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- Clinics, and Hospitals

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- H&Ps
- Preoperative notes
- Consults
- Discharge summaries
- ED reports

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- Dermatology
- ENT
- Endocrinology
- Gastroenterology
- Gynecology
- Immunology
- Neurology
- Obstetrics
- Oncology
- Ophthalmology
- Orthopedics
- Pediatrics
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- Pulmonology
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- Trauma
- Urology

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- Genitourinary Surgery
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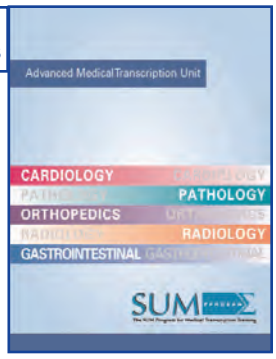

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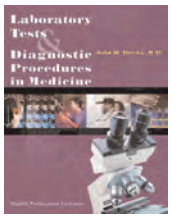

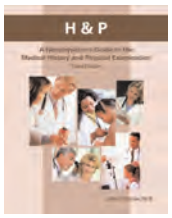

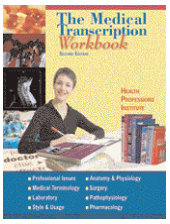

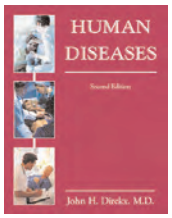

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Guy Talk: Perspectives on Selected Topics in Andriatrics

by John H. Dirckx, M.D.

Even though you may never have seen or heard the word before, you've probably already figured out that andriatrics is a branch of medicine concerned with men's health, a sort of male complement to the more familiar specialty of gynecology. As far as I know, there are no residency programs in andriatrics, and there is certainly no specialty board in the field. But a growing number of U.S. physicians specialize in health concerns unique to men, and some limit their practices to the treatment of gay men.

Urology is a surgical specialty that focuses on the diagnosis and treatment of disorders of the urinary tract. Although more than half of most urologists' patients are women, the urologist has traditionally treated disorders of the male reproductive system. This article discusses some common andriatric/urologic problems.

First, a brief review of male reproductive anatomy. The testicles produce both sperm (reproductive cells capable of fertilizing a female oocyte) and testosterone (a hormone that initiates and maintains secondary sexual characteristics such as male physique, facial and body hair growth, and deeper voice). Each testicle is suspended within the scrotum by a spermatic cord, a complex structure containing smooth muscle, connective tissue, blood vessels, nerves, lymphatics, and the spermatic duct (vas deferens), which conducts sperm to the prostate gland.

The male urethra runs from the bladder to the tip of the penis to carry urine outside the body and also serves as a channel for the ejaculation of semen. The prostate—the word is frequently mispronounced "prostrate" by the laity—is a structure about the size of a walnut that surrounds the urethra just below the neck of the bladder. Both the prostate and the seminal vesicles (small sac-like glands adjacent to the spermatic duct) produce a fluid secretion that contributes to the composition of semen. Smooth muscle fibers in the prostate contract at the time of ejaculation, preventing retrograde flow of semen into the bladder.

Disorders of the prostate (benign hyperplasia and adenocarcinoma) account for a high proportion of visits to urologists by men over 50. **Benign prostatic hyperplasia** (BPH) is an overgrowth of androgen-sensitive glandular tissue that normally accompanies aging. The disorder may remain asymptomatic for years, but most elderly men eventually experience both irritative and mechanical effects. Irritative symptoms result from a heightened sensitivity in the neck of the bladder and prostatic urethra, and include pollakiuria (increased urinary frequency), urgency, nocturia (the need to get up one or more times at night to urinate), and burning or stinging on urination.

Mechanical symptoms arise from distortion of urethral anatomy by prostatic swelling. Compression of the urethra causes hesitancy (difficulty in starting urination), reduction in the force and volume of the urinary stream, inadequate emptying of the bladder, and difficulty in stopping urination (post-voiding dribbling). Recurrent failure to empty the bladder completely can lead to chronic distention and overflow incontinence. Occasionally the presenting symptom of BPH is acute urinary retention, requiring prompt catheterization to relieve distress and prevent complications such as hydronephrosis (filling of the ureters and renal pelves with urine under pressure) and urinary tract infection.

The diagnosis of BPH is based on history, palpation of the gland by digital rectal examination, and the performance of various procedures to rule out cancer (discussed below). Benign enlargement of the prostate is typically symmetrical (not nodular), and the gland feels firm but not stony hard. The examining finger can reach only the posterior lobe of the prostate and most of the right and left lateral lobes. Enlargement of these parts of the gland doesn't necessarily correlate with the degree to which urine flow is compromised by the anterior and median lobes.

Catheterization of the bladder immediately after voiding may yield a substantial volume of retained urine ("post-voiding residual"). Other diagnostic procedures sometimes used include endoscopy (examination of the prostatic urethra with a urethroscope or cystoscope) and voiding cystourethrography (VCUG), a radiographic study of the bladder and urethra during voiding after injection of contrast medium through a catheter.

The symptoms of benign prostatic hyperplasia often respond to oral medicines. Two major classes of prescription drug, 5α-reductase (5-alpha-reductase) inhibitors and α-1 (alpha-1) adrenergic blockers, are currently approved for this indication.

In order to exert its physiologic effects, testosterone must be converted to its active form, dihydrotestosterone, by the enzyme 5α-reductase. Drugs that inhibit this enzyme reduce

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the formation of dihydrotestosterone and thus reverse prostatic hyperplasia, which is hormone-dependent. The two 5α-reductase inhibitors currently approved are dutasteride (Avodart) and finasteride (Proscar). (Finasteride is also marketed under the trade name Propecia for the treatment of male-pattern baldness, which is also dependent on dihydrotestosterone.)

The 5α-reductase inhibitors reduce gland bulk very slowly, and weeks or months of treatment may be needed before results are seen. Possible side effects of these agents are diminished libido, erectile dysfunction, and breast swelling and tenderness. Finasteride and dutasteride can also delay the diagnosis of prostatic carcinoma by lowering the serum level of prostate specific antigen (PSA), to be discussed below.

Alpha-1 adrenergic blockers are the other class of prescription drug currently used to treat BPH: alfusozin (Uroxatral), doxazosin (Cardura), prazosin (Minipress), tamsulozin (Flomax), and terazosin (Hytrin). These agents improve urinary flow by relaxing smooth muscle in the prostate gland. Beneficial effects are often noted within a day or two of starting treatment, but because alpha-1 blockers don't reduce the bulk of the prostate, they may provide little relief for men with very large glands.

Alpha-1 blockers were originally developed to treat hypertension. Doxazosin, prazosin, and terazosin are among drugs in this class that are currently approved for lowering blood pressure. Dizziness, light-headedness, and orthostatic hypotension (a symptomatic drop in blood pressure on standing up from a sitting or reclining position) are experienced fairly often by men taking alpha-1 blockers for prostatic hyperplasia.

Combination therapy with one drug from each class is routinely prescribed by some physicians.

Saw palmetto (*Serenoa repens*) is a small New World palm whose leaf stems (petioles) are edged with sharp spines, hence it name. Extracts of its edible fruit have been used for centuries in folk medicine for a variety of indications, including urologic disorders. In controlled clinical trials, saw palmetto has performed better than placebo in improving urine flow in BPH and has matched the effects of prescription medicines in mild disease. It has also yielded promising results in male-pattern baldness. Limited pharmacologic studies suggest that it has both alpha-1 blocking and alpha-reductase inhibiting properties.

Saw palmetto extract, being a natural product, ranks as a nutraceutical and is thus essentially exempt from oversight by the United States Food and Drug Administration (FDA). It has not been approved by the FDA for any indication, and standards of purity have not been established for it. Available formulations may therefore vary widely in both efficacy and safety.

A generation ago, symptomatic prostatic hyperplasia was routinely managed by various surgical procedures, including total excision of the gland. More recently, the availability of effective oral medicines has greatly reduced the frequency of surgery for this indication. Surgery is still considered appropriate for men with severe disease refractory to drug treatment, those for whom drugs are contraindicated, and those with certain complications (recurrent acute obstruction, frequent urinary tract infections, hydronephrosis, bladder stones, hematuria).

For selected patients, **prostatectomy** (removal of the entire gland) is considered the procedure of choice. The surgeon may use either a perineal or a retropubic approach to the gland. In the latter technique, an incision is made in the lower abdomen and the bladder is dissected free from the anterior abdominal wall. Healing time after either procedure is prolonged, and postoperative complications such as incontinence and sexual dysfunction are relatively common.

For many years the standard surgical technique for BPH has been **transurethral resection of the prostate** (TUR, TURP). Under spinal or general anesthesia, a modified endoscope called a resectoscope is inserted through the penis and advanced to the level of the hyperplastic prostate. The surgeon then shaves away surplus tissue encroaching on the lumen of the urethra by means of an electrical loop, which also seals severed blood vessels. The instrument is equipped with an irrigating system that flushes away blood and tissue.

Obviously this technique also removes the mucosal lining of the prostatic urethra, which takes weeks to heal completely. An irrigating catheter remains in the bladder for about 72 hours after surgery. Mild to severe stress incontinence (leakage of urine with coughing, laughing, or straining), enuresis (bedwetting), and hematuria, often with passage of clots, may persist for several days.

About one third of patients experience some degree of sexual dysfunction after TURP, at least temporarily. Retrograde ejaculation (passage of semen into the bladder during orgasm) results from replacement of prostatic periurethral smooth muscle by scar tissue and is usually permanent. Because TURP removes only part of the gland, it is not as definitive a treatment as prostatectomy. Obstructive symptoms can recur after TURP and may require periodic repetitions of the procedure.

Several modifications of TURP have been devised to reduce the incidence and severity of complications and to shorten healing time. In **transurethral incision of the prostate** (TUIP), longitudinal incisions are made in the prostatic urethra without removal of any tissue. The risk of retrograde ejaculation is less after this type of surgery, but long-term control of symptoms has not been demonstrated.

Holmium laser enucleation of the prostate (HoLEP) uses a holmium laser to resect hyperplastic tissue instead of

the electrical loop of traditional TURP. In **holmium laser ablation of the prostate** (HoLAP), surplus tissue is vaporized rather than trimmed away. **Transurethral ultrasound-guided laser incision of the prostate** (TULIP) resembles TUIP, but the incisions are made with a laser. One of the latter two techniques may suffice when the total volume of the prostate is relatively small. Bleeding during and after surgery is less with laser procedures, which can usually be performed as outpatient surgery.

Still less damaging methods use lasers or microwave radiation to coagulate excessive prostatic tissue. These are brief office procedures with few adverse effects or complications, but long-term outcomes may not match those of more aggressive measures.

The most conservative and least invasive procedure for BPH is placement of a metal **stent** within the prostatic urethra through an endoscope under regional anesthesia. This procedure can be performed in patients with medical conditions that forbid more elaborate surgery. However, adverse effects (dysuria, pollakiuria, incontinence) often occur. The removal of a prostatic stent, which may be deemed necessary in as many as one third of patients, often proves more difficult and invasive than its insertion.

Adenocarcinoma of the prostate is the most common cancer in men and the second most common cause of cancer deaths in men (after lung cancer). A carcinoma is a malignant tumor arising from epithelial tissue; an adenocarcinoma arises from glandular epithelium. Like BPH, prostate cancer represents an overgrowth of hormone-sensitive secretory cells in the prostate. There the similarities end. BPH is by definition benign, while prostatic carcinoma is malignant. BPH does not evolve or degenerate into cancer.

Prostate cancer is more common, occurs at an earlier age, and spreads more aggressively in African-American men. Because malignant changes usually begin near the periphery of the gland, urinary symptoms occur late, if at all. In more than one third of patients, cancer has spread beyond the gland by the time the diagnosis is made. Prostate cancer can invade the bladder, rectum, and other pelvic structures by direct extension and can spread to more remote sites by metastasis. The bones of the spine and pelvis are the most frequent sites of metastasis.

Nowadays, the diagnosis is usually made when the screening of an apparently healthy man by means of digital rectal examination (DRE) or determination of the serum level of prostate specific antigen (PSA) yields abnormal results.

An asymmetrically enlarged or nodular prostate, or one that feels abnormally hard to the examining finger, suggests the presence of malignancy. Although rectal palpation of the prostate is easily performed, requires no special preparation, and causes little discomfort, the procedure has low sensitivity (below 25% in some studies) and even lower specificity (below 10% in some studies).

Prostate specific antigen, an enzyme that helps to maintain the fluidity of semen, is produced by the secretory epithelium of the prostate. (Trace amounts occur in other tissues, including the endometrium and the female breast.) A level below 2 ng/mL (2 mcg/L) appears in the blood of normal men. Elevation of the serum concentration of PSA is highly organ-specific, reliably drawing attention to the prostate, but is not at all disease-specific.

Statistically, most elevations above 4 ng/mL are due to prostatitis, not cancer. Other causes of elevation include benign prostatic hyperplasia, prostatic infarction, recent ejaculation, and even digital examination of the prostate. As many as one third of elevated PSA levels return to normal on followup testing without treatment. Although a level above 9 ng/mL (9 mcg/L) strongly suggests cancer, the PSA level is normal in more than 10% of men with biopsy-proven cancers.

Despite these limitations, the test was hailed as a more sensitive and more specific means of detecting early cancer than digital rectal examination when it was first approved by the FDA in 1992. Both the American Cancer Society and the American Urological Association recommended routine annual PSA screening as well as digital rectal examination for men over 50 (over 40 for African-American men or those with a family history of prostate cancer). After a brief show of resistance, Medicare began covering the cost of one PSA screening per year, and continues to do so. By the end of the twentieth century, PSA screening had become part of the standard of care for men over 50.

But almost from the first, the routine use of PSA testing in asymptomatic men has been the subject of intense controversy. A remarkable increase in the reported incidence of

Urology Abbreviations

α-1 (alpha-1) adrenergic blockers
BPH (benign prostatic hyperplasia)
cGMP (cyclic guanosine monophosphate)
DRE (digital rectal examination)
ED (erectile dysfunction)
eNOS (endothelial nitric oxide synthase)
5α-reductase (5-alpha-reductase) inhibitors
HoLAP (holmium laser ablation of the prostate)
HoLEP (holmium laser enucleation of the prostate)
nNOS (neuronal nitric oxide synthase)
NO (nitric oxide)
PDE-5 (phosphodiesterase-5) inhibitors
PSA (prostate specific antigen)
  age-adjusted PSA
  PSA density
  PSA velocity
PVN (paraventricular nucleus)
TRUS (transrectal ultrasound)
TUIP (transurethral incision of the prostate)
TULIP (transurethral ultrasound-guided laser incision of the prostate)
TUR, TURP (transurethral resection of the prostate)
VCUG (voiding cystourethrography)

Perhaps of greatest importance, to date no study has shown conclusively that early screening, detection, and treatment of prostatic carcinoma reduce mortality. The American College of Preventive Medicine has concluded that there is insufficient evidence to recommend routine screening with either DRE or PSA in men of any age.

prostatic carcinoma during the 1990s seemed to confirm the value of PSA screening. But it quickly became evident that the test was yielding an unacceptably high level of false positive and false negative results. A false positive test for prostate cancer causes needless anguish and expense and often leads to unnecessary treatment. Conversely, a false negative result from this highly touted procedure can generate an erroneous conviction that one is free of cancer and delay diagnosis until the disease has reached an advanced stage.

The lack of both sensitivity and specificity for prostate cancer is an inherent and irremediable deficiency of PSA screening. Lowering the cutoff level between normal and abnormal PSA increases the chance of detecting cancer at the expense of increasing the rate of false-positive results.

Efforts to enhance the value of the test by modifying the procedure or its interpretation have included determination of PSA velocity (rate of change in PSA level with the passage of time), age-adjusted PSA (using higher cut-off levels for older men), PSA density (the ratio of PSA level to the size of the prostate), and the ratio of free to protein-bound PSA (lower in malignant disease). None of these has shown clear-cut benefits in large statistical studies.

Many prostate cancers grow very slowly and never cause symptoms. Indeed, foci of cancer are found in the prostates of 40% of men dying after age 50 of other causes. PSA screening, by detecting many small cancers that would never become life-threatening, subjects some patients to basically futile treatment that may have adverse effects such as urinary incontinence and erectile dysfunction.

The 10-year survival rate of diagnosed prostatic carcinoma is about 90%. For that reason, virtually all authorities now oppose routine periodic digital rectal examination and PSA screening of asymptomatic men with life expectancies of less than 10 years, on the grounds that the risks of false-positive results and of adverse consequences of aggressive treatment outweigh any possible benefit in survival or quality of life.

Perhaps of greatest importance, to date no study *has shown conclusively that early screening, detection, and treatment of prostatic carcinoma reduce mortality*. The American College of Preventive Medicine has concluded that there is insufficient evidence to recommend routine screening with either DRE or PSA in men of any age. Both the U.S. Preventive Services Task Force and the National Cancer Institute advise against routine PSA testing.

Routine screening for prostate cancer can nonetheless be expected to continue indefinitely. Patients' expectations (driven by media hype and misguided "wellness" programs), practitioners' fear of legal liability for missing a cancer by omitting the test, and the profit motive (never far below the surface in the healthcare "industry") will no doubt keep DRE and PSA screening an active part of medical practice until a more reliable method takes their place.

Suspicion of prostatic carcinoma, whether based on symptoms or on abnormal results of screening, is followed up by biopsy of the gland. **Prostatic biopsy** is usually performed in conjunction with a transrectal ultrasound (TRUS) examination to assess the size and configuration of the gland. Under ultrasonic guidance, the examiner secures specimens from 10 or more sites with a spring-loaded biopsy needle that enters the gland and extracts a core of tissue in a fraction of a second.

Routine preparation for this procedure includes a cleansing enema, increased fluid intake to distend the bladder, prophylactic antibiotics, and local anesthesia. Alternative methods, less often used, obtain tissue through a a perineal incision or through an endoscope inserted into the urethra. Adverse effects include pain, infection, and bleeding in urine, semen, or stool.

If sampled tissue is cancerous, microscopic examination shows cellular changes characteristic of malignancy, including varying degrees of anaplasia—a lack of the cellular differentiation that is typical of normal prostate tissue. Anaplasia is graded on a scale of 1 (nearly normal glandular differentiation) to 5 (total lack of differentiation).

The Gleason score (developed about 40 years ago by the American pathologist Donald F. Gleason) has been found useful in converting biopsy findings into prognostic information and in planning treatment. This score is determined by simply adding the grades of the two least differentiated specimens. A Gleason score of 2 or 3 is associated with a relatively favorable prognosis, a score of 9 or 10 with a poor prognosis.

The secretory cells of the prostate are highly sensitive to hormonal stimulation by testosterone. Neither BPH nor adenocarcinoma of the prostate occurs in eunuchs (men without testicles). Measures that reduce or block androgenic stimulation of cancer cells can slow the progression of the primary tumor and suppress growth of metastases. Antiandrogen therapy is often indicated in both early and advanced disease.

The most drastic and definitive means of stopping hormonal stimulation of prostate cancer is bilateral orchiectomy (castration, removal of both testicles). Pharmacologic agents often used are the androgen receptor blocker flutamide (Eulexin, Flutamin) and the gonadotropin-releasing hormone (GnRH) agonist leuprolide (Eligard, Lupron, Viadur).

Surgical procedures for advanced or highly malignant prostatic carcinoma include radical prostatectomy, which is associated with a considerable risk of urinary incontinence and erectile dysfunction, external x-ray or proton beam radiation, or transperineal implantation of radioactive isotopes.

If any topics remain taboo these days in the news and entertainment media and in polite conversation, erectile dysfunction (ED) isn't one of them. Public figures discuss it frankly on national television, and pharmaceutical companies hawk their remedies for it with increasingly salacious TV commercials.

The older and less explicit term, impotence, was borrowed by medicine from the language of the law, where it refers to the inability of a person of either sex to have sexual intercourse. In the legal setting, impotence refers principally to an anatomic deficiency—in its most extreme form, absence of the penis or of the vagina—but also includes physiologic and psychological incapacity. Impotence has been recognized in both civil and canon law as an impediment to marriage and as grounds for the annulment of a marriage that has already taken place.

Erectile dysfunction is defined as the inability of a sexually mature male to achieve penile erection, or to maintain erection of sufficient firmness and duration to penetrate the vagina of a sexual partner. This topic lies at the heart of an immense body of folklore and urban legendry going back thousands of years. In remote antiquity it was often blamed on sorcery, a curse invoked by an enemy or by a jealous or rejected lover. That notion persists even today in some primitive cultures. Ancient and medieval works on medicine and pharmacology recommend a variety of botanical and other substances as infallible treatments for sexual impotence or as amulets to prevent it. Often the same substances figure in the composition of aphrodisiacs (love potions).

Erectile tissue occurs in the lips and nipples of both sexes as well as in the clitoris, penis, and other parts of the reproductive system. Its characteristic structural feature is a rich bed of anastomosing blood vessels and sinuses with unique inflow and outflow dynamics. When, under erotic stimulation, the arteriolar channels leading into a zone of erectile tissue open wide, the vascular bed becomes engorged and swelling occurs. The venules draining the tissue are so placed that this swelling compresses them, reducing outflow and increasing engorgement. Active constriction of the venules draining erectile tissue is not believed to occur.

Erectile tissue and its overlying skin are supplied with specialized nerve endings that respond to tumescence with heightened erotic sensations, enhancing the degree of engorgement.

By far the largest aggregation of erectile tissue in either sex occurs in the corpora cavernosa of the penis. Immediately beneath the skin of the penis is a tough fibrous envelope (Buck's fascia) that surrounds all three corpora. Dilatation of the vascular beds in erectile tissue compresses the venules that drain it against this unyielding fascial covering, promoting further vascular engorgement.

Penile erection occurs through an interplay of nervous and vascular phenomena and by a variety of physiologic processes. Direct physical stimulation of the penis causes erection through a spinal reflex involving sacral parasympathetic nerves. Like all spinal reflexes, this response is independent of brain function and can therefore occur in a man with complete transection of the spinal cord at the cervical level. And it is nonspecific, often occurring outside an erotic context. Erections are often observed in newborn babies—for example, as a result of manipulation during circumcision.

If any topics remain taboo these days in the news and entertainment media and in polite conversation, erectile dysfunction (ED) isn't one of them. Public figures discuss it frankly on national television, and pharmaceutical companies hawk their remedies for it with increasingly salacious TV commercials.

Erotic ideation due to visual stimuli or fantasy provokes erection by a more complex mechanism involving corticospinal tracts leading to the pelvic splanchnic nerves. Penile erection occurs regularly during REM (rapid eye movement) sleep by yet a third mechanism.

During the past 25 years, animal research has led to the identification of nitric oxide (NO) as a crucial neurotransmitter and vasodilator in the phenomenon of erection. Parasympathetic nerve endings induce dilatation in the arterioles that supply erectile tissue by releasing nitric oxide. Because nitric oxide is instantly degraded by tissue enzymes, nerve stimulation by itself is insufficient to produce a sustained erection. However, once vascular dilatation and engorgement occur, the endothelium (the lining membrane of blood vessels) begins to produce its own supply of NO through the action of an enzyme called endothelial nitric oxide synthase (eNOS). This triggers a biochemical cascade in which further engorgement leads to additional release of NO until maximal firmness of tissue is reached. It has recently been discovered that NO also acts as a neurotransmitter in the central nervous system (CNS), specifically in the paraventricular nucleus (PVN) of the hypothalamus, which is known to be concerned with psychologically induced erection.

The vast majority of penile erections subside spontaneously. At the height of male sexual arousal, however, ejaculation of semen can occur as sympathetic nerves trigger powerful contractions of the bulbospongiosus muscle. Also called the *bulbocavernosus*, this is a sheath of smooth muscle fibers surrounding the structures at the root of the penis, including the seminal vesicles and the urethra.

From puberty onward, penile erection occurring during REM sleep is occasionally associated with erotic dreaming. In men without regular sexual outlets, this may culminate in ejaculation (nocturnal emission, "wet dream"), which may or may not awaken the sleeper.

Ejaculation is accompanied by a climax of sexual pleasure (orgasm) that is felt almost exclusively in the perineum. As in women, male orgasm is accompanied by tachycardia, elevation of blood pressure, and rhythmic contractions of the anal sphincter. Male orgasm is followed almost at once by a transitory loss of libido (sexual desire) and by detumescence of

Surgical procedures for advanced or highly malignant prostatic carcinoma include radical prostatectomy, which is associated with a considerable risk of urinary incontinence and erectile dysfunction, erectile dysfunction, external x-ray or proton beam radiation, or transperineal implantation of radioactive isotopes.

the penis due to constriction of the arterioles supplying its erectile tissue.

Most men experience occasional failure to achieve adequate erection because of adverse physical or psychological circumstances: pain, negative emotions (anger, anxiety, grief, guilt), alcoholic intoxication or drug use, visual or auditory distractions. As currently used in medicine, the term *erectile dysfunction* implies a persistent problem.

Risk factors for ED include hypertension, cigarette smoking, and lack of physical exercise. The incidence of the disorder rises steadily with advancing years, chiefly because of degenerative changes in blood vessels. Arteriosclerosis can inhibit erection in a purely mechanical fashion by reducing the blood supply to the penis.

One form of vascular ED results from arteriosclerosis of the distal aorta, one or both common iliac arteries, or all three vessels. Leriche syndrome consists of reduction or absence of femoral pulses, intermittent claudication (pain in calf muscles induced by walking and relieved by rest), and erectile dysfunction. The diagnosis is confirmed by vascular imaging, and treatment is by replacement of the diseased vessels with a graft.

Depression is often accompanied by loss of libido and erectile failure. Damage to certain areas of the cerebral cortex, the hypothalamus, or the spinal cord by trauma, surgery, or disease (vascular, degenerative, infectious, or malignant) can diminish or abolish the neural signals for erection that are normally triggered by visual or auditory stimuli or erotic fantasy. As mentioned earlier, some men with spinal cord transection can achieve erection after manual stimulation of the penis.

Severing of pelvic splanchnic nerves during prostatic or colorectal surgery or other procedures can eliminate the neural stimulation that opens vascular channels to initiate erection. Even minor surgery such as inguinal hernia repair and vasectomy is sometimes followed by varying degrees of sexual dysfunction. Other sources of neurologic ED are damage to pelvic nerves by radiation therapy and frequent or prolonged bicycle riding.

As noted earlier, the treatment of BPH with drugs that block the synthesis of dihydrotestosterone can cause erectile dysfunction. Deficiency of testosterone or of thyroid hormone due to failure of the gland producing it is sometimes associated with loss of libido and varying degrees of sexual dysfunction. Injections of testosterone have helped some men to overcome ED.

Numerous prescription medicines have been implicated as causes of erectile failure, in particular antihypertensives, anticonvulsants, antihistamines, histamine H_2-receptor antagonists, antiparkinson drugs, nonsteroidal anti-inflammatory drugs, tranquilizers, sedatives, and muscle relaxants. Regular abuse of alcohol or recreational drugs, including marijuana, can also cause chronic sexual dysfunction.

ED is a frequent complication of diabetes mellitus. As noted earlier, the paraventricular nucleus (PVN) of the hypothalamus is known to be involved in centrally mediated penile erection. Studies in rats with chemically induced diabetes mellitus have demonstrated an association between erectile dysfunction and a reduction of neuronal nitric oxide synthase (nNOS) in the PVN. Replenishing nNOS in the PVN by gene transfer significantly restores erectile responses. These studies clarify one mechanism of ED in diabetes and suggest a promising form of therapy.

Sexual impotence, although a seemingly inexhaustible source of ribald humor, is no joke to the victim. For most men, a persistent problem in achieving an erection is emotionally devastating. The resulting anxiety and depression typically aggravate and perpetuate the problem. Many sufferers are too embarrassed to seek medical treatment. Some turn in desperation to expensive but useless folk or quack remedies.

But until the last third of the twentieth century, scientific medicine had little to offer the patient with ED. When no underlying disorder could be identified, the condition was assumed to be of psychological origin, and treated with counseling, psychoanalysis, or psychotropic drugs. Testosterone injections were often administered without clear-cut indications; any favorable results achieved probably represented a placebo effect for most patients.

The alkaloid yohimbine, derived from the bark of a West African evergreen tree (*Pausinystalia yohimbe*), has a long-standing reputation as an aphrodisiac for both men and women. For most of the twentieth century it was one of the very few drugs endorsed by the medical profession as a treatment for ED. Pharmacologic studies have shown that it is an alpha-2 adrenergic receptor antagonist and that it alters dopamine and serotonin metabolism, but controlled studies have failed to demonstrate consistent improvement in erectile function. The drug is nonetheless currently available in both over-the-counter and prescription formulations.

For want of adequate pharmacologic agents to treat ED, various purely mechanical measures have been used with some success. For example, penile erectile tissue can be engorged with blood by external application of a vacuum, and the erection maintained by placement of an elastic ring around the base of the penis. Semirigid rods of synthetic material can be surgically implanted in the corpora cavernosa, creating a permanent partial erection. Or collapsible tubes may be implanted, which are distended with fluid from a reservoir in the thigh by activation of a pump placed in the scrotum.

In the 1970s it was discovered that local injection of adrenergic blocking agents such as papaverine and phentolamine directly into the penis can promote erection in men with various types of ED. Injectable alprostadil (Caverject) was

found to be an acceptable form of therapy despite the discomfort of the injections. The drug is also effective in the form of a urethral suppository or a topical cream.

The arrival in the 1990s of a new class of drugs for ED, the phosphodiesterase-5 (PDE-5) inhibitors, had a profound and lasting impact on many aspects of human sexuality. The prototype of these drugs was designed to treat hypertension by dilating blood vessels. Although it failed in clinical trials as an antihypertensive, it had an unexpected and seemingly miraculous "side effect"—and the rest is history.

To understand how these agents work, you need another small dose of biochemistry. Nitric oxide does not dilate blood vessels directly. Rather, it triggers the release of cyclic guanosine monophosphate (cGMP) type 5, which relaxes smooth muscle fibers in arteriolar walls. This agent is rapidly degraded by the enzyme phosphodiesterase-5 (PDE-5). Phosphodiesterase inhibitors prevent the rapid disappearance of cGMP and permit freer and more prolonged dilatation of vascular channels in erectile tissue.

The phosphodiesterase inhibitors currently on the market, as everyone over the age of 10 probably knows, are sildenafil (Viagra), tadalafil (Cialis), and vardenafil (Levitra). Although these are prescription drugs, they can be obtained—for a price—with minimal or no physician contact on the Internet and elsewhere. Tens of thousands of men without ED are taking them in the vain hope of enhancing sexual pleasure.

They have nonetheless revolutionized the medical management of ED. Except in cases of severe penile deformity, vascular disease, or nerve damage, PDE-5 inhibitors provide prompt correction of the problem from the first dose in a high percentage of cases, whether the cause is physical or psychological. Indeed, this is so well known to the general public that most men who consult physicians nowadays for the treatment of sexual dysfunction are unwilling to submit to psychological evaluation, blood tests, physical examination, or involvement of a sexual partner in diagnostic assessment. They want to try a phosphodiesterase inhibitor first, and only if that fails are they agreeable to diagnostic evaluation.

Sexual impotence, although a seemingly inexhaustible source of ribald humor, is no joke to the victim. For most men, a persistent problem in achieving an erection is emotionally devastating. The resulting anxiety and depression typically aggravate and perpetuate the problem. Many sufferers are too embarrassed to seek medical treatment.

John H. Dirckx, M.D., is the author of *Laboratory Tests and Diagnostic Procedures in Medicine* (2004), *Human Diseases*, 2nd ed. (2003), *H&P: A Nonphysician's Guide to the Medical History and Physical Examination*, 3rd ed. (2001), published by Health Professions Institute. He is medical editor of all HPI publications.

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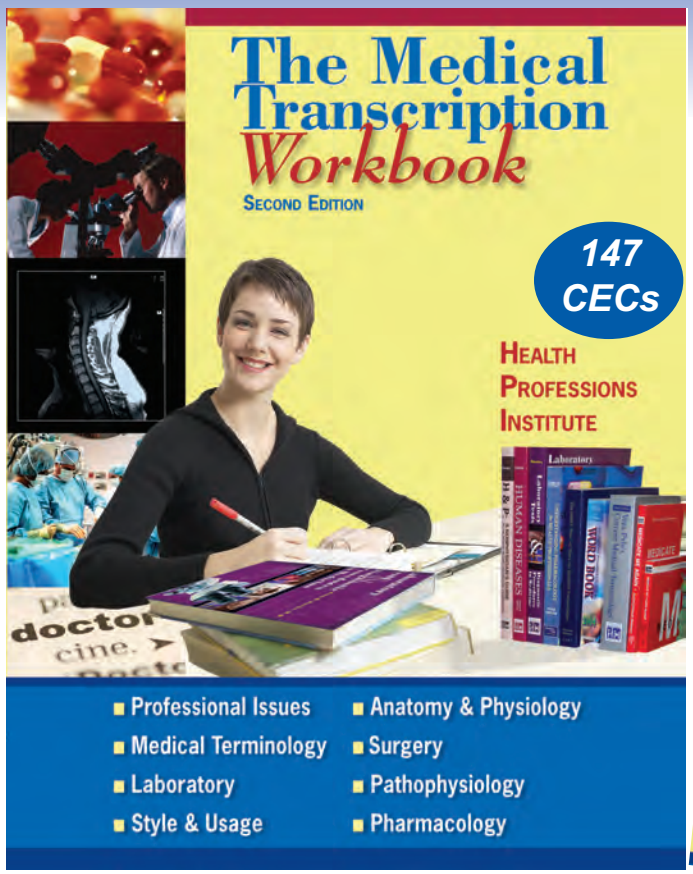

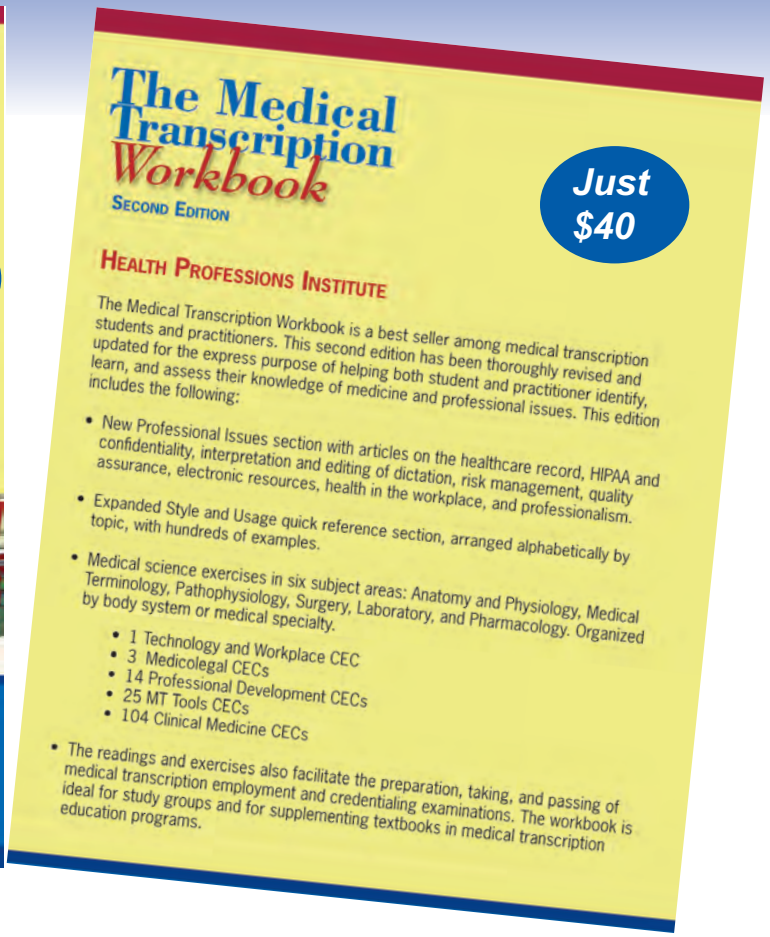

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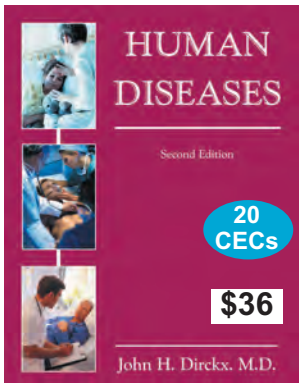

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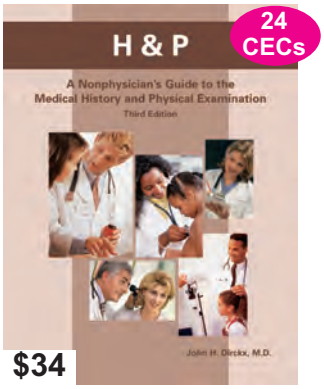

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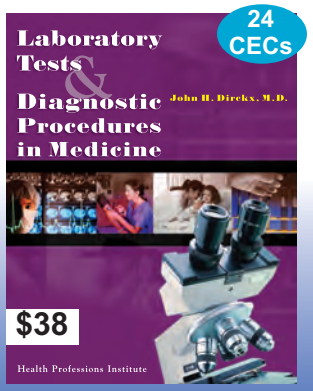

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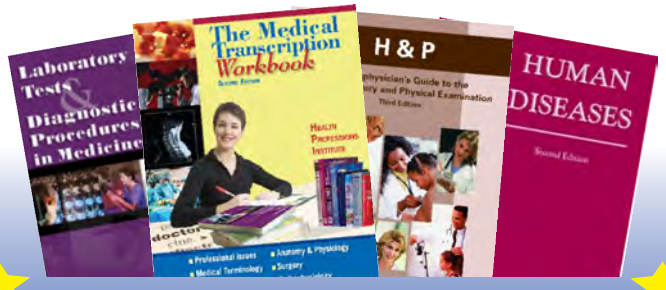

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