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VASCULOGENIC IMPOTENCE

Arterial and Venous Surgery

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Impotence is defined as the inability to achieve or maintain an erection of sufficient rigidity to allow vaginal penetration. It is estimated that some form of erectile dysfunction will develop in 52% of men between the ages of 40 and 70 years, affecting 25 million men in the United States alone.[41] The incidence of erectile dysfunction over the past decades has remained relatively constant. Improvement in the treatment of hypertension and cardiovascular disease has been offset by greater longevity and demographic trends toward a larger geriatric population. Advances in the treatment of erectile function, notably the introduction of sildenafil in 1997, have increased public awareness of the disorder and the number of men seeking treatment.

Improved understanding of the physiology of normal erection and of the pathophysiology of erectile dysfunction has resulted in the current variety of therapeutic modalities. These treatments range in their invasiveness, efficacy, and target population, but all have limitations. Sildenafil is most effective for men with mild-to-moderate erectile dysfunction. Intracavernous vasoactive agents are of limited use in patients with veno-occlusive disease. For patients in whom none of these therapies have proved to be efficacious, a variety of arterial revascularization or venous ligation procedures have been developed in the hope of restoring spontaneous erectile function.

Numerous series in the literature over the past 2 decades detail refinements in penile arterial and venous surgical techniques, along with patient selection, diagnostic testing, and follow-up for these procedures. When taken at face value, many of these reports suggest impressive results; however, considering the limitations of study designs, there is a developing consensus that vascular surgical procedures may produce disappointing results for patients with vasculogenic erectile dysfunction. This realization has led to the call for surgery to be performed for specific indications, with full disclosure to the patient of potential pitfalls, at institutions where objective follow-up information can be gathered. This article reviews surgical procedures for arterial revascularization and venous ligation in the treatment of erectile dysfunction and the limitations of current diagnostic and follow-up criteria. A summary is provided of the limited role these procedures have in the current therapeutic spectrum for erectile dysfunction.

PENILE VASCULAR ANATOMY

The arterial blood supply to the penis normally originates from the paired internal pudendal arteries, which are branches of the ipsilateral inferior epigastric artery. The common penile artery arises from the internal

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pudendal artery and then branches to form the bulbourethral, dorsal, and cavernous arteries. The bulbourethral branch supplies the bulb, corpus spongiosum, and the glans to some extent. The corpora cavernosa receive their blood from the paired cavernous arteries during tumescence. The dorsal artery is responsible for engorgement of the glans penis during erection.

The veins of the penis drain into three systems.[6] The superficial dorsal veins are small venous channels in the subcutaneous layer of the skin and subcutaneous tissue of the penis. The superficial dorsal veins usually empty into the saphenous vein. The intermediate system includes the deep dorsal vein and circumflex veins and is formed from the sinusoids of the corpora, which initially drain into small venules under the tunica albuginea. These venules then form subtunical venular plexuses that penetrate the tunica albuginea as the emissary veins. The emissary veins course obliquely through the tunica albuginea to join the deep dorsal vein. The deep system is composed of the cavernous and crural veins. The proximal third of the penis is drained by the cavernous veins, which arise from the penile hilum. In addition, crural veins arise on the dorsomedial surface of each corpus cavernosum and drain into the internal pudendal vein.

PHYSIOLOGY OF PENILE ERECTION

The smooth muscle tissue of the cavernous sinusoids and the arterioles controls tumescence and detumescence of the penis.[14] The sympathetic and parasympathetic nervous systems coordinate to regulate the tonicity of the cavernous smooth muscle. Cholinergic transmission supports erection, but a noncholinergic nonadrenergic (NANC) neurotransmitter seems to have a central role in erection. Nitric oxide has been demonstrated to be the NANC neurotransmitter responsible for this mediated mechanism of erection.[3, 51] Erection occurs when the smooth muscles of the sinusoids and the arterioles relax in response to nitric oxide, resulting in a large increase in arterial flow and rapid filling of the sinusoidal spaces. With this crucial distension of the sinusoids, the subtunical venules between the sinusoidal walls and the tunica albuginea are compressed, resulting in reduced venous outflow and rigid erection. Adrenergic impulses control detumescence. Norepinephrine released from postganglionic sympathetic nerve endings causes contraction of the sinusoidal smooth muscle, opening the subtunical venular plexus. Detumescence is the result of prompt drainage of intracavernous blood.

PATHOGENESIS OF VASCULOGENIC ERECTILE DYSFUNCTION

Arterial injury may lead to decreased or insufficient inflow to achieve normal erections. Although patients typically describe difficulty attaining erection, they retain the capacity to maintain the erection. Traumatic arterial injury can cause the acute onset of impotence. Blunt pelvic trauma, particularly with disruption of the membranoprostatic urethra, can result in arterial injury and impotence. With the exception of injuries to the perineum, penetrating injury to the penile arterial system is rare. More commonly, the acute vascular injury is the result of pelvic surgery.

Arterial insufficiency is most commonly the result of progressive systemic arteriosclerosis. The onset of erectile dysfunction is gradual in this setting, and arterial insufficiency and veno-occlusive dysfunction may occur. Risk factors for the development of atherosclerosis in the cavernous arteries are the same as for peripheral vascular disease in general and include hypertension, hypercholesterolemia, tobacco abuse, and diabetes mellitus.

Erectile failure owing to venous insufficiency in the presence of adequate arterial inflow can be termed *veno-occlusive erectile dysfunction.* The importance of direct injury at the cellular level to the sinusoidal endothelium and smooth musculature in the pathogenesis of veno-occlusive erectile dysfunction has become apparent. Such damage limits the ability of the smooth muscle to relax within the corpora, resulting in reduced sinusoidal dilation. Any factor leading to an alteration of the fibroelastic component of the trabeculae can result in incomplete venous occlusion with subsequent venous leakage and erectile failure.[47] Ischemic injury owing to priapism with subsequent fibrosis of the erectile tissue long has been recognized as a cause of impotence.

Alternately, venous leakage may be the result of the presence or development of excessively large venous channels through the corpora cavernosa. Usually, these large veins are congenital in origin. Degeneration of the tu-

nica albuginea results in inadequate compression of the subtunical and emissary veins and may occur with aging or Peyronie's disease. Venous shunts between the cavernosa and the spongiosum, congenital or acquired, can result in impotence. Acquired shunts may be the result of surgical correction of priapism or caused by blunt trauma to the penis in the erect state.[49]

DIAGNOSTIC EVALUATION

Following a careful and complete history, physical examination, and basic laboratory studies, several examinations are useful in further defining the etiology of erectile dysfunction in terms of arterial insufficiency, the presence of venous leakage, and the anatomy of the functional deficits.

Combined Intracavernous Injection and Stimulation Testing

The discovery of pharmacologically inducible erection was a major advance in the diagnosis of erectile dysfunction. The combined injection and stimulation test (CIS test)[37] is a simple and effective procedure that should be the first diagnostic evaluation performed. The patient's response to intracavernous injection of vasodilators is enhanced by tactile or audiovisual sexual stimulation. On this basis, the CIS test uses tactile input to improve the quality of erection obtained after intracavernous injection. Additionally, self-stimulation can give some indication of the status of the patient's reflexogenic erectile response and provide data concerning potential psychologic inhibition during testing.

The CIS test is performed by placing the patient in an isolated examining room in the supine position. A total of 10 μg of prostaglandin E_1 or 30 mg of papaverine with 1.0 mg of phentolamine is injected into one of the corpora cavernosa with a 28-gauge needle, and the patient is observed for 15 minutes. A sustained erection lasting for more than 15 minutes with good rigidity signifies that arterial or venous insufficiency is unlikely, and no further vascular work-up is advised. If the patient only has a partial erectile response after 15 minutes, the addition of manual genital stimulation may result in sustained rigid erection. This event signifies that reflexogenic erection is relatively intact, and that the patient is not inhibited.

If full erection is obtained initially or after the addition of genital self-stimulation, venous integrity then is evaluated indirectly. The erection is assessed again by inspection and palpation after a period of rest and nonstimulation. Patients who sustain erection during this period have an intact veno-occlusive mechanism. Patients who rapidly detumesce after obtaining rigid erection with intracavernous injection have a degree of venous leakage as the result of primary venous dysfunction or secondary to α-mediated smooth muscle contraction.

Duplex Ultrasonography Scanning

Duplex ultrasonography testing combines real-time ultrasonography to image the internal penile structures[33] with pulsed Doppler scanning to examine the flow characteristics of the cavernous arterial system. The echogenicity of the penile tissues is observed, and the diameters of the cavernous arteries are measured before and after intracavernous injection of a vasodilator as a functional test of the cavernous arterial function. The thickness of the cavernous artery wall can be estimated and the artery observed for pulsation. Healthy arteries will be noted to have strong thin walls with strong pulsations, whereas arteries with diffuse artherosclerosis will be thick walled, irregular, and have minimal pulsations. The peak flow velocity of blood within the cavernous arteries is measured using Doppler ultrasonography and compared with normal controls. Because arterial diameter and flow rate change during different phases of erection, the parameters are measured 5 minutes after the injection and, if necessary, after self-stimulation. Sharp systolic waveforms with minimal diastolic flow are characteristic of normal penile hemodynamics. Other waveforms can suggest arterial disease or failure of the veno-occlusive mechanism.

Duplex ultrasonography scanning can be combined with the CIS test in the office to arrive at a simple minimally invasive differential diagnosis of erectile dysfunction. When performed properly, duplex ultrasonography is equal to, and may even be superior to, pharmacologic arteriography[45] for the diagnosis of arteriogenic impotence. The disadvantages of this procedure are the high cost of

the equipment necessary to perform the test and the operator-dependency of results, a problem shared by all ultrasonographic diagnostic procedures.

Cavernous Arterial Occlusion Pressure

After intracavernous injection of papaverine and phentolamine, normal saline solution is infused to raise the intracavernous pressure to more than 150 mm Hg. When the infusion is stopped, the pressure at which the arterial pulse becomes audible is the cavernous artery occlusion pressure. A direct measurement of the cavernous arterial occlusion pressure is determined, and the functional integrity of the cavernous arteries is ascertained. The cavernous artery occlusion pressure can be used to calculate a gradient between the systemic arterial pressure as reflected in the brachial artery and penile arterial inflow.[46] A difference of more than 36 mm Hg is considered to represent abnormal cavernous artery function. Cavernosometry and cavernosography then are performed through the same infusion ports to evaluate venous integrity.

Pharmacologic Arteriography

Although pharmacologic arteriography has many potential pitfalls, it is considered the gold standard in the evaluation of arterial anatomy. Because of low arterial flow through the cavernous arteries in the flaccid penis, the cavernous arteries may be visualized poorly and mistakenly judged to be diseased. This misdiagnosis also may occur with excessive α-stimulation from anxiety and apprehension during penile arteriography and from pain. With the introduction of intracavernous vasodilator injection, excellent visualization of the cavernous arteries with local anesthesia can be obtained. Owing to the potential for complications and its high cost, the test only should be performed in preparation for penile revascularization surgery. For patients in whom a definitive diagnosis is desirable but, for other reasons, are not candidates for arterial reconstructive surgery, duplex ultrasonography scanning before and after intracavernous injection of a vasodilator may provide a better assessment of penile arterial function than arteriography.[45] Duplex ultrasonography is far less expensive and virtually risk free.

Diagnosis of Venous Leakage—Cavernosometry and Cavernosography

After the diagnosis of presumed venous leakage is made by the CIS test, pharmacologic cavernosometry may be performed to quantify the degree of veno-occlusive dysfunction. Because of its invasive nature, cavernosometry should be restricted to patients in whom surgery is contemplated. If cavernosometry is abnormal, cavernosography is performed to identify the site of venous leakage.[34] Intracavernous injection of vasoactive agents is used to activate the erectile mechanism. A major advantage of this injection is that the vasodilator eliminates psychologic inhibition; however, an occasional false-positive result still will occur owing to extreme apprehension, even after the maximal amount of papaverine and phentolamine has been injected, and caution is suggested in interpreting the results.

PENILE REVASCULARIZATION

The allure of penile revascularization surgery is the potential to increase arterial blood flow to the penis sufficient to restore spontaneous physiologic erections. Although this result is the ultimate goal of all men seeking treatment for erectile dysfunction, revascularization surgery remains controversial, with imprecise indications and disputed success rates. Initial enthusiasm in the 1970s resulted in a wide variety of patients undergoing revascularization procedures. Long-term follow-up of these patients revealed less remarkable results. The refinement of diagnostic and patient selection criteria over the past 17 years has led to a narrowed set of indications for penile revascularization surgery. Surgery has proved efficacy in young men with arterial insufficiency secondary to pelvic trauma,[17] who are a small subset of men for whom erectile dysfunction is an issue. Some surgeons continue to advocate revascularization as an option for patients who have attempted to use less invasive methods of therapy, such as oral medication, intracavernous injection, or vacuum constriction devices, but have failed these methods. Some surgeons believe that if a patient finds these interventions to be inadequate or unacceptable, revascularization surgery is a reasonable option and an alternative to prosthesis implantation.

Surgical Procedures

The first report of penile revascularization was published by Michal and co-workers[42] in 1972. Their technique used a direct anastomosis between the inferior epigastric artery and the corpus cavernosum that allowed flow rates of more than 100 mL/minute and often produced intraoperative erections. The limitations of the procedure soon became apparent as priapism or anastomotic thrombosis occurred in most patients. Furthermore, animal studies showed that cavernosal tissues underwent fibrosis and degeneration when exposed to direct arterial blood pressure.[7, 48] Modifications to limit the epigastric flow by banding the donor artery kept the anastomosis open for a period of time, but the rate of stenosis and eventual thrombosis of the bypass still approached 100% by 1 year.

The dorsal penile artery and the cavernosal arteries arise from the pundendal artery and share extensive collaterals. This anatomic relationship formed the basis for the Michal II procedure.[43] The inferior epigastric artery was anastomosed end-to-side with the dorsal penile artery. Michal reported a 56% success rate after the procedure. In 1988 Goldstein[18] reported success in approximately 80% of patients using a similar technique. The optimal candidates for the technique were young men with localized obstruction of the internal pudendal artery or common penile artery secondary to trauma.

Virag and co-workers[63] introduced an indirect approach to revascularization using the epigastric artery anastomosed end-to-side to the deep dorsal vein. His group reported normal erectile function in 49% of patients and improved function in an additional 20%. Over the following years, Virag performed several variations of the technique, including the creation of window between the dorsal vein and corporal body. Furlow and Fisher[16] have reported on 21 cases of epigastric artery to dorsal vein anastomosis with ligation of the proximal and distal deep dorsal vein and all circumflex veins. They reported a success rate of 62%.

In 1984 Hauri[23] introduced his three-vessel anastomotic technique using a side-to-side anastomosis between the dorsal artery and vein covered by a spatulated epigastric artery.[23] This approach remains in use today with a success rate as high as 80%.[24]

In 1987, Crespo and colleagues[11] reported a reverse autologous saphenous vein graft from the femoral artery to the cavernous artery for direct revascularization. The success rate at 6 months was 78%. Thrombosis at the anastomotic site remained a problem with this technique.

In 1987 Carmignani and co-workers[9] introduced a technique in which the inferior epigastric artery was anastomosed end-to-end to the proximal end of a transected dorsal artery. The distal stump then was anastomosed to the contralateral dorsal artery in an end-to-side manner. Various other modifications of the Hauri or Virag procedures have been reported, with success rates greater than 60%.[2, 19, 40]

Evaluations of the Furlow and Virag V-procedures were reported by Lizza and Zorgniotti in 1988.[31] They found successful results in three of four (75%) patients undergoing the Furlow procedure compared with 78% of nine patients undergoing the Virag V. A long-term follow-up of 42 patients was reported in 1994. A total of 24 patients underwent the Furlow procedure, 12 the Virag V, 2 the Crespo, 1 the modified Michal I, and 2 Michal II procedures. The outcome was considered successful in 19 of 36 patients (53%) at 1 year versus 4 of 10 (40%) at 5 years.

Series published in the past 5 to 10 years have used a variety of the previously described procedures (Table 1).[13, 24, 26, 27, 40, 67] Long-term successful results in these studies, with admittedly varied patient populations, indications, and techniques, have ranged from 25%[2] to 80%.[24]

Laparoscopy recently has been introduced as a method of reducing the invasiveness of revascularization procedures. The pararectal incision used to harvest the inferior epigastric artery has been implicated in postoperative pain, bleeding, and hernia formation.[21] Seventeen patients underwent laparascopic harvesting of artery using an intraperitoneal[61] or extraperitoneal[21] approach with an overall reduction in surgical morbidity.

Complications

The most frequently reported complication of revascularization surgery is hyperemia of the glans, occurring in 7%[67] to 13%[40] of patients. Prompt attention is needed in these patients to avoid glanular tissue loss. Distal vein ligation has been suggested at the time of surgery to avoid this complication.[13] Other commonly reported complications include

Table 1. PENILE REVASCULARIZATION SERIES SINCE 1995

Study	Date	Number of Patients	Age (years)	Procedure	Follow-up	Type	Outcome
Zumbe et al[67]	1999	124	22–71	DA/IEA/DDV anastomosis	Mean, 54 months	US, questionnaire	59.7% Spontaneous erections
Manning et al[40]	1998	62	19–70 (mean, 48)	Virag, Hauri, modified Hauri	Mean, 29 and 41 months	Questionnaire, Doppler US, angiography, ICI	34% Spontaneous erections, 20% ICI response
Sarramon et al[53]	1997	114	20–72 (mean, 48)	DDV arterialization (70 patients) Michal II (44 patients)	Mean, 17 months	Not specified	47.8% "Success" 14.6% "Improved"
Anafarta et al[2]	1997	24	NA	IEA to DDV with proximal vein ligation	Mean, 24 months	Not specified	25% "Restored erectile potency"
Trombetta et al[61]	1997	2	45	IEA to DA, IEA to DDV	3 Months	Patient report, Doppler US, arteriogram	100% Spontaneous erections
Hatzinger et al[21]	1997	15	30–60 (mean, 49)	DA/IEA/DDV anastomoses	12 Months, median	Not specified	53% Spontaneous erections 27% ICI response
Jarow and DeFranzo[26]	1996	11	19–53 (mean, 41.9)	DA bypass (9), DDV arterialization (2)	12 to 84 Months (mean, 50)	Doppler US, patient report	64% Spontaneous erections 27% ICI response
DePalma et al[12]	1995	67	18–79	Aortoiliac reconstruction, DA bypass, DDV arterialization, venous ligations	34 to 48 Months	Doppler US, arteriography, DICC, ICI, questionnaires	Aortoiliac: 58% spontaneous erections DA bypass: 27% spontaneous erections DDV arterialization: 33% spontaneous erections Vein ligation: 27% spontaneous erections
Kaufman et al[27]	1995	16	<50 (mean, 35 ± 9)	DA to DDV, 4 also with venous ligation, 3 also with IEA to DA	At least 6 Months	Patient report	40% Excellent or normal erections 53% Improved erections 7% Unchanged
Knoll[29]	1995	26	23–51 (mean, 41)	DDV arterialization	18–52 Months (mean, 37)	Questionnaire, Doppler US, ICI, biothesiometry, cavernosography, cavernosometry	31% Spontaneous erections 35% ICI response

DA = dorsal artery; IEA = inferior epigastric artery; DDV = deep dorsal vein; US = ultrasonography; ICI = intracavernous injection; NA = not available.

hematomas, thromboses of the vascular anastomosis, and infection.[68]

Discussion

Controversy persists regarding the clinical utility of penile revascularization. Recently published series claim long-term efficacy rates of more than 60%. The confusion is exacerbated by the widely varied patient populations, diagnostic modalities, surgical techniques, definitions of success, and methodology of determining successful outcomes in these studies. Sharlip[57] summarized these problems in his 1990 address to the International Society for Impotence Research by enumerating five key weaknesses in the design and execution of the studies: (1) imprecise patient selection, (2) the lack of objective standardized follow-up, (3) the lack of rational physiologic surgical concepts, (4) sham or placebo effects, and (5) potential surgeon prejudice.

Although almost 10 years have passed since Sharlip's critique, these key issues continue to fuel the debate. There is no imaging test for reasonable preoperative patient selection or objective postoperative assessment of success.[59] Pudendal angiography is considered the gold standard for morphologic assessment of penile arterial supply but does not offer a functional analysis after surgery. Additionally, there has been no correlation between anastomotic patency as measured by angiography and subjective patient satisfaction after the surgical procedure.[30] In his comprehensive review of the results of revascularization, Sohn[59] concluded that no correlation exists between any objective hemodynamic tests and the subjective outcomes reported by patients.

Although revascularization may increase arterial flow into the corporal bodies, this change alone does not guarantee the restoration of erectile function. Impaired neuronal and endothelium-dependent corporal smooth muscle relaxation, corporal fibrosis owing to hypertensive effects, or diabetes-induced connective tissue damage leading to veno-occlusive dysfunction are among the reasons why surgery fails despite a patent postoperative anastomosis. End-organ damage at the cellular level may be the overriding factor in an unknown percentage of men.

The nonstandardized subjective scores reported in most recent studies along with the differences in patient selection and surgical technique result in the unfortunate fact that the series meaningfully cannot be compared statistically. The debate over these issues has improved the objectivity of results reporting in some instances, but problems persist. The Consensus Development Conference on Impotence held by the National Institutes of Health in 1992 declared that procedures for penile revascularization should "have a very limited role and probably should be restricted to the clinical investigation setting in medical centers with experienced personnel."

Undoubtedly, penile revascularization surgery has benefited numerous men with vasculogenic erectile dysfunction. In the future, better diagnostic testing and more objective study analyses may help to resolve some of the current controversies regarding the role of these procedures. Until then, penile revascularization seems best suited to young men with no other risk factors who are unlikely to have underlying cavernosal dysfunction and who undergo revascularization to address arterial disruption secondary to trauma.[19]

PENILE VENO-OCCLUSIVE DISEASE

The first attempts to treat veno-occlusive erectile dysfunction through a surgical approach were performed in 1902 by Wooten and in 1908 by Lydston who ligated the deep dorsal vein of the penis with temporary success in return of erectile dysfunction. Penile vein ligation was not commonly performed until 1985.[65] The initial approach of single-vessel ligation of the dorsal vein was expanded owing to poor results. A range of ligation procedures varied in their aggressiveness have emerged and range from dorsal and accessory vein ligation to complete ligation and excision of the dorsal, cavernous, and crural veins as described by Lue.[36] Recently, dorsal vein embolization has been used alone[58] or in combination with surgery[15] to decrease the invasiveness of therapy.

Lue[38] recently has described a technique of proximal crural ligation for the treatment of crural venous leakage.[38] This technique is effective in younger men with congenital or trauma-induced deficiencies but is not recommended for patients with smooth muscle or tunical atrophy.

Deep dorsal vein arterialization has been performed under the rationale of increasing venous outflow pressures and compensating

Table 2. PENILE VEIN LIGATION SERIES SINCE 1995

Study	Date	Number of Patients	Age (years)	Procedure	Follow-up	Type	Outcome
Lue[38]	1999	9	<40	Crural vein ligation, repair of spongiosal leak, and/or DDV ligation	29 Months (range, 4–47)	Doppler US, cavernosography, cavernosometry, patient report, rigidity	77% Success
Sasso et al[54]	1999	23	20–50 (mean, 41)	Superficial, deep dorsal, circumflex, and emissary vein ligation	12 Months, long-term	Not specified	74% Spontaneous erection at 12 Months 55% Long-term
Popken et al[50]	1999	122	19–78 (mean, 49)	Superficial, deep, and circumflex vein ligation	70 Months	Questionnaire	14% Spontaneous erections 19% ICI response
Al Assal et al[1]	1998	325	18–62 (mean, 45)	Ligation of DDV, abnormal veins, cavernosal veins, cavernosal-spongiosum shunts	1–13 Years	Not specified	Age <40 years: 76% "cured" Age >40 years; 58% "cured"
Basar et al[4]	1998	26	NA	Venous ligation	25 ± 2.1 Months	Not specified	6 Months 15% complete erection, 23% partial erection
Schulttheiss et al[56]	1997	147	NA	DDV ligation	NA	Patient report, questionnaire	11.2% Spontaneous erection 19% ICI response
Berardinucci et al[5]	1996	100	23–77 (mean, 58)	DDV excision	Mean, 45 Months	Patient report	31% Spontaneous erections
Sasso et al[55]	1996	44	20–56 (mean, 42)	DDV ligation, corporopexy	2–96 Months	Patient report	44.2% Spontaneous erections, 9.3% ICI response
Hassan et al[20]	1995	32	33–77 (mean, 45)	Cavernosal and crural vein ligations	>22 Months	Questionnaire	31% Spontaneous erections, 28% ICI response
Kim and McVary[28]	1995	15	29–68 (mean, 50)	DDV/cavernous ± crural vein excision	19–45 Months (mean, 29)	Questionnaire	60% Spontaneous erections
Vale et al[62]	1995	27	26–63 (mean, 56)	DDV excision	3 and 12 Months	Questionnaire	45% Spontaneous erections 18% ICI response

DDV = deep dorsal vein; US = ultrasonography; ICI = intracavernous injection; NA = not available.

for veno-occlusive dysfunction.[53, 64] Surgeons also have reported success using this procedure in treating mixed arterial and veno-occlusive disease.[32, 53, 64] Arterial bypass procedures alone are considered inadequate in treating mixed vasculogenic impotence.[10]

Success rates for surgical procedures for veno-occlusive disease generally have been poor (Table 2). Success rates within the first year range from 23% to 80%[65] but consistently decrease on longer follow-up (14% to 77% after 1 year).[38, 56]

Complications

Proximal penile numbness has been reported after ligation procedures, which is usually transient and probably related to division of the suspensory ligament.[36] Techniques that spare the suspensory ligament may avoid this problem.[5] Penile curvatures, painful erections, wound infections, and areas of skin necrosis have been reported.

Discussion

As true for arterial revascularization procedures, initial enthusiasm for surgical treatment of veno-occlusive erectile dysfunction was tempered by disappointing long-term results. Reasons advanced for the inadequate long-term correction of disease included inadequate surgical ligation of veins, the development of collateral bypasses, especially spongiosal leaks, corporal myopathy, and neurotransmitter deficiencies.[25] With better understanding of the physiology of erection on the cellular and biochemical level, this last reason is supported increasingly as the most likely cause of surgical failures.

Disease states, such as diabetes, hyperlipidemia, and hypertension, along with substances such as nicotine, can cause damage to corporal smooth muscle cells. During erection, the relaxation of the smooth muscle cells mediated by nitric oxide allows corporal sinusoids to fill with blood. The expanding erectile tissues compress the subtunical plexus so that blood becomes trapped within the corporal bodies. Damage at the cellular level produces a deficit in erectile physiology that is not compensated for directly by venous ligation. Surgery may address a symptom of the disease but not the disease process. This fact may account for the relatively poor results seen over the long-term in patients with smooth muscle dysfunction.

The clear indications for the surgical treatment of veno-occlusive erectile dysfunction remain patients with excessive venous channels through the corpora, or with venous shunts from the corpora to the glans or spongiosum. In these patients, if there is no coexisting arterial disease, surgery has the potential to restore the normal physiologic state. In all other cases, surgery is an attempt to compensate for an underlying defect that may prove to be insurmountable by such methods in the long run.

FUTURE DIRECTIONS

Future refinements in study methodology and patient selection are likely to delineate patients who may benefit from surgery from those who will not. Advances in erectile dysfunction therapy are likely to follow recent advances in the understanding of erectile dysfunction pathophysiology at the cellular level. Pharmacologic treatments to alter the balance of forces that regulate penile tumescence/detumescence may become more effective as the involved neurochemical mediators are further defined.

Pharmacologically induced angiogenesis is another area that shows great potential. Vascular endothelial growth factor (VEGF), an angiogenic factor, has been shown to induce new blood vessel formation in animal models, including the rabbit hind limb model of ischemia[60] and the porcine ischemic heart model,[22] and currently is undergoing human trials for coronary artery disease. Initial studies show that VEGF restores corporeal smooth muscle relaxation in a hypercholesterolemic New Zealand white rabbit model[8] and an ischemic rat model[39] of erectile dysfunction.

SUMMARY

Arterial revascularization and venous ligation procedures have been introduced within the past 2 decades. Each procedure has in common with the other the fact that initial applications of the operations were widespread among the population of men with vasculogenic erectile dysfunction. In each case, disappointing long-term results led to more limited use of surgery targeting specific groups that clearly would benefit from the

procedures. The wider application of these procedures in vasculogenic erectile dysfunction is not supported by the available results. The Clinical Guidelines Panel of the American Urological Association supported this view in 1996 after a meta-analysis of literature reports and declared that venous and arterial surgery was not justified in routine use, especially in patients with arteriosclerosis.[44] Further studies are likely to refine patient selection but are unlikely to expand the therapeutic use of these procedures.

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