frequency of significant skin irritation with the testosterone patch, many practitioners prefer one of the other modes, usually based on patient preference. For dosage and administration of gel testosterone formulations, the clinician should refer to the package insert. For IM testosterone, a good starting dosage is 100 mg every 2 weeks. Pellet implantation is an office procedure, but adequate testosterone levels are typically maintained for 3 to 4 months.

Side Effects of Testosterone Therapy

Testosterone therapy causes decreased sperm production and usually decreased testicular volume, and it may cause acne, oily skin, and breast tenderness. In addition, testosterone can increase hematocrit and cause life-threatening erythrocytosis. If the hematocrit becomes elevated, then testosterone replacement should be suspended. Occasionally, a patient requires phlebotomy to avoid or treat dangerous erythrocytosis. Testosterone replacement is not a treatment for infertility and has in fact been investigated as method of male contraception. Patients who are interested in fathering children should not take testosterone. If necessary, human chorionic gonadotropin (HCG) may be administered for men wishing to preserve fertility.

Only oral testosterone has negative affects on the liver, so it is not necessary to monitor liver function during treatment. Testosterone therapy may worsen obstructive sleep apnea and congestive heart failure; therefore, patients in whom these conditions are untreated should not be started on testosterone therapy. Present data suggest that testosterone therapy does not worsen levels of high-density lipoproteins. Patients should be aware that testosterone therapy suppresses endogenous testosterone and sperm production. Depending on the duration of treatment, it may take 1 to 2 months or longer after treatment discontinuation for testosterone and sperm production to return to baseline levels.

Testosterone Therapy and the Prostate

In general, testosterone therapy should be avoided in patients with a history of prostate or breast cancer. However, this is a controversial issue. Some urologists may decide that replacement is acceptable under specific circumstances because there are data to suggest that testosterone replacement may not pose an undue risk of prostate cancer recurrence or progression. In addition, there is no conclusive evidence that testosterone replacement has any effect on benign prostatic hypertrophy (BPH).

Monitoring Testosterone Treatment

TT should be measured 5 to 12 weeks after initiation of testosterone treatment. If IM testosterone is being administered every 2 weeks, morning TT levels should be checked 1 week after an injection. If a testosterone gel is used, morning TT should be checked at least 1 month after initiation of treatment. Hematocrit values should be assessed at 3 months and 6 months and then annually. If the hematocrit level is greater then 54%, the treatment should be stopped. If testosterone therapy was started to help with osteoporosis or osteopenia, a bone mineral density test should be done after 1 to 2 years. If there is an abnormality in the PSA level or it increases by 0.75 ng/mL in 1 year, referral to a urologist should be considered. If the TT level is low, the clinician should increase the dosage and reassess after 5 to 12 weeks. The goal of therapy should be a level between 400 and 600 ng/dL. Finally, therapy should be discontinued if the patient does not have clinical improvement.

B. Erectile Dysfunction

ED is defined as the inability to maintain an erection for satisfactory sexual function in the absence of premature ejaculation. Premature ejaculation is not technically a form of ED. Impotence is the most common cause of ED in the United States and is defined by an inability to attain or maintain adequate penile rigidity sufficient for intercourse. Other important, although less common, causes of ED are Peyronie's disease and trauma.

ED affects millions of American men. According to the Massachusetts Male Aging Study, 52% of men older than 40 years of age are afflicted with some form of impotence. The prevalence of impotence triples between age 40 and 70 years. By age 70 years, 15% of men experience complete ED. Age and physical health are the most important predictors of the onset of ED. Smoking is the most important lifestyle variable.

Recently, many clinics that specifically treat ED and premature ejaculation have opened across America to meet a growing need for treatment of these conditions. However, these clinics often charge money for treatments and medications that primary care physicians can provide and are covered by insurance. Therefore, it is more important than ever for primary care physicians to have a thorough understanding of this disease process.

MECHANISM OF ERECTION

Psychogenic or tactile sexual stimulation is usually the initial step in the pathway leading to penile erection. Nerve signals are carried through the pelvic plexus into the cavernous nerves of the penis. The pelvic plexus receives input from both the sympathetic and the parasympathetic nervous system. Sympathetic fibers originate in the thoracolumbar spinal cord, and parasympathetic fibers originate in the second through fourth sacral spinal cord segments (S2 through S4). Afferent somatic sensory signals are carried from the penis through the pudendal nerve to S2 through S4. This information is routed both to the brain and to spinal cord autonomic centers. Adrenergic innervation appears to play a role in the process of detumescence. High concentrations of norepinephrine have been demonstrated in the tissue of the corpora cavernosa and tributary arterioles. Afferent signals capable of initiating erection can originate within the brain, as with psychogenic stimulation, or they can result from tactile stimulation. There is no discreet center for psychogenic erections; however, the temporal lobe appears to be important.

Sexual stimulation causes the release of nitric oxide (NO) by the cavernous nerves into the neuromuscular junction