INTERNATIONAL JOURNAL OF UROLOGY
International Journal of Urology (2019) doi: 10.1111/iju.14157

Review Article

Surgical niche for the treatment of erectile dysfunction

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Abbreviations & Acronyms
AE = adverse effect
AI = arterial insufficiency
CES-D = Center for Epidemiologic Studies Depression Scale
CVOD = corporoveno-occlusive dysfunction
DDV = deep dorsal vein
DICC = dynamic infusion cavernosometry and cavernosography
DM = diabetes mellitus
DPA = dorsal penile artery
DV = dorsal vein
ED = erectile dysfunction
EFD = erectile function domain
F/U = follow-up
FFV5 = Furlow-Fisher modification of the Virag V procedure
HTN = hypertension
ICI = intracavernosal injection
IEA = inferior epigastric artery
IIEF = International Index of Erectile Function
LOE = level of evidence
MF = maintenance flow
NA = not available
NR = not reported
NSQ = non-standardized questionnaire
PAV = para-arterial vein
PDDU = penile duplex Doppler ultrasonography
PDE5i = phosphodiesterase type 5 inhibitor
PP = penile prosthesis
SEwP = spontaneous erection without pharmacotherapy
VOD = veno-occlusive dysfunction

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Received 18 August 2019; accepted 4 November 2019.

Abstract: Penile erection implicates arterial inflow, sinusoidal relaxation and corporoveno-occlusive function. By far the most widely recognized vascular etiologies responsible for organic erectile dysfunction can be divided into arterial insufficiency, corporoveno-occlusive dysfunction or mixed type, with corporoveno-occlusive dysfunction representing the most common finding. In arteriogenic erectile dysfunction, corpora cavernosa show lower oxygen tension, leading to a diminished volume of cavernosal smooth muscle and consequential corporoveno-occlusive dysfunction. Current studies support the contention that corporoveno-occlusive dysfunction is an effect rather than the cause of erectile dysfunction. Surgical interventions have consisted primarily of penile revascularization surgery for arterial insufficiency and penile venous surgery for corporoveno-occlusive dysfunction, whatever the mechanism. However, the surgical effectiveness remained debatable and unproven, mostly owing to the lack of consistent hemodynamic assessment, standardized select patient and validated outcome measures, as well as various surgical procedures. Penile vascular surgery has been disclaimed to be the treatment of choice based on the currently available guidelines. However, reports on penile revascularization surgery support its utility in treating arterial insufficiency in otherwise healthy patients aged <55 years with erectile dysfunction of late attributable to arterial occlusive disease. Furthermore, it is noteworthy that penile venous surgery might be beneficial for selected patients with corporoveno-occlusive dysfunction, especially with a better understanding of the innovated venous anatomy of the penis. Penile vascular surgery might remain a viable alternative for the treatment of erectile dysfunction, and could have found its niche in the possibility of obtaining spontaneous, unaided and natural erection.

Key words: arterial insufficiency, erectile dysfunction, penile revascularization, veno-occlusive dysfunction, venous stripping.

Introduction

Management of ED has remained a difficult issue, even with great strides in basic science and clinical medicine during the past several years. The pathophysiology has been elucidated with characterized etiologies by anatomical, vascular, neurological, hormonal and systemic causes, with a psychogenic component to some degree.[1] Vascular risk factors, encompassing diabetes, hypertension, dyslipidemia, cardiovascular disease and metabolic syndrome, have been shown to be predisposed to ED.[2,3]

By far the most widely recognized vascular etiologies responsible for organic ED can be divided into AI, CVOD or mixed type, with CVOD representing the most common finding.[4,5] It is of paramount importance that penile erection implicates arterial inflow, sinusoidal relaxation and corporoveno-occlusive function. In arteriogenic ED, corpora cavernosa show lower oxygen tension,[6] leading to a diminished volume of cavernosal smooth muscle and consequential CVOD.[7] Nevertheless, the inability to trap blood inside corpora cavernosa is also a salient etiology responsible for ED.[8] CVOD, also acknowledged as venogenic ED or venous leakage, accounts for the most appreciable vascular etiology,[9,10] which was recognized in 85% of men appraised for ED, irrespective of age.[11,12] CVOD might be secondary to an

inadequate impediment of corporoveno-occlusion. Precedent studies have emphasized that the tunica albuginea played a pivotal role in corporoveno-occlusion during penile erection.[13] Divergent loci and degree of venous leak, occurring anywhere along the tunica albuginea, have been shown in a canine model and in ED patients.[14,15] Further studies have suggested that CVOD developed in response to endothelial dysfunction and impairment of trabecular smooth muscle owing to multifaceted degenerating processes in most patients.[16] There was a body of evidence ascribing several factors to CVOD, incorporating arterial disease; congenital vascular anomalies; alterations in the cavernosal smooth muscle, trabecula or tunica albuginea; trauma; postpriapism; psychogenic factors; and unknown origin. Current studies support the contention that CVOD is an effect rather than the cause of ED.[17]

Surgical interventions have consisted primarily of penile revascularization surgery for AI and penile venous surgery for CVOD, whatever the mechanism. Surgical niche relies on the potentiality of attaining unaided and natural erection of the penis, even though penile implant has remained the final alternative.

Penile anatomy

Tunica albuginea, encircling corpora cavernosa, acts as a bilayered structure with a complete inner circular layer and an incomplete outer longitudinal layer spreading over the bulbospongiosus and ischiocavernosus proximally, and extending into the glans penis to fashion the distal ligament (Fig. 1).[18] The inner circular layer is circumferentially uniform with projection of intracavernous pillars. The fibro-skeleton collaborating with erection-related vasculatures provided full erection while elicited.[19,20]

The blood supply of the superficial structure of the penis depends on branches from the femoral artery, which provide primary vascularization of the skin and subcutaneous tissues with the anterior scrotal wall included (Fig. 2). The vascular supply of the deep structure arises from the internal pudendal artery, with accessory arteries occasionally originating from branches of vesical, femoral, obturator and external iliac arteries. The internal pudendal artery branches into the common penile, perineal and inferior rectal arteries. The common penile artery ramifies into the dorsal penile, bulbourethral and cavernosal arteries (Fig. 3). DPA supplies blood to the tunica albuginea. The vascular supplies of the corpus spongiosum and urethra stem from the bulbourethral artery to a great extent, sending perforating tributaries to the corpus cavernosum. Furthermore, cavernosal arteries subdivide into a few helicine arteries, contributing to the cavernosal sinusoids. Distally, these three arteries coalesce to provide collateral blood supply to the glans penis.

Venous drainage of the penis supervenes primarily through the superficial and DDVs, set apart by Buck's fascia. The superficial dorsal vein drains blood from the foreskin into saphenous and external pudendal veins, which contributes insignificantly to penile tumescence. Traditionally, single DDV lying in the median grove is considered to receive blood from the glans, corpora spongiosum and corpora cavernosa by way of emissary and circumflex veins, serving as the noticeable fabric involved in channeling blood with detumescence.[21,22] With advanced revelation and better understanding of erection-related veins of the penis, a DDV, a pair of cavernosal veins, and two pairs of PAVs in between the Buck's fascia and tunica albuginea have been shown to be implicated in erection of the penis (Fig. 4).[23] Draining blood from emissary veins, cavernosal veins are situated between the DDV and DPA, enveloped in a distinct perivascular sheath, and reside closely on the tunica albuginea. The cavernosal veins run alongside each corresponding corpus cavernosum and course through the entire length of penis, which might coalesce to one channel at the penile base and drain into the Santorini's plexus. PAVs sandwich each corresponding DPA, coursing along the full penile length. Unlike medial PAV draining emissary veins from the respective corpus cavernosum, lateral PAV receives casual circumflex veins from the corpus spongiosum. Both the medial and lateral PAVs might channel into the DDV or the Santorini's plexus alone at the level of the infrapubic angle. The proportion for drainage of corpora cavernosa through DDV, cavernosal veins and PAVs are 60.5% (range 50.3–69.7%), 11.9% (range 5.8–22.9%) and 11.4% (range 5.2–15.0%) on their own.[24]

Evaluation

With the intention to document the diagnosis of CVOD, prevailing methods have been DICC[25–27] and PDDU[28] with subsequent protocol.[29–33] One protocol of DICC for diagnosis of CVOD was generally adopted. ICP was measured at baseline and at 2-min intervals for 8–10 min after administering 60 mg of papaverine intracavernously. During the infusion phase, the rate of infusion flow was 20 mL/min increments to provoke an ICP of >150 mmHg. At the end of the infusion, the time to decrease to baseline pressure was defined as pressure decay or pressure loss. The rate of infusion flow required to maintain the ICP at 90 mmHg is defined as MF. Subsequently, cavernosography is carried out at an ICP of 90 mmHg. The criteria used to define the CVOD entails MF of ≥30 mL/min, and a pressure loss of >40 mmHg/30 s. However, DICC is not commonly utilized any more in current practice owing to the paucity of concurrence in diagnostic criteria. To further assess suspicious AI, angiography is commonly used as a confirmatory evaluation. Digital subtraction angiography and computed tomography angiography are minimally invasive, and might act as a feasible substitute for the conventional angiographic procedure.[34] Likewise, computed tomography cavernosography could conspicuously raise precision to recognize veins implicated in CVOD.[35] However, there are significant discrepancies in outcomes between studies utilizing various methods.[36,37] In regard to the diagnosis of AI and VOD, hitherto there is no unanimity about convincing criteria. Despite the anatomical details on angiography, the outcomes entails judicious interpretation associated with the functional outcomes acquired from PDDU and/or DICC.[38]

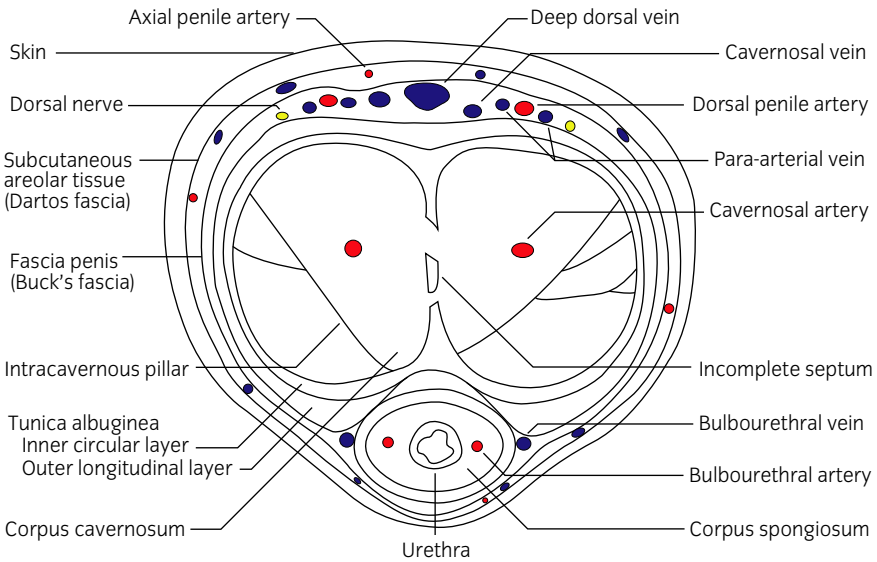

Fig. 1 A transverse section in the midportion of the penis. Tunica albuginea, a bi-layered structure with a complete inner circular layer and an incomplete outer longitudinal layer, encircles the corpora cavernosa. Intracavernous pillars radiate from the inner circular layer, acting as struts. The fibro-skeleton collaborating with erection-related vasculatures provided full erection while elicited.

Penile revascularization surgery

In view of concurrent improvements both in the blood flow of lower extremities and erectile function after endarterectomy of a blocked aortic bifurcation, Leriche first perceived an association of AI with ED.[39] Reduced blood flow into the penis owing to any arterial lesion, whatever the vascular size, might be responsible for ED.[40] For young patients sustaining pelvic trauma and long-distance cyclists, ED has occurred in response to traumatic stenosis of pudendal or cavernous arteries.[41–43] To cure potentially AI, an attempt has been made to skip the lesions, which block the vascular bed of the hypogastric-cavernous artery, through an alternative arterial pathway.

In 1973, Michal *et al*. first devised a procedure composed of direct anastomosis of a donor artery to the corpora cavernosa.[44] After that initial launch, penile revascularization, primarily microarterial bypass surgery, has been subjected to a few refinements. The Michal II technique aimed to increase inflow with end-to-side anastomosis of both the IEA and a DPA (Fig. 5).[45] The other modifications for Michal's techniques have been published over the past decades.[46,47]

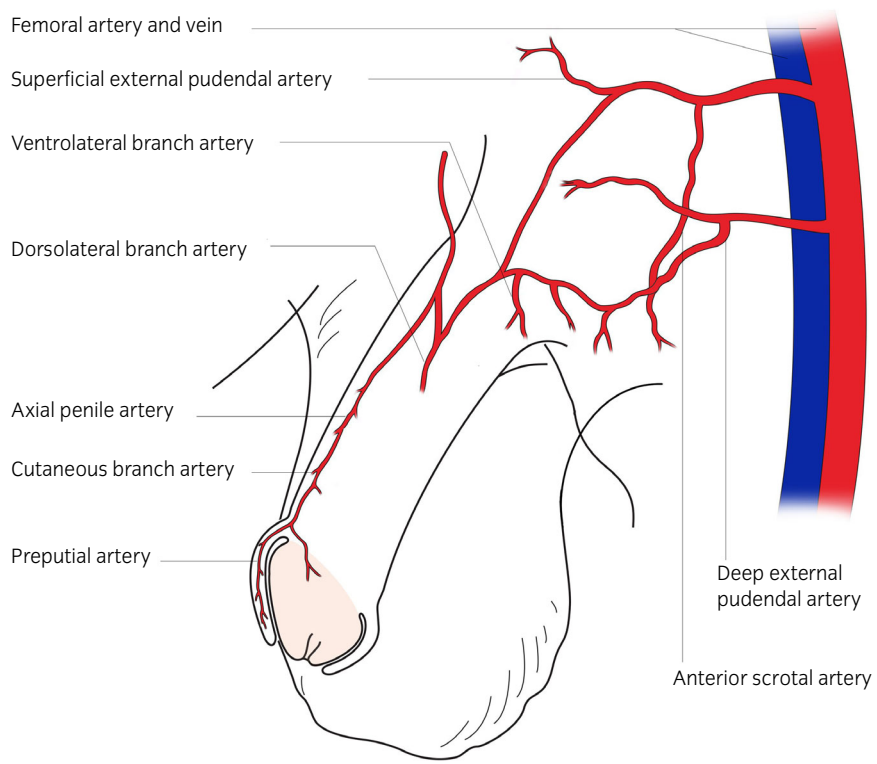

Fig. 2 The blood supply of the superficial structure of the penis depends on branches from the femoral artery, which provide primary vascularization of the skin and subcutaneous tissues.

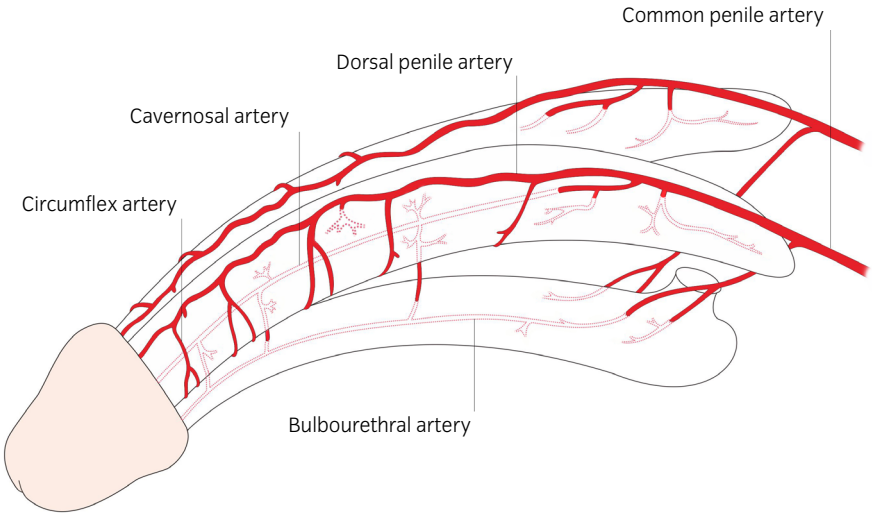

Fig. 3 The vascular supply of the deep structure of the penis arises from the internal pudendal artery. The common penile artery, one branch of the internal pudendal artery, further ramifies into the dorsal penile, bulbourethral and cavernosal arteries.

Crespo *et al.* reported a technique entailing anastomosis of the femoral artery to the DPA or cavernous artery with an interposed autologous saphenous vein graft.[48] As for a variant technique, direct anastomosis of both the IEA and a cavernous artery was achieved.[49,50] Nevertheless, the attempt to anastomose IEA to a cavernous artery yielded a low success rate.[51,52] These surgeries dictate the need for control of the cavernosous artery with dissection of erectile tissue nearby, usually complicated with fibrosis of the donor artery at the site passing through the tunica. The approach was eventually abandoned. DePalma proposed to utilize aortoiliac reconstruction for restoration of erectile function in patients with vasculogenic ED.[53] By virtue of pudendal canal decompression through the perineal approach, Shafik announced an improvement rate of 80% in patients sustaining stricture or occlusion of the distal internal pudendal artery with poor or non-visualization of penile artery.[54]

In 1981, Virag *et al.* initiated the conception of venous arterializations, and proclaimed the Virag I procedure by incorporating IEA into anastomosis directly to the DDV

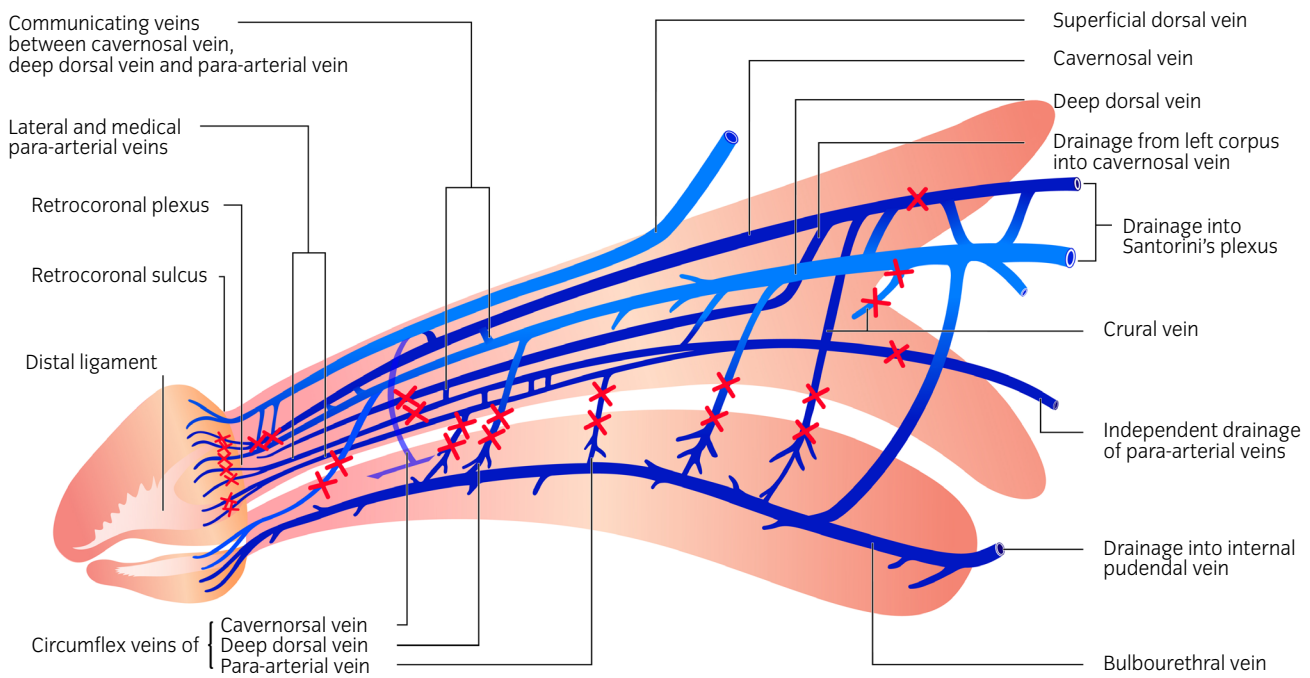

Fig. 4 Schematic illustration of the innovative venous anatomy in the human penis. Lateral view. The DDV lies in the median grove, receiving blood from the glans penis, corpus spongiosum and corpora cavernosa by way of emissary and circumflex veins. A pair of cavernosal veins, and two pairs of PAVs are located in between the Buck's fascia and tunica albuginea. PAVs sandwich each corresponding DPA, coursing along the full length of the penis bilaterally. The innovated venous anatomy of the penis has served as a blueprint for penile venous stripping, and ligation sites (mark "X") are resorted to on the tunical level.

without ligation of the proximal vein.[55] Afterwards, Virag offered modified techniques (Virag II–VI).[56–58] The proximal DDV was ligated in the Virag II procedure. The IEA was anastomosed to the DDV in an end-to-end fashion in the Virag III procedure. Integrated into the Virag I, II and III procedures, a direct shunt was created between the tunica albuginea of the corpus cavernosum and the DDV in the Virag IV, V and VI procedures. Based on the basic procedure, many researchers endeavored to propose various techniques. Furlow and Fisher devised an approach to anastomose the IEA to DDV associated with ligating distal and proximal DDV to avoid the risk of glanular hyperemia (Fig. 6).[59] Shah *et al*. advocated a technique, the Parulkar–Shah antegrade dorsal vein arterialization procedure, which allows for antegrade arterialization of the dorsal vein with beneficial impact on blood flow in the direction of preserved venous valves.[60] Adopting the identical principle applied in the Virag procedure, the distal half of the dorsal vein was divided after complete mobilization at the level of the coronary sulcus, and flipped for end-to-end anastomosis to the IEA on the symphysis pubis.

With respect to anastomosis of both the DPA and IEA, there exists one major obstacle: the discrepancy of lumens between the two blood vessels, which contributes to reduced flow rates, and is vulnerable to thrombosis and consequential occlusion. Hauri advocated "triple anastomosis" that admits a supplementary arteriovenous shunt with good arterial run-off and a high flow rate. This procedure is comprised mainly of direct arterial anastomosis of IEA to DPA, and incorporation of DDV into the anastomosis (Fig. 7).[61,62] In that the principles of penile revascularization encompass proximal or distal ligation of the arterialized vein, ligation of the circumflex vessels, destruction of the valves in the dorsal vein, as well as shunts between artery and vein, many other variations have been proposed.[63–68]

Apart from conventional microvascular surgery, revascularization of larger donor vessels, such as the aorta, common iliac artery and internal iliac artery, has been described. By way of restoration of vascular inflow or restriction of outflow, the technique and principle aim to ultimately enhance erectile function.

Indication

Controversy exists over the indication for penile revascularization surgery. Some investigators consider patients with ED as a result of pelvic trauma as fit for the procedure,[69] whereas opponents preclude such cases from the operation owing to the risk of the negative impact of a neurological disorder on erectile function.

Even with no unanimity on the surgical indication, the ED Guideline Update Panel took for granted that such an arterial reconstructive surgery would be potentially beneficial to an otherwise healthy patient aged <55 years with ED of late attributable to arterial occlusive disease (Table 1).[33] Unlike patients with ED from other causes, select patients with isolated arteriogenic ED are potentially eligible for penile revascularization procedures.[70–72]

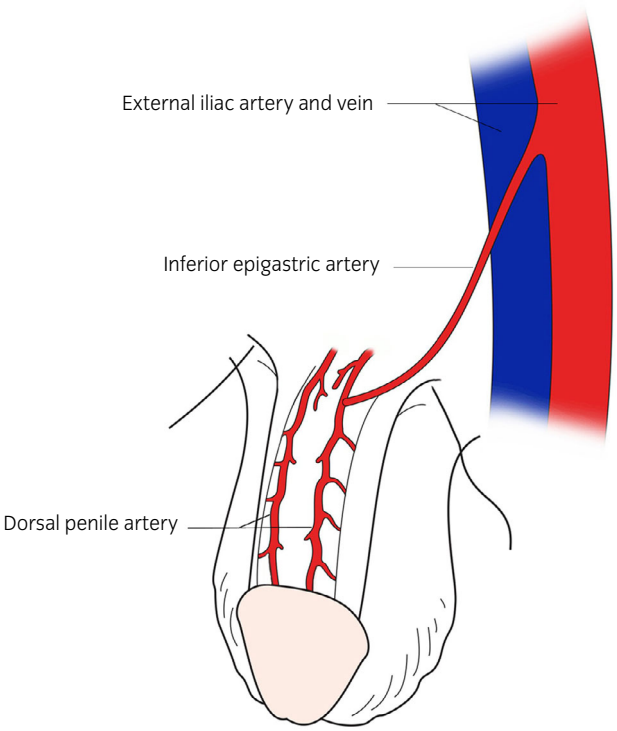

Fig. 5 Anastomosis of the IEA to dorsal penile arteries in an end-to-side fashion (Michal II procedure, true revascularization).

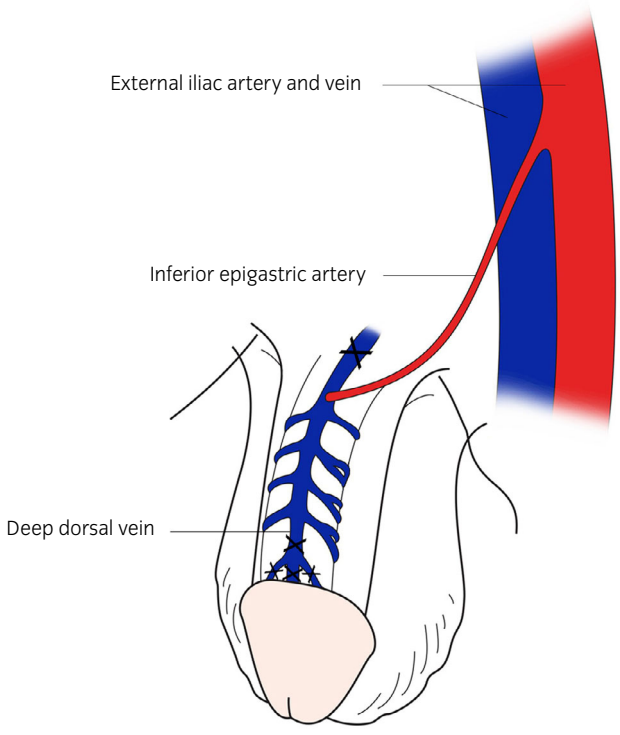

Fig. 6 Anastomosis of the IEA to the DDV with additional proximal and/or distal vein ligation (venous arterializations, Furlow and Fisher procedure). The marks "X" indicate the sites for ligation.

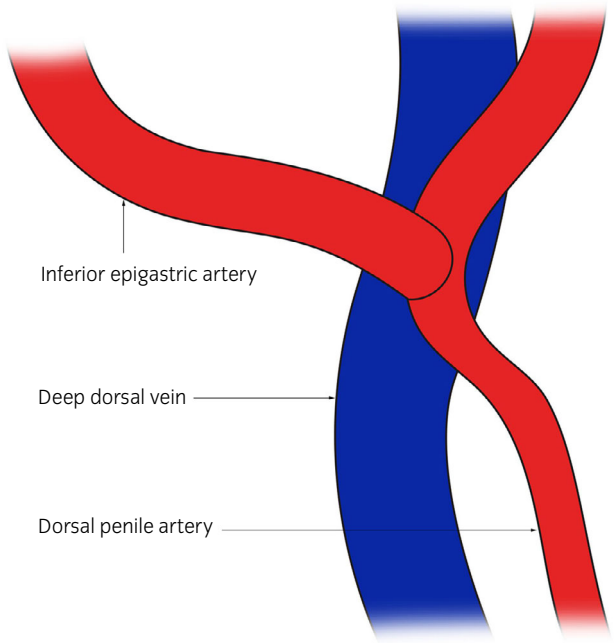

Fig. 7 Anastomosis of the IEA to the DDV and DPA (triple anastomosis with arterial-venous shunt, Hauri's procedure).

Table 1 Inclusion criteria in selecting patients for penile revascularization

1. Age <55 years
2. Absence of vascular risk factors (diabetes, hypertension, tobacco use, hypercholesterolemia)
3. No evidence of neurological erectile dysfunction (e.g. multiple sclerosis, pelvic surgery, lumbosacral radiculopathies etc.)
4. No hormonal abnormalities
5. No evidence of active psychiatric disorders (severe depression, bipolar disease, schizophrenia)
6. No evidence of Peyronie's disease
7. Absence of premature ejaculation
8. Report of acute or chronic perineal or pelvic trauma
9. No evidence of corporo-occlusive dysfunction by duplex Doppler ultrasound and cavernosometry
10. Focal occlusive disease of the common penile or cavernosal arteries documented by penile duplex Doppler ultrasound, cavernosometry and selective internal pudendal arteriography

Surgical technique (microvascular arterial bypass surgery)

The surgical approach for penile revascularization depends on the severity of arterial occlusion, as well as the anatomical variation of the penile artery. The operative procedure involves three steps, as follows.

Dorsal artery and/or vein dissection

A scrotal or infrapubic incision is made at the surgeons' discretion. As for the scrotal approach, an inguinal-scrotal incision is made on the opposite side of the donor-harvested IEA. The intraoperative maneuver to invert the penile shaft and elevate the fundiform ligament with hooks helps reduce the risk of inadvertent damage to the fundiform ligament.[73] After that, dissection and isolation of the dorsal artery or vein can proceed further.

IEA harvesting

For establishment of supplementary arterial blood flow, IEA is commonly utilized for penile revascularization surgery. An open technique, either pararectal or transverse abdominal, is used to harvest the donor IEA.[73,74] To avoid a wide incision and facilitate convalescence, some investigators advocate laparoscopic harvest of the IEA.[75–77] The other minimally invasive procedure to harvest the IEA, the robot-assisted technique, is an alternative supplement to the surgery.[78] The IEA is recognized, transected at the level near the umbilicus and mobilized up to its origin near the femoral artery. Throughout the internal ring, inguinal ligament and external ring, IEA is further transferred to overlie the dorsum of the penile shaft after passing beneath the fundiform ligament. It is feasible to use papaverine hydrochloride topically to obviate the arterial spasm if required.

Microsurgical anastomosis

Vascular anastomosis is carried out at the base of the penis. There are three salient steps towards revasularization surgery of the penis with the assistance of a microsurgical technique in the clinical setting: (i) to anastomose both the IEA and DPA in an end-to-end or end-to-side fashion (true revascularization); (ii) to anastomose both the IEA and DDV with auxiliary ligation of the distal and/or proximal vein (venous arterialization); and (iii) to anastomose the IEA to DDV and DPA (triple anastomosis with an arterio-ovenous shunt).[79] If the dorsal vein is used for revascularization, the dorsal vein is probed in both directions via cutdown to destroy the valves, rendering blood flow into corpora cavernosa retrogradely. The operating microscope is introduced to carry out the anastomosis using interrupted 10-0 nylon microsutures. After vascular anastomosis is completed and scrupulous hemostasis is achieved, the penile shaft is returned into its anatomical position and the wound is closed in layers.

Sexual abstinence, a practice of refraining from all aspects of sexual activity, is advised for >6 weeks postoperatively. Anticoagulants, usually aspirin, can be instituted for 6 months to prevent the formation of blood clots within blood vessels. Selective internal iliac arteriography might be used to document the patency of microvascular anastomosis.

Surgical results

In the era of the 1970s and 1980s, microvascular arterial bypass surgery was carried out to serve as a routine practice for the treatment of AI responsible for ED. However, the surgical effectiveness remained debatable and unproven, mostly owing to the lack of consistent hemodynamic assessment, standardized select patient and validated outcome measures, as well as various surgical procedures. A systematic review

and meta-analysis was carried out by Babaei *et al.* to survey the effectiveness of penile revascularization surgery for the management of arteriogenic ED.[80] Several factors have been identified in sequence to impinge on successful outcomes, including age, the presence of a venous leak, history of smoking and surgical technique utilized. Men aged <30 years sustaining traumatic arterial occlusive disease of the penis would benefit preferably from penile revascularization surgery. Nevertheless, the investigators disclosed that the data were inadequate for analysis attributable to discrepant outcome measurements and non-randomized controlled trials in all studies.

In 2016, the ED Guideline Update Panel carried out a review of accessible literature on vascular surgery for ED, and described the Arterial Occlusive Disease Index patient as a healthy man aged <55 years with recently acquired ED from focal arterial occlusive disease without significant risk factors, such as diabetes, smoking or others. The reported success rate for IEA-to-DDV penile revascularization surgery was 36–80%, and for the IEA-to-DPA procedure was of 91%. A conclusion has been drawn that the effectiveness of penile revascularization surgery remained to be determined due to the disadvantage of a small sample size of 50 patients. A summary of studies evaluating revascularization surgery for the management of ED is adapted in Table 2.[33]

Complications

More complex procedures are more likely subject to related complications. Complications are not uncommon, and occur in approximately 25% of patients receiving penile revascularization surgery.[81,82] Penile edema is expected, but it usually resolves in a week or two. Penile vascularization surgery might potentially invite hyperesthesia of the glans and devastating spongy necrosis. Other complications include loss of penile length (28%), decreased penile sensitivity (24.7%), wound hematoma (7.8–25%), sepsis (3.5%), inguinal hernias (2.8%), wound infection (2.8%) and urinary tract infection (2.6%).[83,84] Trauma to the dorsal nerve might jeopardize penile sensation, which can be obviated by utilizing an operating microscope for microvascular dissection.[85] Penile artery shunt syndrome has been reported to result in persistent ED after revascularization.[86] Embolization of the aberrant obturator artery can be carried out with resolution of the shunt.

Priapism as a sequela of IEA–corpus cavernosum anastomosis is one major complication, which results from the development of non-ischemic priapism and consequential fibrosis of cavernosal tissure. The procedure was soon abandoned. Dorsal vein arterializations, IEA-to-DDV anastomosis, can be complicated with hyperemia of the glans, provoking ulcers of the skin, penile pain and compression of urethra. Glanular hyperemia accounts for 4–21% of patients receiving the surgery, and shows a disastrous complexity that requires surgical intervention in many cases.[81,82,84,87–90] To ward off overperfusion of the glans penis, ligation of all the contributing veins and venules distal to the site for IEA–DDV anastomosis is warranted.

Critical guideline issued

It has been deemed to be controversial and remains unproved for penile revascularization surgery. In the fourth Paris International Consultation on Sexual Medicine in 2015, the panel recommended that men with arteriogenic ED eligible for the procedure should conform to the criteria that characterized the Arterial Occlusive Disease Index patient as no older than 55 years with recently acquired ED attributable to focal arterial occlusive disease, lacking significant risk factors, such as diabetes, smoking or others (LOE = 3; strength of recommendation = C; option). The surgery might not be beneficial to the patients with diabetes or vascular risk factors (LOE = 3; strength of recommendation = B; recommended). Various types of feasible procedures exist for microvascular arterial bypass surgery. Current research cannot prove that one procedure surpasses another (LOE = 3; strength of recommendation = C; option). For vascular repair of aortoiliac occlusive disease, the internal iliac artery should be spared to diminish the risk of associated ED (LOE = 3; strength of recommendation = B; recommended).[33]

In 2018, the American Urological Association Guidelines Panel recommended that penile arterial reconstruction might be considered for young men with ED and focal pelvic/penile arterial occlusion, as well as without documented generalized vascular disease or veno-occlusive dysfunction (conditional recommendation; evidence level: grade C).[91] The European Association of Urology guidelines on ED, premature ejaculation, penile curvature and priapism recommended that penile revascularisation might be taken into account in young patients suffering pelvic or perineal trauma with confirmed AI on penile pharmaco-arteriography.[92]

Penile venous surgery

With the intention to achieve the desired physiological effect, penile venous surgery aims to increase venous impediment for the retention of blood within the corpora cavernosa during penile erection.[93,94] Italian Francesco Parona, in 1873, first described an injection of hypertonic saline into the varicose dorsal vein of the penis on a young patient with impotence to induce sclerosis with a subsequent decrease of inordinate outflow of penile veins. Not until the turn of the 20th century did the conception arise that occlusion of venous channels from the penis by surgery could be beneficial to ED.[95] Some USA doctors initiated resection or ligation of the dorsal vein of the penis; for example James Duncan in 1895, Joe Wooten in 1902 and Frank Lydston in 1908.[95] Oswald Swinney Lowsley, in the early 1930s, proposed an advanced perineal crural technique by the plication of the ischiocavernosus and bulbocavernosus muscles with interrupted mattress sutures, in conjunction with the plication of a simple dorsal vein.[95]

After vanishing in the literature for several decades, surgery for CVOD resurged during the 1980s with the advancement of the physiology of penile erection.[96] Surgical procedures for ligation of penile veins have, over time, progressed from preliminary techniques with ligation of a single vessel, the DDV,

Table 2 Summary of studies evaluating revascularization surgery for the treatment of ED

Reference	Year (*n*)	Mean F/U (months)	Mean age (years)	Study design, diagnosis, population	Surgery type	End-point, measurements	Successful outcome	Adverse and other events
Large vessel revascularization								
Flanigan[135]	1985 (5)	NA	NA	Vasculogenic ED, aortoiliac occlusive disease	Aortobifemoral bypass with internal iliac	NA	100% restored erectile function	Penile/brachial arterial pressure improved from 0.42 to 0.80
Krotovsky[136]	1991 (29)	NA	NA	Aortoiliac occlusive disease	Common femoral to DPA	NA	21% with restored erectile function, 55% with maintained or improved erections	
Urigo[137]	1994 (23)	NA	NA	Aortoiliac occlusive disease, vasculogenic ED	Large vessel angioplasty	NA	57% with improved erections (common or external iliac), 66% (external + internal iliac) and 100% (internal iliac alone) restored erectile function	
DePalma[138]	1995 (17)	38	40–48	Aortoiliac occlusive disease, vasculogenic ED, unresponsive to ICI	Aortoiliac reconstruction	DICC, angiography, NSQ	58% with SEwP, 18% required ICI or vacuum erection device	
Gur[139]	2013 (35)	NA	57	Common iliac occlusion	Recanalization	IIEF-5	62% with improved erections; IIEF-5 score increased from 14 to 20	
Small vessel revascularization								
Grasso[65]	1992 (22)	12	NA	NA	IEA to DV	PDDU, nocturnal penile tumescence	55% with SEwP	
DePalma[138]	1995 (23)	33–48	40–48	Inclusion: unresponsive to ICI, small vessel arterial disease, AI or VOD; exclusion: DM, use of anti-HTN medications, large vessel disease	IEA to DPA (11), IEA to DV (12)	DICC, angiography, NSQ	27–33% with SEwP, 45–47% with ICI	AE: glans hyperemia 17% (in patients with IEA to DV)
Jarow[140]	1996 (11)	12–84 (50)	42	Inclusion: age <60 years, focal arterial lesion (pudendal or common penile); exclusion: vascular risk factors, VOD; surgical ligation of internal iliac artery (2), pelvic fracture (3), blunt trauma (6)	IEA to DPA (8) and DV (2), superficial femoral artery to DPA (1)	Satisfactory intercourse without additional therapy; DICC, PDDU, angiography	Postoperative 82%, last F/U 64% (91% if including ICI)	Long-term F/U 91% improved; AE: hemorrhage 18%
Sarramon[84]	1997 (114)	17	48	91% with failed ICI, 9% refused ICI; 91% with vascular pathology, 9% with neuropathy, psychiatric or hypogonadism; AI (39), VOD (23), or AI + VOD (53); no exclusion for age, vascular risk factors, or smoking	Michal II (44), FFV5 (70), procedure individualized	DICC, PDDU, angiography	1 month: 70% good, 12% improved; last F/U: 48% good, 15% improved	VOD treated with FFV5 most successful, AI treated with Michal II least successful; AEs: glans hyperemia 21% of FFV5, parietal hematoma (8%), parietal sepsis (4%)
Ang[141]	1997 (6)	20	44	33% AI, 67% AI + VOD	IEA to DV	DICC, angiography	33% excellent, 33% improved	
Manning[89]	1998 (62)	41	48	Unresponsive to ICI; exclusion criteria NR; 84% AI + VOD, 21% neurogenic ED, 13% psychogenic ED	Virag (7), Hauri (13), Lobelenz (42)	SEwP or ICI responsive erections; PDDU, angiography, NSQ	54% overall (34% SEwP; 20% with ICI), ≤50 *vs* >50 years old (68% *vs* 39%), <2 *vs* ≥2 vascular risk factors (58% *vs* 48%), DM (43%)	AEs: glans hyperemia 13%, shunt thrombosis 8%, inguinal hernia 7%; progressive failure rates over time (85% at 6 months, 54% at >30 months); Hauri least successful, modified triple anastomosis most successful
Lukkarinen[82]	1998 (26)	≥12	46–51	AI with focal pudendal lesion, unresponsive to oral and ICI; exclusion: DM	Virag V (6), Hauri (4), FFV5 (14)	"good" = erection during intercourse without therapy, "moderate" = erection with ICI; DICC, PDDU	46% good, 33% moderate	AE: glans hyperemia 13%

Table 2 (Continued)

Reference	Year (*n*)	Mean F/U (months)	Mean age (years)	Study design, diagnosis, population	Surgery type	End-point, measurements	Successful outcome	Adverse and other events
Sarramon[142]	2001 (38)	61	52	Retrospective, IIEF mailed, compared outcomes with ED controls used during IIEF validation, unresponsive to ICI; inclusion: >45 years old (patients with AI), AI and VOD	IEA to DV	DICC, PDDU, IIEF	25% with no ED, 28% with mild ED	All IIEF domains improved over ED controls
Vardi[143]	2004 (52)	71	29	Penile arterial occlusion by angiography; exclusion: severe VOD and no contrast seen in spongiosa tissue (cavernosography), 12 patients with prior pelvic trauma	IEA to DV and/or DPA	Satisfactory intercourse without additional therapy, NSQ	48% with SEwP, 29% with PDE5i or ICI, <28 *vs* ≥28 years old (73% *vs* 23%), smoker vs nonsmoker (29% *vs* 57%), moderate *vs* no VOD (27% *vs* 73%)	Type of procedure non-significant; additional 29% responded to ICI or PDE5i; no differences in 2- *vs* 5-year F/U
Kawanishi[144]	2004 (51)	21	32	AI with focal lesions, 33 patients with blunt trauma; inclusion: age <50 years, no history of DM, HTN, hyperlipidemia, cardiovascular risk factors (smoking permitted), or VOD	Hauri (26), FFV5 (23)	Satisfactory intercourse without additional therapy; DICC, PDDU, angiography	86% (3 years), 68% (5 years), Hauri *vs* FFV5 at 5 years (66% *vs* 71%)	AEs: glans hyperemia 4%, hemorrhage 2%, scar contracture 2%
Kayigil[145]	2008 (43)	22	60	AI (13), VOD (21), AI + VOD (9); exclusion: vascular risk factors	FFV5	PDDU, IIEF-15, success: ≥5-point increase	61%, IIEF-15 pre- *vs* postoperatively (19 *vs* 29)	No age difference for <60 *vs* ≥60 years
Munarriz[74]	2009 (71)	35	31	Retrospective; inclusion: age <55 years; exclusion: vascular risk factors, neurologic ED, untreated hormonal abnormalities, psychogenic disorders, Peyronie's disease, premature ejaculation, VOD	IEA to DPA	DICC, PDDU, angiography, IIEF, CES-D, Erectile Dysfunction Inventory of Treatment Satisfaction	IIEF-EFD pre- *vs* postoperatively (14 *vs* 24), 55% with EFD score ≥26, 73% with EFD score ≥21, 81% somewhat to very satisfied	AEs: loss of penile length (28%), decreased penile sensation (25%), inguinal hernia (3%)
Raynor[78]	2010 (5)	NS	17–25	Traumatic pelvic injuries, normal erections before injury, AI, confirmed lesions	Modified Virag V	PDDU, angiography	80% success	Reported roboticassisted harvesting of IEA
Zuckerman[146]	2012 (17)	37	33	Retrospective, cohort with pelvic fracture urethral injury, performed before urethral reconstruction if bilateral pudendal artery occlusion (4) or if AI unresponsive to ICI (13), younger patients with few comorbidities	IEA to DPA	PDDU, angiography	82% (overall): 6% (SEwP), 29% (PDE5i), 18% (ICI), 29% (PDE ± ICI)	Dorsal peak systolic velocity increased from 27 to 28 cm/s preoperatively to 45–47; no AE
Kayigil[147]	2012 (110)	73	43	Prospective, AI (18), VOD (79), AI + VOD (13); no exclusion for vascular risk factors or age	FFV5	≥5-point increase (IIEF-5) or >26; DICC, PDDU, IIEF	IIEF score >26: 82% (3 months), 77% (1 year), 66% (3 years), 64% (5 years); IIEF-5 score before *vs* after procedure (7 *vs* 17), >2 vascular risk factors *vs* 0 (20% *vs* 93%), age <50 *vs* ≥50 years (66% *vs* 60%)	No difference among AI, VOD, or mixed; AEs: glans hyperemia (6%), minor loss of penile skin sensation (5%), incisional hernia (5%)

Adapted and revised from Trost *et al.*[33]

to more complex surgeries. Plication of the penile crura, spongiolysis and excision of the penile veins can be carried out alone or in combination.[97,98] There resided disputable issues on the presence of a standard approach, which involves ligation and excision of the DDV and its tributaries,[96,99,100] and the existence of a surgical technique targeted at the locations where the leakage developed shown on cavernosography.[101] Some investigators carried out a more expansive operation with ligation of the cavernosal veins, crural veins and DDV with its tributaries.[102] It has been generally believed that only early success with few endurable cures was achieved from these sorts of surgeries. There were many techniques used for complete removal of penile veins; however, the procedures might engender thermal injury due to electrocautery and, to some extent, damage the integrity of the skeletal muscles surrounding the bulbous spongiosun and crura, and smooth muscles of cavernosa as well.[101,103–106] Although some urologists embraced the theoretical principle from a favorable viewpoint,[107,108] the American Urological Association panel in 1996 adapted the guidelines on ED and recommended that penile venous surgery was not justified in routine use. In reality, this surgery has been held in abeyance.[109–111]

In tandem with a better understanding of penile venous anatomy, painstaking studies have been carried out on defrosted and fresh cadavers to omit the psychological impact on penile erection.[23] Investigators concluded that penile veins are crucial in attaining a rigid erection.[112–114] Renovation of the venous anatomy of the penis has inspired the latest refinement of venous surgery. Hsu *et al*. resorted to an even more radical technique, refined penile venous stripping, achieved using local anesthesia as an ambulatory surgery.[115,116]

Indication

Penile venous surgery for CVOD could be reserved for patients with medically refractory ED. Several publications have recommended penile venous surgery in a highly selected population. Several factors have been identified to augur well in young men with primary ED, including preoperative duration of ED of ≤7 years, normal hormone levels, adequate arterial flow, normal corpus cavernosum electromyography and MF of ≤45 mL/min on DICC.[117]

Cakan *et al.* advised that long-term results could be improved remarkably with the aid of cautious appraisal of candidates.[118] Positive prognostic factors involved preoperative age <40 years, duration of ED <2 years, non-smoker patients, non-neurogenic disease and distal disease. Cayan recommended crural ligation to treat primary CVOD in young patients. In anticipation of a successful outcome, two components have been recognized in young men, including normal circulation of penile arteries without offending factors, such as diabetes.[102] Hsu *et al*. reported a good response to a refined penile venous stripping procedure in highly selected patients.[116] Exclusion criteria included untreated chronic systemic disease (e.g. chronic liver disease, renal failure, diabetes mellitus, hormonal insufficiency or psychological disorders), major pelvic surgery, pelvic trauma, post-prostate surgery and other obvious etiologies. Flores *et al*. drew a conclusion that in a highly selected population of young men with isolated crural venous leak, crural ligation surgery cured 70% and improved erectile function in more than 90% of men.[119]

Surgical technique (penile venous ligation/stripping surgery)

By virtue of the conventional conceptualized venous anatomy of the penis, the DDV is lined on both sides with one set of DPAs in amidst the tunica albuginea and Buck's fascia. For correction of the venous leakage shown on radiography, diverse techniques have been proposed.

Regarding access to the venous system of the penis, the trans-scrotal approach was reported with favorable outcome by Mulhall *et al*.[120] Rahman *et al.* favored a 3-inch inguinoscrotal incision approach.[121] While a parapenile inguinal incision is made, the penile shaft is delivered in a gooseneck fashion into the operative area. Incising Buck's fascia overlying the dorsal aspect of the penis, DDV is ready for identification and isolation. Attention must be paid to prevent injuring one set of DPAs and dorsal nerves, respectively. DDV is stripped from the area 1-cm proximal to the coronary sulcus to the level of the symphysis pubis, in conjunction with transection and ligation of its tributaries. Crural veins, cavernosal veins and suspensory ligaments are all spared.[109] To make an amendment to a solitary crural venous leak, an umbilical tape is utilized to ligate the crus proximal to the inlet of cavernous artery in expectation of shutting off the venous leakage.[97]

While the ligation procedure is completed and scrupulous hemostasis is achieved, the penis shaft is returned to its anatomical position and the wound is closed in layers. In the clinical setting, sexual abstinence, a practice of refraining from all aspects of sexual activity, is advised for >6 weeks postoperatively. For moving toward the DDV complex overlying the anterior aspect of the prostate, the laparoscopically trans-extraperitoneal approach was proposed for penile venous surgery. Suture ligation was carried out cautiously, while leaving the dorsum of the penis intact. Nevertheless, there was still no obtainable data in terms of the success rate through the laparoscopic approach over a long-term follow-up period.[122]

For currently refined penile venous stripping surgery, the innovative venous anatomy of the penis has served as a blueprint.[115,123] A circumferential incision is first made, and Buck's fascia overlying the dorsal aspect of the penis is entered. DDV is ready for identification and stripping initiated from the level of the coronary sulcus distally. With the trunk of the DDV serving as guidance, a pull-through maneuver of the DDV system is achieved stepwise up to the level of the penile hilum proximally to minimize damage of penile tissue. Likewise, cavernosal veins are managed in the same fashion. Ligation of engorged PAVs and circumflex veins are also executed. After that, an infrapubic incision is carried down to deliver the trunks of cavernosal veins and DDV, respectively. Cardiovascular and DDV systems deep seated in the infrapubic space are subsequently transected at the level of the infrapubic angle distal to the entrance of the DDV into the Santorini's plexus. Crural veins are ligated as close as

Table 3 Summary of studies evaluating venous surgery for the treatment of ED

Reference	Year (*n*)	Mean F/U (months)	Mean age (years)	Study design, diagnosis, population	Surgery type	End point, measurements	Successful outcome	Adverse and other events
DePalma[138]	1995 (27)	33–48	40–48	Inclusion: unresponsive to ICI, VOD; exclusion: DM, use of anti-HTN medications, large vessel disease	Venous ligation	DICC, angiography, NSQ	33% with SEwP, 44% with ICI	
Kim[125]	1995 (15)	19–45 (29)	NA	VOD on PDDU and DICC	Excising the deep dorsal, cavernous and, if necessary, crural veins	Questionnaire	60% erections sufficient for unaided coitus on more than 75% of attempts	AE: contracture of the penis 40%
Ng[148]	1995 (30)	15.6	40	VOD on DICC	ligation and stripping of the deep dorsal vein, ligation of all its emissary and circumflex tributaries, with crural plication and suspensory ligament re-approximation	NA	adequate erections for normal coitus at 1, 3, 6, 9 and 12 months were 76.2%, 61.9%, 52.4%, 42.9% and 38.1%, respectively	
Hassan[149]	1995 (32)	8–66 (29)	33–77 (45)	VOD on DICC	Ligation of the penile cavernosal and crural veins	DICC, patient report, questionnaire	31% with SEwP, 28% full erection with ICI	
Schultheiss[117]	1997 (126)	6–76	20–71 (48)	VOD on PDDU or DICC	DDV ligation	Patient report, questionnaire	11.2% spontaneous erections	AE: 15.1% skin necrosis requiring surgical revision (1), penile deviation (3), sensitivity impairment (8), minor haematoma (1), pain in the glans or shaft region (4), (subjective) penile shortening (2)
Lukkarinen[82]	1998 (21)	≥12	46–51	VOD (DICC), unresponsive to oral and ICI; exclusion: DM	Venous ligation for VOD	"Good" = erection during intercourse without therapy, "moderate" = erection with ICI; DICC, PDDU	Venous ligation (29% good, 52% moderate),	Age not significant predictor; rapid decrease in efficacy from 76% (VOD surgery) initially to 29% at last F/U
Al Assal[150]	1998 (325)	1–13	18–62 (45)	NA	Ligation of DDV, abnormal veins, cavernosal veins, cavernosal-spongiosum shunts	NS	Age <40 years: 76% cured; age >40 years: 58% cured	
Basar[151]	1998 (26)	25	NA	NA	Venous ligation	NS	at 6 months: 15% complete erection, 23% partial erection	
Sasso[131]	1999 (23)	12	20–50 (41)	VOD fulfilling at least 3 criteria: mild cavernous leak on cavernosometry, >30% cavernous smooth muscle tissue, normal analogical corpus cavernosum electromyography, cavernosal oxygen tension >65 mmHg at erection, >50 years	Superficial deep dorsal, Circumflex and emissary vein ligation	NS	74% spontaneous erections at 12 months, 55% long-term	

Table 3 (Continued)

Reference	Year (*n*)	Mean F/U (months)	Mean age (years)	Study design, diagnosis, population	Surgery type	End point, measurements	Successful outcome	Adverse and other events
Popken[130]	1999 (122)	70	19–78 (49)	Non-responders to ICI with a maintenance flow of less than 40 mL min^{-1}	Superficial, deep and circumflex vein ligation	Questionnaire	14% spontaneous erections, 19% ICI response	
Da Ros[132]	2000 (32)	>36	23–66 (55)	VOD on cavernosometry	Extended ligations (occasionally crural plication)	Patient report	22% spontaneous erections	
Cakan[118]	2004 (134)	54	21–72 (39)	VOD on PDDU and DICC	Extended ligations (no crural vein ligation)	Patient report	25.7% spontaneous or PDE5i-supported erections	
Rahman[121]	2005 (11)	34	22–39 (28)	Congenital VOD on PDDU, RigiScan and DICC	Crural ligation	IIEF score	80% subjective success score from 8.9 to 17.5 (median)	
Hsu[128]	2006 (45)	37	58	Persistent VOD, prior venous ligation surgery; exclusion: DM, renal failure, hormonal insufficiency, trauma; compared redo venous ligation (45) with men not electing redo surgery (38; controls)	Redo venous ligation	IIEF-5, cavernosography	IIEF-5 pre- *vs* postoperatively, first *vs* second ligation (10 *vs* 17 *vs* 21); 9%, 7%, 7% requiring PDE5i, ICI, and PP, respectively	91% positive response to second venous surgery
Cayan[102]	2008 (26)	43	35	Inclusion: VOD, unresponsive to PDE5i, ICI; exclusion psychogenic ED; 12% with comorbid conditions	Superficial and deep DV ligation, crural ligation	PDDU, cavernosography, IIEF-5	IIEF-5 pre- *vs* postoperatively (7 *vs* 16), complete erectile restoration (42%), partial restoration (31%)	2% of men presenting with sexual dysfunction qualified; no major AE
Hsu[127]	2010 (16)	92	39	Prior venous surgery, VOD; exclusion: trauma, DM, hormonal insufficiency	Redo venous ligation	IIEF-5, cavernosography	IIEF-5 pre- *vs* postoperatively (9 *vs* 17); 31%, 6%, 13% requiring PDE5i, ICI, and PP, respectively	
Flores[119]	2011 (17)	16	29	Isolated crural venous leak on DICC; exclusion: Peyronie's disease, response to ICI; 20% with single vascular risk factor	Crural ligation	DICC, IIEF-6	66% with SEwP, 33% required PDE5i, 93% with improved erectile function; IIEF-6 pre- *vs* postoperatively (18 *vs* 24)	

Table 3 (Continued)

Reference	Year (*n*)	Mean F/U (months)	Mean age (years)	Study design, diagnosis, population	Surgery type	End point, measurements	Successful outcome	Adverse and other events
Shaeer[152]	2014 (26)	48	39	Prospective, VOD; venous ligation performed, then ICI administered; group 1 responded to first ICI, group 2 responded to second ICI attempt, group 3 did not respond and underwent PP; excluded: AI, psychogenic, neurogenic, hormonal, fibrotic, traumatic etiologies; vascular risk factors and deep system VOD	Venous ligation, PP in non-responders	DICC, PDDU, IIEF-5; success = no ED on IIEF-5	Success: group 1 (75%), group 2 (0%); IIEF-5 before *vs* after procedure: group 1 (4 *vs* 23), group 2 (4 *vs* 13), group 3 (3 *vs* 25)	

Adapted and revised from Rao and Donatucci,[133] Sohn *et al.*[79] and Trost *et al.*[33]

possible to the level of the bulbocavernosus muscle. The erection-related penile veins are hitherto all ligated (Fig. 4). With electrocautery spared to avoid destruction of tissue, ligation of the penile veins and hemostasis is achieved with interrupted 6-0 nylon sutures throughout the entire procedure.[124] Anticoagulants, usually aspirin, can be reinstituted as early as 1 week postoperatively.

Surgical results

Most researchers presented retrospective studies. Furthermore, the data cannot be analyzed owing to a paucity of standardized patient selection, inconsistent hemodynamic evaluation, various operative procedures, different outcome measures and different follow-up periods.

Short-term outcomes of penile venous surgery on early publications seemed encouraging, nevertheless, there yielded no comparatively positive results during the period of long-term follow up.[96,99,100,102] Success rates varied from 23% to 80% within the first year, yet deteriorated consistently while time elapsed on follow up.[109,117,118] The decrease in success rate with time lapse has been ascribed most likely to insufficient ligation, reconnected penile veins, cavernoso-spongiosal leakage, dysfunctional smooth muscle and unidentified vascular lesions, as well as inadequate patient selection.[96,99,100,108,125]

In the literature, Wespes *et al.* reported that 50% of selected patients afforded restoration of erectile function after resection of the DDV.[126] Cayan declared that 26 patients benefited from resection of the penile veins, including superficial and DDVs, as well as ligation of the cavernous vein and two crura with umbilical tape. IIEF scores improved significantly in biostatistics during a follow-up period of >12 months postoperatively.[102] Good results have also been attained in an attempt to directly ligate the offending channels of penile veins among patients with congenital or post-traumatic lesions induced by solitary venous leakage.[119]

Using the latest refined penile venous stripping surgery, Hsu *et al.* proclaimed that 90.4% of selected patients achieved an improvement rate on long-term follow up.[116] As for patients experiencing declined capability of erection attributable to prior venous surgeries, whether penile deformity was present or not, salvaging penile venous stripping surgery has been reported to give modest results as well.[127] Furthermore, Hsu inferred that clinically relapsed ED occurred in patients receiving prior venous surgery as a sequela of residual rather than recurrent veins of the penis.[128] Wen *et al.* proposed a remedy for CVOD through the synergistic effects exerted by venous surgery and medication with oral sildenafil.[129] For the treatment of ED, a summary of evaluations appraising penile venous surgery is adapted in Table 3.[33]

Complications

Complications can occur during or after a surgical procedure. Among them, listed as perioperative complications are transient or irreversible numbness, skin necrosis, penile pain,

penile curvature, wound or tape infection, penile deformity, painful erections, lymphedema, postoperative shortening and inadvertent ligation of the penile artery.[82,102,117,125,130–134] Berardinucci *et al*. described complication rates of 15%, which include penile deviation, adhesions amidst the skin and the penis, as well as transient paresthesia of the penis.[109] Freedman *et al*. reported complications including penile shortening (43%), excessive penile edema (33%), hypoesthesias (20%), excessive pain (17%), hematoma (15%) and scarring requiring revision (2%).[101] Ligation of crural veins, spongiolysis and closure of spongiocavernosal shunts have served as adjunct procedures that provided no supplementary benefit, but higher morbidities. Hsu *et al*. reported no associated significant complications in their series on long-term follow up.[116] Minor complications seemed to be negligible, including temporary edema of the penis, formation of hematoma and bruising.

Critical guideline issued

In 2015, the panel in the fourth Paris International Consultation on Sexual Medicine concluded that venous surgery or embolization is not recommended for the treatment of CVOD; regardless, the procedures can be carried out under the circumstances of clinical research with informed consent, standardized methods of diagnosis and surgical treatment. Standardized questionnaires (IIEF) and long-term (a minimum of 24 months) follow up are also required (LOE = 4; strength of recommendation = C; option).[33]

In 2018, the American Urological Association Guidelines Panel proclaimed that penile venous surgery is not recommended for patients with ED (moderate recommendation; evidence level: grade C).[91] The European Association of Urology guidelines on ED, premature ejaculation, penile curvature and priapism also disclaimed penile venous surgery to be an option for management of ED.[92]

Conclusions

Nowadays, there is no convincing evidence available to draw conclusions that supplant these guidelines recommended by the panels on ED. Numerous physicians query whether or not penile vascular surgery addresses the underlying pathology. Sarcastically, the same concern should apply to current options recommended in the guidelines. Integration or combination of vascular surgery into goal-approached therapies, which include psychological counseling, medical (e.g. phosphodiesterase type 5 inhibitor and intracavernosal injection), non-surgical (e.g. vacuum device and low-intensity shock wave therapy) and alternative surgical intervention (e.g. penile prosthesis), would optimize patient-centered management strategies. It is noteworthy that penile venous surgery might be beneficial to selected patients with CVOD, especially with a better understanding of the innovative venous anatomy of the penis. To afford opportunities for the improvement of ED, penile vascular surgery remains a viable option, and has found its niche in the possibility of obtaining spontaneous, unaided and natural erection.

Acknowledgments

We are grateful to Mr Jacky Wang and Ms Hsiu-Chen Lu for their gracious assistance with artwork, which was greatly enhanced.

Conflict of interest

None declared.

References

1 Saenz de Tejada I, Angulo J, Cellek S *et al*. Pathophysiology of erectile dysfunction. *J. Sex. Med.* 2005; **2**: 26–39.
2 Rosen RC, Fisher WA, Eardley I, Niederberger C, Nadel A, Sand M. The multinational Men's Attitudes to Life Events and Sexuality (MALES) study: I. Prevalence of erectile dysfunction and related health concerns in the general population. *Curr. Med. Res. Opin.* 2004; **20**: 607–17.
3 Gratzke C, Angulo J, Chitaley K *et al*. Anatomy, physiology, and pathophysiology of erectile dysfunction. *J. Sex. Med.* 2010; **7**: 445–75.
4 Chung WS, Shim BS, Park YY. Hemodynamic insult by vascular risk factors and pharmacologic erection in men with erectile dysfunction: Doppler sonography study. *World J. Urol.* 2000; **18**: 427–30.
5 Koca O, Caliskan S, Ozturk MI, Gunes M, Kilicoglu G, Karaman MI. Vasculogenic erectile dysfunction and metabolic syndrome. *J. Sex. Med.* 2010; **7**: 3997–4002.
6 Tarhan F, Kuyumcuoglu U, Kolsuz A, Ozgul A, Canguven O. Cavernous oxygen tension in the patients with erectile dysfunction. *Int. J. Impot. Res.* 1997; **9**: 149–53.
7 Nehra A, Azadzoi KM, Moreland RB *et al*. Cavernosal expandability is an erectile tissue mechanical property which predicts trabecular histology in an animal model of vasculogenic erectile dysfunction. *J. Urol.* 1998; **159**: 2229–36.
8 Krane RJ, Goldstein I, Saenz de Tejada I. Impotence. *N. Engl. J. Med.* 1989; **321**: 1648–59.
9 Lue TF, Takamura T, Umraiya M, Schmidt RA, Tanagho EA. Hemodynamics of canine corpora cavernosa during erection. *Urology* 1984; **24**: 347–52.
10 Lue TF, Hricak H, Schmidt RA, Tanagho EA. Functional evaluation of penile veins by cavernosography in papaverine-induced erection. *J. Urol.* 1986; **135**: 479–82.
11 Rajfer J, Rosciszewski A, Mehringer M. Prevalence of corporeal venous leakage in impotent men. *J. Urol.* 1988; **140**: 69–71.
12 Fuchs AM, Mehringer CM, Rajfer J. Anatomy of penile venous drainage in potent and impotent men during cavernosography. *J. Urol.* 1989; **141**: 1353–6.
13 Lue TF. Physiology of penile erection and pathophysiology of erectile dysfunction. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA (eds). *Campbell-Walsh Urology*. Elsevier Saunders, Philadelphia, 2012; 688–720.
14 Fournier GR Jr, Juenemann KP, Lue TF, Tanagho EA. Mechanisms of venous occlusion during canine penile erection: an anatomic demonstration. *J. Urol.* 1987; **137**: 163–7.
15 Virag R, Paul JF. New classification of anomalous venous drainage using caverno-computed tomography in men with erectile dysfunction. *J. Sex. Med.* 2011; **8**: 1439–44.
16 Montorsi F, Adaikan G, Becher E *et al*. Summary of the recommendations on sexual dysfunctions in men. *J. Sex. Med.* 2010; **7**: 3572–88.
17 Nehra A, Goldstein I, Pabby A *et al*. Mechanisms of venous leakage: a prospective clinicopathological correlation of corporeal function and structure. *J. Urol.* 1996; **156**: 1320–9.
18 Hsu GL, Brock G, Martinez-pinerio L, Nunes L, Von Heyden B, Lue TF. The three-dimensional structure of the human tunica albuginea: anatomical and ultrastructural level. *Int. J. Impot. Res.* 1992; **4**: 117–29.
19 Hsu GL, Hsieh CH, Wen HS *et al*. Anatomy of the human penis: the relationship of the architecture between skeletal and smooth muscles. *J. Androl.* 2004; **25**: 426–31.
20 Hsieh CH, Hsu GL, Huang YP, Tsai MH, Chen HS. Tunical outer layer plays an essential role in penile veno-occlusive mechanism evidenced from

electrocautery effects to the corpora cavernosa in defrosted human cadavers. *Urology*2015; **86**: 1129–36.
21 Aboseif SR, Breza J, Lue TF, Tanagho EA. Penile venous drainage in erectile dysfunction. Anatomical, radiological and functional considerations. *Br. J. Urol.* 1989; **64**: 183–90.
22 Moscovici J, Galinier P, Hammoudi S, Lefebvre D, Juricic M, Vaysse P. Contribution to the study of the venous vasculature of the penis. *Surg. Radiol. Anat.* 1999; **21**: 193–9.
23 Hsu GL, Hsieh CH, Wen HS, Chen YC, Chen SC, Mok MS. Penile venous anatomy: an additional description and its clinical implication. *J. Androl.* 2003; **24**: 921–7.
24 Hsu G-L, Hung Y-P, Tsai M-H *et al*. The venous drainage of the corpora cavernosa in the human penis. *Arab J. Urol.* 2013; **11**: 384–91.
25 Meuleman EJ, Diemont WL. Investigation of erectile dysfunction. Diagnostic testing for vascular factors in erectile dysfunction. *Urol. Clin. North Am.* 1995; **22**: 803–19.
26 Broderick GA. Evidence based assessment of erectile dysfunction. *Int. J. Impot. Res.* 1998; **10**(Suppl 2): S64–73.
27 McMahon CG, Touma K. Predictive value of patient history and correlation of nocturnal penile tumescence, colour duplex Doppler ultrasonography and dynamic cavernosometry and cavernosography in the evaluation of erectile dysfunction. *Int. J. Impot. Res.* 1999; **11**: 47–51.
28 Sarteschi LM, Montorsi F, Menchini Fabris F, Guazzoni G, Lencioni R, Rigatti P. Cavernous arterial and arteriolar circulation in patients with erectile dysfunction: a power Doppler study. *J. Urol.* 1998; **159**: 428–32.
29 Lee B, Sikka SC, Randrup ER *et al*. Standardization of penile blood flow parameters in normal men using intracavernous prostaglandin E1 and visual sexual stimulation. *J. Urol.* 1993; **149**: 49–52.
30 Yu GW, Schwab FJ, Melograna FS, DePalma RG, Miller HC, Rickholt AL. Preoperative and postoperative dynamic cavernosography and cavernosometry: objective assessment of venous ligation for impotence. *J. Urol.* 1992; **147**: 618–22.
31 Motiwala HG. Dynamic pharmacocavernosometry: a search for an ideal approach. *Urol. Int.* 1993; **51**: 1–8.
32 Alphs HH, Navai N, Kohler TS, McVary KT. Preoperative clinical and diagnostic characteristics of patients who require delayed IPP after primary Peyronies repair. *J. Sex. Med.* 2010; **7**: 1262–8.
33 Trost LW, Munarriz R, Wang R, Morey A, Levine L. External mechanical devices and vascular surgery for erectile dysfunction. *J. Sex. Med.* 2016; **13**: 1579–617.
34 Pereira JA, Bilhim T, Rio Tinto H, Fernandes L, Martins Pisco J, Goyri-O'Neill J. Radiologic anatomy of arteriogenic erectile dysfunction: a systematized approach. *Acta Med. Port.* 2013; **26**: 219–25.
35 Kawanishi Y, Izumi K, Muguruma H *et al*. Three-dimensional CT cavernosography: reconsidering venous ligation surgery on the basis of the modern technology. *BJU Int.* 2011; **107**: 1442–6.
36 Teloken PE, Park K, Parker M, Guhring P, Narus J, Mulhall JP. The false diagnosis of venous leak: prevalence and predictors. *J. Sex. Med.* 2011; **8**: 2344–9.
37 Dabaja AA, Teloken P, Mulhall JP. A critical analysis of candidacy for penile revascularization. *J. Sex. Med.* 2014; **11**: 2327–32.
38 McMahon CG. Correlation of penile duplex ultrasonography, PBI, DICC and angiography in the diagnosis of impotence. *Int. J. Impot. Res.* 1998; **10**: 153–8.
39 Leriche R. Désobliterations arterielles hautes (oblitération de la terminaison de l'aorte) comme cause des insuffisances circulatoires des membres inférieurs. *Bull. Soc. Chir Paris* 1923; **49**: 1404.
40 Newman HF, Northup JD, Devlin J. Mechanism of human penile erection. *Invest. Urol.* 1964; **1**: 350–3.
41 Levine FJ, Greenfield AJ, Goldstein I. Arteriographically determined occlusive disease within the hypogastric-cavernous bed in impotent patients following blunt perineal and pelvic trauma. *J. Urol.* 1990; **144**: 11147–53.
42 Sangkum P, Levy J, Yafi FA, Hellstrom WJ. Erectile dysfunction in urethral stricture and pelvic fracture urethral injury patients: diagnosis, treatment, and outcomes. *Andrology* 2015; **3**: 443–9.
43 Ricchiuti VS, Haas CA, Seftel AD, Chelimsky T, Goldstein I. Pudendal nerve injury associated with avid bicycling. *J. Urol.* 1999; **162**: 2099–100.
44 Michal V, Kramar R, Pospichal J. Hejhal L. Direct arterial anastomosis on corpora cavernosa penis in the therapy of erective impotence. *Rozhl. Chir.* 1973; **52**: 587–90.
45 Michal V, Kramar R, Pospichal J, Hejhal L. Gefässchirurgie erektiler Impotenz. *Sexualmedizin* 1976; **5**: 15.
46 Sharlip ID. Penile arteriography in impotence after pelvic trauma. *J. Urol.* 1981; **126**: 477–81.
47 Goldstein I. Arterial revascularization procedures. *Semin. Urol.* 1986; **4**: 252–8.
48 Crespo E, Soltanik E, Bove D, Farrell G. Treatment of vasculogenic sexual impotence by revascularizing cavernous and/or dorsal arteries using microvascular techniques. *Urology* 1982; **20**: 271–5.
49 MacGregor RJ, Konnak JW. Treatment of vasculogenic erectile dysfunction by direct anastomosis of the inferior epigastric artery to the central artery to the corpus cavernosum. *J. Urol.* 1982; **127**: 136–9.
50 Carmignani G, Pirozzi F, Spano G, Corbu C, De Stefani S. Cavernous artery revascularization in vasculogenic impotence: new simplified technique. *Urology* 1987; **30**: 23–6.
51 Shaw WW, Zorgniotti AW. Surgical techniques in penile revascularization. *Urology* 1984; **23**: 76–8.
52 Konnak JW, Ohl DA. Microsurgical penile revascularization using the central corporeal penile artery. *J. Urol.* 1989; **142**: 305–8.
53 DePalma RG, Edwards CM, Schwab FJ, Steinberg DL. Modern management of impotence associated with aortic surgery. In: Bargan JJ, Yao ST (eds). *Arterial Surgery: New Diagnostic and Operative Techniques*. Grune and Stratton, Orlando, 1988; 337–48.
54 Shafik A. Pudendal canal decompression in the treatment of erectile dysfunction. *Arch. Androl.* 1994; **32**: 141–9.
55 Virag R, Zwang G, Dermange H, Legman M, Penven JP. Investigation and surgical treatment of vasculogenic impotency (author's transl). *J. Mal. Vasc.* 1980; **5**: 205–9.
56 Virag R. Revascularization of the penis. In: Bennet AH (ed). *Management of Male Impotence*. Williams and Wilkins, Baltimore, 1982; 219–37.
57 Virag R, Saltiel H, Floresco J, Shoukry K, DufourB. Surgical treatment of vascular impotence by arterialization of the dorsal vein of the penis. Experience of 292 cases. *Chirurgie* 1988; **114**: 703–14.
58 Virag R, Bennett AH. Arterial and venous surgery for vasculogenic impotence: a combined French and American experience. *Arch. Ital. Urol. Nefrol. Androl.* 1991; **63**: 95–100.
59 Furlow WL, Fisher J. Deep dorsal vein arterialization: clinical experience with a new technique for penile revascularization. *J. Urol.* 1988; **139**: 543.
60 Shah RS, Parulkar B, Kulkarni V. Antegrade dorsal arterialization (Parulkar-Shah ADVA procedure) for penile revascularization. *Int. J. Impot. Res.* 1994; **6**.
61 Hauri D. A new operative technique in vasculogenic erectile impotence. *World J. Urol.* 1986; **4**: 237–49.
62 Hauri D. Penile revascularization surgery in erectile dysfunction. *Urol. Int.* 2003; **70**: 132–40.
63 Benhamou AC. Technic for the indirect revascularization of corpus cavernosum in sexual impotence from distal arterial origin by double epigastro-veno-cavernous fistula. *Presse Med.* 1985; **14**: 691–3.
64 Juenemann KP. Penile revascularization. In: Mulcahy JJ, ed. *Diagnosis and Management of Male Sexual Dysfunction*. New York: Lippincott Williams & Wilkins, 1992; 1282.
65 Grasso M, Lania C, Castelli M, Deiana G, Francesca F, Rigatti P. Deep dorsal vein arterialization in vasculogenic impotence: our experience. *Arch. Ital. Urol. Nefrol. Androl.* 1992; **64**: 309–12.
66 Lobelenz M, Junemann KP, Kohrmann KU *et al*. Penile revascularization in nonresponders to intracavernous injections using a modified microsurgical technique. *Eur. Urol.* 1992; **21**: 120–5.
67 Schramek P, Engelmann U, Kaufmann F. Microsurgical arteriovenous revascularization in the treatment of vasculogenic impotence. *J. Urol.* 1992; **147**: 1028–31.
68 Frankovicova M, Thoma A. Microvascular treatment of impotence. *Plast. Reconstr. Surg.* 1993; **91**: 99–102.
69 Sharaby JS, Benet AE, Melman A. Penile revascularization. *Urol. Clin. North Am.* 1995; **22**: 821–32.
70 Johnsen NV, Kaufman MR, Dmochowski RR, Milam DF. Erectile dysfunction following pelvic fracture urethral injury. *Sex. Med. Rev.* 2018; **6**: 114–23.
71 Dicks B, Bastuba M, Goldstein I. Penile revascularization–contemporary update. *Asian J. Androl.* 2013; **15**: 5–9.

72 Rowe C, Ganick S, Munarriz R. Traumatic vasculogenic erectile dysfunction: role of penile microarterial bypass surgery. *Curr. Urol. Rep.* 2010; **11**: 427–31.

73 Goldstein I, Bastuba M, Lurie A, Lubisich J. Penile revascularization. *J. Sex. Med.* 2008; **5**: 2018–21.

74 Munarriz R, Uberoi J, Fantini G, Martinez D, Lee C. Microvascular arterial bypass surgery: long-term outcomes using validated instruments. *J. Urol.* 2009; **182**: 643–8.

75 Lund GO, Winfield HN, Donovan JF. Laparoscopically assisted penile revascularization for vasculogenic impotence. *J. Urol.* 1995; **153**: 1923–6.

76 Hatzinger M, Seemann O, Grenacher L, Rassweiler J. Laparoscopy-assisted penile revascularization: a new method. *J. Endourol.* 1997; **11**: 269–72.

77 Kawanishi Y, Kimura K, Nakanishi R, Fukawa T, Numata A. A minimally invasive method for harvesting the epigastric artery for penile revascularization. *BJU Int.* 2004; **94**: 1391–6.

78 Raynor MC, Davis R, Hellstrom WJ. Robot-assisted vessel harvesting for penile revascularization. *J. Sex. Med.* 2010; **7**: 293–7.

79 Sohn M, Hatzinger M, Goldstein I, Krishnamurti S. Standard operating procedures for vascular surgery in erectile dysfunction: revascularization and venous procedures. *J. Sex. Med.* 2013; **10**: 172–9.

80 Babaei AR, Safarinejad MR, Kolahi AA. Penile revascularization for erectile dysfunction: a systematic review and meta-analysis of effectiveness and complications. *Urol. J.* 2009; **6**: 1–7.

81 Bleustein CB, Melman A. Hypervascularity of the glans penis diagnosed with cutaneous temperature measurements. *Int. J. Impot. Res.* 2002; **14**: 543–4.

82 Lukkarinen O, Tonttila P, Hellstrom P, Leinonen S. Non-prosthetic surgery in the treatment of erectile dysfunction. A retrospective study of 45 impotent patients in the University of. *Oulu. Scand. J. Urol. Nephrol.* 1998; **32**: 42–6.

83 Munarriz R. Penile microvascular arterial bypass surgery: indications, outcomes, and complications. *ScientificWorldJournal* 2010; **10**: 1556–65.

84 Sarramon JP, Bertrand N, Malavaud B, Rischmann P. Microrevascularisation of the penis in vascular impotence. *Int. J. Impot. Res.* 1997; **9**: 127–33.

85 Zorgniotti AW, Lizza EF. *Diagnosis and Management of Impotence*. BC Decker, Philadelphia, 1991.

86 Pavlinec JG, Hakky TS, Yang C, Massis K, Munarriz R, Carrion RE. Penile artery shunt syndrome: a novel cause of erectile dysfunction after penile revascularization surgery. *J. Sex. Med.* 2014; **11**: 2338–41.

87 Wolf JS Jr, Lue TF. High-flow priapism and glans hypervascularization following deep dorsal vein arterialization for vasculogenic impotence. *Urol. Int.* 1992; **49**: 227–9.

88 DePalma RG. Vascular surgery for impotence: a review. *Int. J. Impot. Res.* 1997; **9**: 61–7.

89 Manning M, Junemann KP, Scheepe JR, Braun P, Krautschick A, Alken P. Long-term followup and selection criteria for penile revascularization in erectile failure. *J. Urol.* 1998; **160**: 1680–4.

90 Kawanishi Y, Kimura K, Yamaguchi K *et al*. Microsurgical penile revascularization in patients with pure arteriogenic erectile dysfunction. *Nihon Hinyokika Gakkai Zasshi* 2000; **91**: 62–8.

91 Burnett AL, Nehra A, Breau RH *et al*. Erectile dysfunction: AUA guideline. *J. Urol.* 2018; **200**: 633–41.

92 EAU guidelines on erectile dysfunction, premature ejaculation, penile curvature and priapism, 2018.

93 Shafik A, Shafik I, El Sibai O, Shafik AA. On the pathogenesis of penile venous leakage: role of the tunica albuginea. *BMC Urol.* 2007; **7**: 14.

94 Hellstrom WJ, Montague DK, Moncada I *et al*. Implants, mechanical devices, and vascular surgery for erectile dysfunction. *J. Sex. Med.* 2010; **7**: 501–23.

95 Das S. Early history of venogenic impotence. *Int. J. Impot. Res.* 1994; **6**: 183–9.

96 Wespes E, Schulman CC. Venous leakage: surgical treatment of a curable cause of impotence. *J. Urol.* 1985; **133**: 796–8.

97 Lue TF. Surgery for crural venous leakage. *Urology* 1999; **54**: 739–41.

98 Bennett AH, Rivard DJ, Blanc RP, Moran M. Reconstructive surgery for vasculogenic impotence. *J. Urol.* 1986; **136**: 599–601.

99 Williams G, Mulcahy MJ, Hartnell G, Kiely E. Diagnosis and treatment of venous leakage: a curable cause of impotence. *Br. J. Urol.* 1988; **61**: 151–5.

100 Stief CG, Djamilian M, Truss MC, Tan H, Thon WF, Jonas U. Prognostic factors for the postoperative outcome of penile venous surgery for venogenic erectile dysfunction. *J. Urol.* 1994; **151**: 880–3.

101 Freedman AL, Costa Neto F, Mehringer CM, Rajfer J. Long-term results of penile vein ligation for impotence from venous leakage. *J. Urol.* 1993; **149**: 1301–3.

102 Cayan S. Primary penile venous leakage surgery with crural ligation in men with erectile dysfunction. *J. Urol.* 2008; **180**: 1056–9.

103 Puech-Leao P, Reis JM, Glina S, Reichelt AC. Leakage through the crural edge of corpus cavernosum. Diagnosis and treatment. *Eur. Urol.* 1987; **13**: 163–5.

104 Lewis RW. Venous surgery for impotence. *Urol. Clin. North Am.* 1988; **15**: 115–21.

105 Rossman B, Mieza M, Melman A. Penile vein ligation for corporeal incompetence: an evaluation of short-term and long-term results. *J. Urol.* 1990; **144**: 679–82.

106 Katzenwadel A, Popken G, Wetterauer U. Penile venous surgery for cavernosal venous leakage: long-term results and retrospective studies. *Urol. Int.* 1993; **50**: 71–6.

107 Lue TF. Penile venous surgery. *Urol. Clin. North Am.* 1989; **16**: 607–11.

108 Vale JA, Feneley MR, Lees WR, Kirby RS. Venous leak surgery: long-term follow-up of patients undergoing excision and ligation of the deep dorsal vein of the penis. *Br. J. Urol.* 1995; **76**: 192–5.

109 Berardinucci D, Morales A, Heaton JP, Fenemore J, Bloom S. Surgical treatment of penile veno-occlusive dysfunction: is it justified? *Urology* 1996; **47**: 88–92.

110 Montague DK, Barada JH, Belker AM *et al*. Clinical guidelines panel on erectile dysfunction: summary report on the treatment of organic erectile dysfunction. *Am. Urol. Assoc. J. Urol.* 1996; **156**: 2007–11.

111 McMahon CG Current diagnosis and management of erectile dysfunction. *Med. J. Australia* 2019; **210**: 469–76.

112 Hsieh CH, Wang CJ, Hsu GL *et al*. Penile veins play a pivotal role in erection: the haemodynamic evidence. *Int. J. Androl.* 2005; **28**: 88–92.

113 Hsu GL, Hung YP, Tsai MH *et al*. Penile veins are the principal component in erectile rigidity: a study of penile venous stripping on defrosted human cadavers. *J. Androl.* 2012; **33**: 1176–85.

114 Hsieh CH, Liu SP, Hsu GL *et al*. Advances in understanding of mammalian penile evolution, human penile anatomy and human erection physiology: clinical implications for physicians and surgeons. *Med. Sci. Monit.* 2012; **18**: RA118–25.

115 Hsu GL, Hsieh CH, Chen HS *et al*. The advancement of pure local anesthesia for penile surgeries: can an outpatient basis be sustainable? *J. Androl.* 2007; **28**: 200–5.

116 Hsu GL, Chen HS, Hsieh CH, Lee WY, Chen KL, Chang CH. Clinical experience of a refined penile venous stripping surgery procedure for patients with erectile dysfunction: is it a viable option? *J. Androl.* 2010; **31**: 271–80.

117 Schultheiss D, Truss MC, Becker AJ, Stief CG, Jonas U. Long-term results following dorsal penile vein ligation in 126 patients with veno-occlusive dysfunction. *Int. J. Impot. Res.* 1997; **9**: 205–9.

118 Cakan M, Yalcinkaya F, Demirel F, Ozgunay T, Altug U. Is dorsale penile vein ligation (dpvl) still a treatment option in veno-occlusive dysfunction? *Int. Urol. Nephrol.* 2004; **36**: 381–7.

119 Flores S, Tal R, O'Brien K, Mulhall JP. Outcomes of crural ligation surgery for isolated crural venous leak. *J. Sex. Med.* 2011; **8**: 3495–9.

120 Mulhall JP, Martin D, Ergin E, Kim F. Crural ligation surgery for the young male with venogenic erectile dysfunction: technique. *Tech. Urol.* 2001; **7**: 290–3.

121 Rahman NU, Dean RC, Carrion R, Bochinski D, Lue TF. Crural ligation for primary erectile dysfunction: a case series. *J. Urol.* 2005; **173**: 2064–6.

122 Scheplev P, Kadirov Z, Aliev A, Garin N, Gvasaliya B, eds. *Extraperitoneal Laparoscopic Ligation of Veins of Periprostatic Venous Plexus for Veno-Occlusive Erectile Dysfunction*. European Society for Sexual Medicine (ESSM), Copenhagen, Denmark, 2005; p53: P-07-347.

123 Molodysky E, Liu SP, Huang SJ, Hsu GL. Penile vascular surgery for treating erectile dysfunction: current role and future direction. *Arab. J. Urol.* 2013; **11**: 254–66.

124 Hsu GL, Hsieh CH, Wen HS *et al*. The effect of electrocoagulation on the sinusoids in the human penis. *J. Androl.* 2004; **25**: 954–9.

125 Kim ED, McVary KT. Long-term results with penile vein ligation for venogenic impotence. *J. Urol.* 1995; **153**: 655–8.

126 Wespes E, Delcour C, Preserowitz L, Herbaut AG, Struyven J, Schulman C. Impotence due to corporeal veno-occlusive dysfunction: long-term follow-up of venous surgery. *Eur. Urol.* 1992; **21**: 115–9.

127 Hsu GL, Chen HS, Hsieh CH, Lee WY, Chen KL, Chang CH. Salvaging penile venous stripping surgery. *J. Androl.* 2010; **31**: 250–60.

128 Hsu GL, Chen HS, Hsieh CH *et al*. Insufficient response to venous stripping surgery: is the penile vein recurrent or residual? *J. Androl.* 2006; **27**: 700–6.

129 Wen HS, Hsieh CH, Hsu GL *et al*. The synergism of penile venous surgery and oral sildenafil in treating patients with erectile dysfunction. *Int. J. Androl.* 2005; **28**: 297–303.

130 Popken G, Katzenwadel A, Wetterauer U. Long-term results of dorsal penile vein ligation for symptomatic treatment of erectile dysfunction. *Andrologia* 1999; **31**(Suppl 1): 77–82.

131 Sasso F, Gulino G, Weir J, Viggiano AM, Alcini E. Patient selection criteria in the surgical treatment of veno-occlusive dysfunction. *J. Urol.* 1999; **161**: 1145–7.

132 Da Ros CT, Teloken C, Antonini CC, Sogari PR, Souto CA. Long-term results of penile vein ligation for erectile dysfunction due to cavernovenous disease. *Tech. Urol.* 2000; **6**: 172–4.

133 Rao DS, Donatucci CF. Vasculogenic impotence. Arterial and venous surgery. *Urol. Clin. North Am.* 2001; **28**: 309–19.

134 Sohn M. Surgical Treatment of Erectile Dysfunction. In: Porst H, Buvat J (eds). *Standard Practice in Sexual Medicine*. Blackwell Publishing, Massachusetts, 2006; 126–48.

135 Flanigan DP, Sobinsky KR, Schuler JJ, Buchbinder D, Borozan PG, Meyer JP. Internal iliac artery revascularization in the treatment of vasculogenic impotence. *Arch. Surg.* 1985; **120**: 271–4.

136 Krotovsky GS, Turpitko SA, Gerasimov VB *et al*. Surgical treatment and prevention of vasculopathic impotence in conjunction with revascularisation of the lower extremities in Leriche's syndrome. *J. Cardiovasc. Surg. (Torino)* 1991; **32**: 340–3.

137 Urigo F, Pischedda A, Maiore M *et al*. Role of arteriography and percutaneous transluminal angioplasty in the diagnosis and treatment of arterial vasculogenic impotence. *Radiol. Med.* 1994; **88**: 86–92.

138 DePalma RG, Olding M, Yu GW *et al*. Vascular interventions for impotence: lessons learned. *J. Vasc. Surg.* 1995; **21**: 576–84.

139 Gur S, Ozkan U, Onder H, Tekbas G, Oguzkurt L. The effect of endovascular revascularization of common iliac artery occlusions on erectile function. *Cardiovasc. Intervent. Radiol.* 2013; **36**: 84–9.

140 Jarow JP, DeFranzo AJ. Long-term results of arterial bypass surgery for impotence secondary to segmental vascular disease. *J. Urol.* 1996; **156**: 982–5.

141 Ang LP, Lim PH. Penile revascularisation for vascular impotence. *Singapore Med. J.* 1997; **38**: 285–8.

142 Sarramon JP, Malavaud B, Braud F, Bertrand N, Vaessen C, Rischmann P. Evaluation of male sexual function by the International Index of Erectile Function after deep dorsal vein arterialization of the penis. *J. Urol.* 2001; **166**: 576–80.

143 Vardi Y, Gruenwald I, Gedalia U, Nassar S, Engel A, Har-Shai Y. Evaluation of penile revascularization for erectile dysfunction: a 10-year follow-up. *Int. J. Impot. Res.* 2004; **16**: 181–6.

144 Kawanishi Y, Kimura K, Nakanishi R, Kojima K, Numata A. Penile revascularization surgery for arteriogenic erectile dysfunction: the long-term efficacy rate calculated by survival analysis. *BJU Int.* 2004; **94**: 361–8.

145 Kayigil O, Agras K, Okulu E. Is deep dorsal vein arterialization effective in elderly patients? *Int. Urol. Nephrol.* 2008; **40**: 125–31.

146 Zuckerman JM, McCammon KA, Tisdale BE *et al*. Outcome of penile revascularization for arteriogenic erectile dysfunction after pelvic fracture urethral injuries. *Urology* 2012; **80**: 1369–73.

147 Kayigil O, Okulu E, Aldemir M, Onen E. Penile revascularization in vasculogenic erectile dysfunction (ED): long-term follow-up. *BJU Int.* 2012; **109**: 109–15.

148 Ng FC, Ruan XQ, Wong HT, Lim PH. Venous ligation for cavernous leak impotence–a report on 30 cases. *Ann. Acad. Med. Singapore* 1995; **24**: 716–9.

149 Hassan AA, Hassouna MM, Elhilali MM. Long-term results of penile venous ligation for corporeal venous occlusive dysfunction. *Can. J. Surg.* 1995; **38**: 537–41.

150 Al Assal F, Delgado A, Al Assal R. Venous surgery for venoocclusive dysfunction: long-term results. *Int. J. Impot. Res.* 1998; **10S**: 31.

151 Basar M, Atan A, Yildiz M. Long-term results of venous ligation in patients with veno-occlusive dysfunction. *Int. J. Impot. Res.* 1998; **10S**: 21.

152 Shaeer O, Shaeer K. Same-session dorsal vein ligation and testing by intracavernous injection prior to penile prosthesis implantation (DVL-ICI-PPI). *J. Sex. Med.* 2014; **11**: 2333–7.