CURRENT TRENDS IN UROLOGY Volume 4

Edited by
Martin I. Resnick

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Accurate indications, adverse reactions, and dosage schedules for drugs are provided in this book, but it is possible that they may change. The reader is urged to review the package information data of the manufacturers of the medications mentioned.

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To Vicki, Andy, and Jeff

Preface

In keeping with the concepts of previous volumes the series. *Current Trends in Urology*, attempts to present a group of essays that provide a critical review of changing topics that are of importance to all practicing urologists. Technological developments often receive the most attention, but there has been significant advances in both diagnostic and therapeutic modalities as well. Our understanding of bladder function and male potency has been greatly enhanced over the past several years, and the applications of surgery, radiation therapy and chemotherapy in the effective treatment of many malignancies has greatly improved. Many of these areas are addressed in the current volume.

The contributors to this volume are all recognized experts in their particular field of interest and each of the articles reflects their broad experience and thoughts. As noted in the first volume, it is anticipated that the series will continue to be a constant expression of the development of new ideas and concepts important to us all.

Martin I. Resnick, M.D.

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Contents

Chapter 1

Impotence: New Concepts Regarding Therapy

Thomas F. Lue

A better understanding of erectile dysfunction has been made possible by recent innovative laboratory and clinical investigations into the hemodynamics, mechanism, neurophysiology, and pharmacology of penile erection. As a result, better diagnostic tests and more thereapeutic options are now available. Because basic research is the key to the present and future advances in the evaluation and treatment of erectile dysfunction, a brief review of penile physiology will precede the discussion of the management of impotence.

PHYSIOLOGY OF ERECTION

The hemodynamics of penile erection have been controversial for several decades. Erection was thought to be due to increased arterial flow or decreased venous drainage or a combination of both. Recent human and animal studies have concluded that erection results from increases in arterial flow, venous resistance, and sinusoidal compliance.[1–4] The erectile process comprises six phases: flaccid, latent, tumescent, full erection, rigid erection, and detumescence. The cavernous nerves (autonomic nervous system) control the vascular event leading to full erection. Once the corpora cavernosa are filled, contraction of the ischiocavernosus muscles from activation of the pudendal nerve produces rigid erection with intracavernous pressure above systolic pressure.

Recent studies of corrosion casts of human cadaveric penises and of canine and simian penises fixed in the flaccid and erect states have clarified the mechanism of erection. In the flaccid state, the muscles of the arterioles and the sinusoidal walls are contracted. The high resistance allows a minimal amount of flow into the sinusoids. Activation of the cavernous nerves or injection of smooth muscle relaxants such as papaverine relaxes the arterioles and the sinusoids. This drop in peripheral resistance allows a large amount of arterial flow and traps the blood in the now-compliant sinusoids. Expansion of the sinusoidal system compresses the draining subtunical venular plexus against the tunica albuginea and effectively restricts venous flow during erection.[5]

The recent work of Walsh and Donker,[6] Lue and associates,[7] and Lepor and associates[8] not only improves our understanding of the neuroanatomy of erection but also helps prevent iatrogenic impotence during various pelvic surgeries. The precise course of the cavernous nerves in relation to the prostate and urethra has been well illustrated by these investigations:[6–8] At the apex of the prostate, the nerves are located at the 5 and 7 o'clock positions; at the level of the external sphincter, they are at the 3 and 9 o'clock positions; at the distal urethral bulb, they move to the 1 and 11

o'clock positions to enter the hilum of the penis.

The neurotransmitters controlling the erectile process are under intensive investigation at the present time. Possible candidates include acetylcholine, vasoactive intestinal polypeptide (VIP), endothelial-derived relaxing factor (EDRF), and inhibition of the adrenergic transmission by acetylcholine.[9–13]

Several groups of pharmacologic agents have been found to induce or inhibit penile erection when injected into the corpora cavernosa. The erection inducers include (a) the smooth-muscle relaxants, papaverine and nitroglycerine; (b) the α-adrenergics blockers, phentolamine and phenoxybenzamine; (c) VIP; (d) certain antidepressants such as chloropromazine and trazodone; and (e) prostaglandin E_1. The erection inhibitors include almost all the α-adrenergic agents in clinical use, such as norepinephrine, epinephrine, ephedrine, phenylephrine, metaraminal, and dopamine.[14]

NEW DIAGNOSTIC PROCEDURES

Intracavernous injection of pharmacologic agents has heralded a new era for the functional testing of erectile capability. Experimental studies in the canine and simian models have shown that intracavernous injection of papaverine induces vascular changes similar to those that occur after stimulation of the cavernous nerves, namely increased arterial flow, decreased venous flow, and relaxation of the sinusoids.[15] By applying these findings to the clinical evaluation of impotence, investigators have developed several new techniques to assess the arterial and venous systems before and after injection of pharmacologic agents. These techniques include evaluation of the penile arteries with duplex ultrasonography in papaverine-induced erection, pharmacologic pudendal arteriography, and pharmacologic cavernosometry and cavernosography.

Intracavernous Injection of Pharmacologic Agents

Virag and associates[16] injected of 80 mg of papaverine in patients with psychogenic and organic impotence and reported that, within 12 min, all those with psychogenic impotence developed a full erection that lasted for more than 30 min. Patients with organic impotence did not develop full erection after the injection. Although Buval and associates[17] have reported different findings in psychogenic patients, it is reasonable to assume that, if patients develop full erection within 12 min of injection of 60–80 mg of papaverine, significant vascular impairment is unlikely. However, a lack of full erection after injection does not confirm the presence of vascular disease due to possible psychologic inhibition during such an unnatural test.

Duplex Ultrasound Evaluation of Penile Arteries

Using a sophisticated 10 MHz ultrasound scanner and pulsed Doppler analysis, Lue and associates[18] developed a technique of evaluating the functional capability of the penile arteries. The echogenicity of the corpora cavernosa and the diameter of the cavernous arteries are assessed in the flaccid penis. Several minutes after intracavernous injection of papaverine, the function of the penile arteries is evaluated by recording the increase in diameter, pulsation of the arteries, thickness of the arterial wall, and velocity of the flow in each cavernous and dorsal artery. Although any single parameter may not determine the functional capability, assessment of all these criteria gives a fairly accurate picture of the health of these arteries. My personal experience in evaluating more than 1000 patients with this technique has confirmed its simplicity and superiority compared with other available tests.

Pharmacologic Pudendal Arteriography

Zorgniotti and Lefleur[19] and Virag and associates[16] introduced the technique of selective pudendal arteriography after intracavernous injection of papaverine. Its

major advantage is enhanced visualization of the penile arterial tree without spinal or general anesthesia. Although control standards are lacking, it is the general consensus that arteriography should be done simultaneously with injection to achieve the best visualization.

Cavernosometry and Cavernosography

Cavernosometry and cavernosography were developed by several researchers[20–22] to evaluate the functional capacity of the penile veins. Wagner's technique[20] involves injection of radiopaque dye into the corpora cavernosa during visual erotic stimulation (cavernosography). Virag and associates[21] and Wespes and associates[22] infuse saline intracavernously and record the amount required to initiate and maintain erection (cavernosometry). Recently, pharmacologic agents have been used also to perform these tests (pharmacologic cavernosometry and cavernosography).[23–25] Because intracavernous injection of papaverine has been shown to activate the venous occlusion mechanism in animals,[15] its use makes this a simpler and more physiologic test than previous techniques.

INTRACAVERNOSAL PHARMACOTHERAPY

Virag first reported the use of intracavernous injection of papaverine to improve erectile function in 1982.[26] In a subsequent series of 63 patients who received an average of two injections, 65% reported improvement sufficient for intercourse over an average follow-up period of 13 months.[16] In a recent double-blind, cross-over placebo trial, 29% of organically impotent men reported improved erections after saline injection, and the response rates to papaverine and phenoxybenzamine were not significantly different (T. Keogh, personal communication). In our practice, we have given patients a minimum of three injections of papaverine twice a month and have found that one-third of patients report marked improvement, one-third find some improvement, and the rest experience no change. Although psychologic factors probably account for the majority of the improved results, a few patients were noted to have improved blood flow on pulsed Doppler and ultrasound studies. In those with marked improvement, the majority have elected intermittent injection at monthly intervals; we offer the others the alternative of penile prosthesis or vascular surgery according to the cause of impotence and the patient's own preference.

In 1983, Brindley[27] reported erection leading to intercourse with intracavernous injection of phenoxybenzamine, and one patient was successfully placed on autoinjection. Because of the possible carcinogenicity of phenoxybenzamine, Zorgniotti and Lefleur[19] recommend a combination of papaverine and phentolamine for injection by the patient before sexual intercourse. In their first report of 62 patients with organic impotence,[19] 59 were able to have intercourse after 30 mg of papaverine with 0.5–1.0 mg of phentolamine, and 18 patients were successfully placed on autoinjection. In a subsequent report, successful coitus was achieved by 72% of 250 patients of whom 97 patients were on autoinjection.[28] Sidi and associates[29] also reported the results of autoinjection in 100 patients. They used a trial dosage of a solution containing 25 mg/ml of papaverine and 0.8 mg/ml of phentolamine, adjusted thereafter according to the patient's erectile response. They found that patients with neurogenic impotence required a much lower dose of the combined agent (0.1–1.0 ml) than patients with vasculogenic impotence (0.5–1.5 ml). Because injection of vasoactive agents bypasses psychological, neurological, and hormonal factors to act directly on the penile tissue and vessels, a patient with an intact vascular system and healthy penile tissue will respond much more readily than will a patient with compromised vasculature. In addition, erotic stimuli can further enhance erection by release of neurotransmitters. Therefore, the dose required to achieve erection is usually smaller in autoinjection (immediately before sexual intercourse) than the dose required in office diagnostic tests (without erotic stimuli).

Complications

Immediate complications include ecchymosis, pain, transient paresthesia, facial flushing, dizziness, hypotension, vasovagal syncope, and priapism. Delayed complications that have been reported are infection (cavernositis; T. Takamura, personal communication); gangrene of the glans (S. Dunn, personal communication); and hematoma, ecchymosis, and elevated liver enzymes (I. Goldstein, personal communication). One death from pulmonary embolism after treatment for papaverine-induced priapism was reported recently.[30] After several months to 3 years of usage, palpable localized fibrosis inside the corpora cavernosa or at the tunica albuginea and angulation of the penis have been noted.[31] Experimental studies in monkeys after 100 papaverine injections revealed no change of liver enzymes, some fibrosis near the injection site, and smooth muscle hypertrophy of unknown significance.[32]

The ease of administration, high success rate, and relative lack of major complications have resulted in increased use of intracavernous injection therapy. Careful patient selection, regulation of dosage, meticulous attention to technical detail, and knowledge of the prevention and treatment of complications are essential to success.

Prevention and Treatment of Complications

The best treatment is prevention. Before injection, patients should be informed of the possible complications and side effects. To prevent ecchymosis, we routinely compress the needle puncture site with digital pressure for 2–3 min. In patients taking aspirin or anticoagulants, we recommend compression for at least 5 min. It is difficult to predict which patients will develop dizziness or hypotension. However, in those with a history of postural hypotension or in whom rapid venous runoff is suspected, a lower dosage, should be tried at the first test. If full erection lasts for more than 1 hour during testing, we always terminate it by aspiration or injection of α-adrenergic agents. For autoinjection, we give the patient detailed written instructions with the background and rationale of the treatment, alternatives, possible side effects and complications, and instructions for action in case of an adverse reaction. Patients must contact the physician if the erection lasts for more than 4 hours. The initial dose in neurogenic and psychogenic impotence is 0.2 to 0.3 ml with 0.1-ml increments; in vasculogenic impotence, it is 0.5 ml with 0.25-ml increments. Phentolamine is added only if papaverine alone does not induce adequate erection. The injection frequency must not exceed twice a week. No more injections should be given if the patient feels a lump in the penis. The patient should be examined by the physician every 2–3 months for possible fibrosis of the corpora cavernosa and to obtain blood for liver function testing. With this careful approach, only 2 of 900 patients who have undergone papaverine testing develops priapism, and no patients in our autoinjection group have done so.

We recommeend interruption of drug-induced priapism within 4–6 hours to prevent ischemia and tissue damage. Priapism of less than 24 hours' duration usually responds well either to aspiration or to injection of α-adrenergic agents. In patients with a history of cardiovascular disease, cerebrovascular accident, or poorly controlled hypertension, repeated aspiration is the treatment of choice because α-adrenergic agent may induce unnecessary complications. In patients with neurogenic or psychogenic impotence and none of the risk factors mentioned above, injection of α-adrenergics can be used to achieve rapid detumescence. Those reported include metaraminol (1 mg),[27] dopamine, phenylephrine (10 mg/500 ml saline irrigation),[29] ephedrine (50–100 mg/1–2 ml), norepinephrine (10–20 μg) and epinephrine (10–20 μg).[14] Most often we use direct injection of ephedrine 50 mg/ml or prepare a solution of 10 μg of epinephrine per milliliter of saline (mix 0.1 ml [0.1 mg] of epinephrine with 10 ml of normal saline) and repeat

the injection every 5 min until detumescence takes place. It is important not to continue the injections after detumescence begins because the α-adrenergic agent may enter the systemic circulation rapidly through the newly opened venous channels and cause severe hypertension and tachycardia.

ORAL MEDICATION

Yohimbine, an α-adrenergic blocking agent obtained from the bark of Rubaceae and related trees, has been considered an aphrodisiac for many years. Recent animal experiments by Clark and associates[33] found that it increased arousal in sexually experienced male rats, facilitated copulatory behavior in sexually naive males, and induced sexual activity in previously inactive males.

Although many earlier reports suggested that yohimbine may be effective in treating impotence, these studies lacked a placebo control and objective response criteria. Furthermore, other drugs, such as androgens, were often combined with yohimbine, making interpretation difficult. Recent interest in yohimbine was regenerated after Morales and associates[31] reported that it improved erection in 43% of patients with organic impotence. MacFarlane and associates [35] also reported resumption of normal sexual function in 6 of 20 patients and improvement in 4.

The physiologic basis and site of action of yohimbine are far from clear. The human corpus cavernosum contains a rich amount of α-adrenergic receptors. The induction of erection or detumescence results from the blockade or activation, respectively, of α_1-postsynaptic receptors. Agents that block α_2-receptors, such as yohimbine, release norepinephrine at the postsynaptic site, which should be vasoconstrictive. In recent studies (unpublished data), we injected yohimbine into the canine corpus cavernosum and found that it detumesced in a manner similar to epinephrine. Therefore, it seems more likely that the site of action of yohimbine is in the central nervous system to improve the libido rather than at the penile level.

The recommended dose of yohimbine is 5 mg per os three times a day. The advantage of oral administration and the relatively few side effects (nervousness, elevated blood pressure, and dizziness), have led some to advocate a trial of yohimbine in many impotent patients. Further studies are needed to determine its effect on human penile erection.

PENILE REVASCULARIZATION

Leriche[36] first reported the association between aortoiliac arterial disease and impotence. Since then, many procedures have been attempted to improve the blood supply to the penis in patients with vasculogenic impotence. Reconstructive surgery aimed at restoring the arterial supply to the lower extremities has been reported to improve potency in up to 50% of patients.[37] Transluminal angioplasty has also been tried.[38] In general, patients with isolated stenosis or occlusion of the proximal (iliac or main pudendal) arteries have a much better result after these procedures then those with multiple or terminal penile arterial disease.

Michal and assciates[39] first described an end-to-side anastomosis of the inferior epigastric artery to the corpus cavernosum in an attempt to increase arterial flow to the corpora in patients with distal penile arterial disease. Priapism resulted in some patients and the long-term patency of the rate graft was poor. Hawatmeh and associates[40] reported a 100% failure rate with femoral or epigastric arterial bypass to the corpora cavernosa and cavernosal fibrosis in biopsy specimens. Later, Michal's group[41] proposed a modified approach of end-to-side anastomosis of the epigastric artery to the dorsal penile artery. The graft occlusion rate was still unaccptably high.

Improved results have been reported with microvascular anastomosis to the cavernosal artery; this technique was first described by Crespo and associates[42] (80% success in 174 cases) and later by Mac-

Gregor and Konnak.[43] However, because of the technical difficulty and the small distal runoff, other investigators have been unable to reproduce this good response.

A procedure of anastomosing the epigastric artery to the deep dorsal vein to restore corporal blood flow was reported by Virag[44] to have an 85% initial success in 22 patients. Bennett and associates[45] also reported a successful result in four patients followed for 6–18 months. Although graft thrombosis also occurs, Virag reported a 20% thrombosis rate at 3 years, much less than that with earlier procedures.

In a large series of more than 100 patients who underwent end-to-end anastomosis of the epigastric artery to the proximal dorsal artery, Goldstein[46] reported more than 80% success in patients with erectile impotence secondary to trauma and 30% success in patients with diffuse atherosclerotic vascular disease.

Revascularization awaits further improvement. At present, it is safe to say that patients with isolated proximal arterial disease and patients with traumatic arterial occlusion are the best candidates for these procedures.

TREATMENT OF VENOGENIC IMPOTENCE

Erectile dysfunction resulting from excessive venous drainage from the corpora cavernosa has become a topic of great interest in urology. Abnormal and excessive venous drainage has been found to occur through various pathways: the superficial dorsal, deep dorsal, cavernous and crural veins, and the corpus spongiosum or glans penis. Treatment varies among investigators, but it generally consists of ligation of all circumflex and emissary veins as they join the dorsal vein and excision of the dorsal vein from just proximal to the glans to the level of the suspensory ligament. A new approach to identify and ligate the cavernous and crural veins has also been introduced to treat patients with proximal leakage (unpublished data).

The intial results of venous surgery are encouraging. Ebbehof and Wagner[47] treated three of four patients successfully in 1979. Wespes and Schulman[48] reported return of good erections in 16 of 20 patients. Bennett and associates[45] had successful results in six of eight patients. A recent report by Lewis and Puyau[49] showed 50–75% improvement in their 38 patients.

Some major problems remain in diagnosing and treating patients with excessive venous drainage. At the present time we do not have a reliable noninvasive technique for diagnosing cavernosal fibrosis and cavernous smooth muscle disease, nor can we reliably test the cavernous nerve. Understandably, ligation of venous channels will not help these patients, and their inadvertent inclusion in published series may account for the relatively large percentage of failures reported by different authors.

PENILE PROSTHESES

The penile prosthesis remains the most reliable and most widely used option in the treatment of the impotent patient. The introduction of the Small-Carrion[50] semirigid intracavernous prosthesis and the Scott inflatable prosthesis[51] ushered in the modern era of penile prosthetics in the mid-1970s. Since then, the semirigid prosthesis has been modified to improve concealment and many modifications of the inflatable devices have been made to reduce the rate of mechanical failure.

The most recent advance in penile prostheses is the introduction of the "third generation" devices. The Flexi-Flate and Hydroflex have inflatable cylinders and a self-contained pump and fluid storage system. The OmniPhase uses an ingenious mechanical cable system to achieve rigidity.

The Flexi-Flate (Surgitek) device consists of paired hydraulic cylinders filled with saline, each with its own self-contained pump, reservoir, and relief system. Each cylinder has an expandable, outer silicone sheath and an inner nonexpandable cylinder. A distal pump behind the glans transfers fluid through a one-way valve from the

outer sheath to the inner cylinder, resulting in erection. A pressure-sensitive relief valve transfers the fluid back to the sheath to return the prosthesis to the flaccid state when the device is bent forcefully. The device has a trimmable tail and is available in 11- and 13-mm diameters and in lengths from 10 to 15 cm.[52] Long-term follow-up is not available, but the short-term failure rate has been less than 9.5% (data provided by Surgitek). Some early patients complained of inadvertent deflation during intercourse, which prompted the company to increase the deflation threshold from 500–700 to 1300–1500 cmH_2O. No follow-up data are available since the modification.

The Hydroflex (American Medical Systems) device consists of a pair of prefilled hydraulic cylinders, each with a self-contained rear reservoir, fluid passageway, inflation pump, deflation valve, and a reinforced silicone inflation chamber. Squeezing the inflation pump located underneath the glans transfers fluid from the rear reservoir to the inflation chamber, resulting in erection. Activating the deflation valve located just proximal to the inflation pump results in flaccidity. Inventory consists of two diameters (11 and 13 mm) and four lengths (13, 16, 19, and 22 cm, with 1- and 2-cm rear tip extenders). Clinical results are limited due to its recent introduction. A 99% mechanical success in the first 500 patients has been reported by the company. In a series of 35 patients followed for an average of 11.2 months, Fishman[53] reported that 30 were satisfied with the appearance and function of the prosthesis. However, in patients who had previously had an inflatable prosthesis, the general consensus was that the quality of erection with the Hydroflex was inferior both in length and girth. Several patients complained about the inability to attain a completely flacid penis on complete deflation of the device.

The major advantage of the Flexi-Flate and Hydroflex is the ease of insertion, which can be done under local anesthesia with a single penoscrotal incision.

The OmniPhase (Dacomed) device contains a mechanical cable system consisting of a flexible activator body encased in polytetrafluoroethylene with a silicone coating. Within the activator body sheath is a column of radial polysulfone segments through which a cable under spring pressure is passed. The proximal end of the cable is attached to an activating switch that shortens and lengthens the cable. When the switch is activated, the cable and spring shorten, pulling the segments together, resulting in rigidity. Deactivation lengthens the cable and disengages the segments, resulting in flaccidity. Bending the penis at the base results in alternating rigidity and flaccidity. Each activator body is 13 cm long and is available in 10- and 12-mm diameters. The remaining length is made up of proximal and distal tips of 1 to 8 cm. In a preliminary report of 36 patients who received the prototype, Krane[54] reported that three devices had to be removed for medical reasons, the activator malfunctioned in five, and the cable fractured in seven. The device has since been modified. Of 50 subsequent implants, all were functioning well but for four devices that were removed for medical reasons.

OTHER FORMS OF THERAPY

Other forms of therapy include the vacuum-constriction devices. The principle of these devices is simple: A cylinder is placed over the entire penis, and a vacuum is created by suction to induce tumescence; a constricting band is then placed around the base of the penis to maintain tumescence when the vacuum is released. Several devices are now available. The vacuum-constriction device reportedly provides sufficient penile rigidity in more than 90% of patients. The other device, the ErecAid System, is similar in design. Satisfaction in more than 300 patients is greater than 90% (data provided by Nu-Potent, Inc.).

The advantages of this device are its low cost and noninvasiveness. The major disadvantages are the inconvenience and potential embarrassment of using the vacuum and constricting band. Reported complications include petechiae in up to one-half of patients and ecchymosis, sometimes accompanied by pain and swelling, in up to one-third.[55]

References

1. Wagner G: Erection physiology and endocrinology. In Wagner G and Green R (eds): *Impotence: Physiological, Psychological, and Surgical Diagnosis and Treatment.* New York, Plenum Press, 1981, pp 25–36.
2. Lue TF, Takamura T, Schmidt RA, Palubinshos AJ, Tanagho EA: Hemodynamics of erection in the monkey. *J Urol 130:* 1237–1241, 1983.
3. Lue TF, Takamura T, Umraiya M, Schmidt RA: Hemodynamics of canine corpora cavernosa during erection. *Urology 24:* 347–352, 1984.
4. Padma-Nathan H, Goldstein I, Azadzoi K, et al: In vivo and in vitro studies on the physiology of penile erection. *Semin Urol 4:* 209–216, 1986.
5. Fournier GR, Jr, Juenemann KP, Lue TF, Tanagho EA: Mechanism of venous occlusion during canine penile erection: an anatomic demonstration. *J Urol 137:* 163–167, 1987.
6. Walsh PC, Donker PJ: Impotence following radical prostatectomy: insight into etiology and prevention. *J Urol 128:* 492–497, 1982.
7. Lue TF, Zeineh SJ, Schmidt RA, Tanagho EA: Neuroanatomy of penile erection: Its relevance to iatrogenic impotence. *J Urol 131:* 273–280, 1984.
8. Lepor H, Gregerman M, Crosby R, Mostofi FR, Walsh PC: Precise localization of the autonomic nerves from the pelvic plexus to the corpora cavernosa: a detailed anatomical study of the adult male pelvis. *J Urol 133:* 207–212, 1985.
9. Hedlund H, Andersson KE: Comparison of the responses to adrenoceptor and muscaninic receptor active drugs in isolated human corpus cavernosum and cavernous artery. *J Auton Pharmacol 5:* 81–88, 1985.
10. Willis E, Ottsen B, Wagner G, Sundler F, Fahrenkrug J: Vasoactive intestinal polypeptide (VIP) as a possible neurotransmitter involved in penile erection. *Acta Physiol Scand 113:* 545–547, 1981.
11. Saenz de Tejada I, Cohen RA: Human corpus cavernosum smooth muscle relaxation to acetylcholine is an endothelium mediated response. *J Urol 137:* 184A, 1987.
12. Saenz de Tejada I, Blanco R, Goldstein I, Krane RJ, Cohen RA: Cholinergic neurotransmission in human penile corpus cavernosum smooth muscle. *Federation Proc 44(1):* 454, 1985.
13. Adaiken PG, Kottegoda SR, Ratnam SS: Is vasoactive intestinal polypeptide the principal transmitter involved in human penile erection? *J Urol 135:* 638–640, 1986.
14. Lue TF, Tanagho EA: Physiology of erection and pharmacologic management of impotence. *J Urol 137:* 829–836, 1987.
15. Juenemann KP, Lue TF, Fournier GR, et al: Hemodynamics of papaverine and phentolamine-induced penile erection. *J Urol 136:* 158–161, 1986.
16. Virag R, Frydman D, Legman M, Virag H: Intracavernous injection of papaverine as a diagnostic and therapeutic method in erectile failure. *Angiology 35:* 79–87, 1984.
17. Burat J, Levnaire A, Dehaere JL, et al: Venous incompetence: Critical study of the organic basis of high maintenance flow rates during artificial erection test. *J Urol 135:* 926–928, 1986.
18. Lue TF, Hricak H, Marich KW, Tanagho EA: Vasculogenic impotence evaluated by high-resolution ultrasonography and pulsed Doppler spectrum analysis. *Radiology 155:* 777–781, 1985.
19. Zorgniotti AW, Lefleur RS: Auto-injection of the corpus cavernosum with a vasoactive drug combination for vasculogenic impotence. *J Urol 133:* 39–41, 1985.
20. Wagner G: Differential diagnosis of erectile failure. In Wagner G, Green R (eds): *Impotence: Physiological, Psychological, Surgical Diagnosis and Treatment.* New York, Plenum Press, 1981, chap 8, pp 89–130.
21. Virag R: Arterial and venous hemodynamics in male impotence. In Bennett AH (ed): *Management of Male Impotence,* Baltimore, Williams & Wilkins, 1982, pp 108–126.
22. Wespes E, Delcour C, Struyven J, Schulman CC: Cavernosometry-cavernosography: its role in organic impotence. *Eur Urol 10:* 229–232, 1984.
23. Wespes E, Schulman CC: Parameters of erection. *Br J Urol 56:* 416–417, 1984.
24. Lue TF, Hricak H, Schmidt RA, Tanagho EA: Functional evaluation of penile veins by cavernosography in papaverine-induced erection. *J Urol 135:* 479–482, 1986.
25. Wespes E, Delcour C, Struyven J et al: Pharmaco cavernometry-cavernography in impotence. *Br J Urol 58:* 429–433, 1986.
26. Virag R: Intracavernous injection of papaverine for erectile failure. *Lancet 2:* 938, 1982.
27. Brindley GS: Cavernosal α-blockade: a new technique for investigating and treating erectile impotence. *Br J Psych 143:* 332–337, 1983.
28. Zorgniotti A: Corpus cavernosum blockade for impotence: practical aspects and results in 250 cases. *J Urol (part 2) 135:* 306A, 1986.
29. Sidi AA, Cameron JS, Duffy LM et al: Intracavernous drug-induced erection in the management of male erectile dysfunction: experience with 100 patients. *J Urol 135:* 704–706, 1986.
30. Hashmat A, Abraham J: Papaverine-induced priapism: a lethal complication. *J Urol 137:* 201A, 1987.
31. Sidi AA, Duffy LM, Wasserman N: Short- and long-term complications of vasoactive intracavernous pharmacotherapy (VIP) for impotence. *J Urol 137:* 203A, 1987.
32. Abozeid M, Juenemann KP, Luo JT et al: Chronic papaverine treatment: the effect of repeated injections on the simian erectile response and penile tissue. *J Urol,* in press.
33. Clark JT, Smith ER, Davidson JM: Enhancement of sexual motivation in male rats by yohimbine. *Science 225:* 847, 1984.
34. Morales A, Surridge DHC, Marshall PG et al: Nonhormonal pharmacological treatment of organic impotence. *J Urol 128:* 45–47, 1982.
35. MacFarlane CA, Reynolds WJ, Rosencrantz DR: Yohimbine an alternative treatment for erectile dysfunction. *Weekly Urol Clin Letter* 27(27), 1983.