989; 536-540.

(63) WEAVER FA, YELLIN AE. *Management of postoperative lymphatic leaks by use of isosulphan blue (letter).* J Vasc Surg 1991; 143: 566-567.

(64) SKUDDER PA, GEARY J. *Lymphatic drainage from the groin following surgery of the femoral artery.* J Cardiovasc Surg 1987; 28: 460-463.

(65) MURPHY JL, COLE W, WHITE PM, BARBER GG ET AL. *Lympahtic fistula after reconstruction: a case control study.* Can J Surg 1991; 34: 76-78.

(66) KWAAN JHM, BERNSTEIN M, CONNOLLY JE. *Management of lymph fistula in the groin after arterial reconstruction.* Arch Surg 1979; 114: 1416-1418.

(67) SCHWARTZ MA, SCHANZER H, SKLADANY M, HAIMOV M, STEIN J. *A comparison of conservative therapy and early selective ligation in the treatment of lymphatic complications following vascular procedures.* Am J Surg 1995; 170: 206-208.

(68) VAN SONNENBERG E, WITTICH GR, CASOLA G, ET AL. *Lymphoceles: imaging charateristcs and percutaneous management.* Radiology 1986; 161: 593-596.

(69) POPE AJ, ORMISTON MC, BOGOD DG. *Sclerotheraphy in the treatment of recurrent lymphocele.* Postgrad Med J 1982; 58: 573-574.

(70) MOORE SW. *Resection of the abdominal aorta with defect replaced by homologous graft.* Surg Gynecol Obstet 1954; 99: 745-750.

(71) BERQVIST D, BOWALD S, ERIKSSON I, LANNERSTAD O, TAKOLANDER R. *Small bowel necrosis after aortoiliac reconstruction.* Br J Surg 1986; 73: 28-32.

(72) BREWSTER DC, FRANKLIN DP, CAMBRIA RP, DARLING RC, MONCURE AC, LAMURAGLIA GM, STONE WN, ABBOT WM. *Intestinal ischemia complicating abdominal aortic surgery: clinical review.* Surgery 1991; 109: 447-451.

(73) JÄRVINEN O, LAURIKKA J, SISTO T, TARKKA MR. *Intestinal ischemia following surgery for aortoiliac disease: a review of 502 consecutive aortic reconstructions.* Vasa 1996; 25: 148-153.

(74) JÄRVINEN O, LAURIKKA J, SALENIUS JP, LEPÄNTALO M. *Mesenteric infarction after aortoiliac surgery on the basis of 1752 operations from the National Vascular Registry.* World J Surg 1999; 23: 243-247.

(75) KORNBLITH F, BOLEY SJ. *Anatomy of the splanchnic circulation.* Surg Clin North Am 1992; 72: 1-30.

(76) PATEL A, KALEYA RN, SAMMARTANO RJ. *Pathophysiology of mesenteric ischemia.* Surg Clin North Am 1992; 72: 31-41.

(77) BRANDT LJ, BOLEY SJ. *Colonic ischemia.* Surg Clin North Am 1992; 72: 203-229.

(78) DASLING MC, GROSFELD JL, SHIFFLER MA ET AL. *Superoxide dismutase: a cellular protective enzyme in bowel ischemia.* J Surg Res 1983; 34: 589-596.

(79) PARKS DA, BULKLEY GB, GRANGER DN. *Role of oxygen derived free radicals in digestive tract disease.* Surgery 1983; 94: 415-422.

(80) FARKAS JC, CALVO-VERJAT N, LAURIAN C, MARZELLE J, FICHELLE JM, GIGOU F, BLÉRIOT JP, DAZZA F, CARLET J, CORMIER JM. *Acute colorectal ischemia after aortic surgery: pathophysiology and prognostic criteria.* Ann Vasc Surg 1992; 6: 111-118.

(81) GRANGER DN, MCCORD JM, PARKS DA ET AL. *Xanthine oxidase inhibitors attenuate ischemia-induced vascular permeability changes in cat intestine.* Gastroenterology 1986; 90: 80-84.