Erectile Dysfunction

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KEYWORDS

• Erectile dysfunction • Impotence • Sexual dysfunction • PDE-5 inhibitors

KEY POINTS

- Erectile dysfunction (ED), although recognized as a pathologic condition for thousands of years, was not clearly defined until 1992, thus overcoming one of many barriers to treatment.
- ED is defined as the inability to achieve and/or maintain erection of sufficient rigidity and duration to permit satisfactory sexual performance.
- Causes of ED are psychogenic, vasculogenic, neurogenic, endocrinologic, cavernosal smooth muscle dysfunction, iatrogenic, or pharmacologic; for many patients the cause may be any combination of these.
- Diagnosis and evaluation of ED can be as simple as using a questionnaire but can also involve complex testing and imaging modalities, with varying degrees of reliability and clinical utility.
- Treatment of ED usually follows a stepwise progression, from noninvasive strategies, such as lifestyle modifications and oral medications, all the way to surgical placement of penile prostheses for severe refractory disease.

HISTORY

Although recognized as a pathologic condition for several millennia, the systematic and evidence-based investigation of erectile dysfunction (ED) is a relatively recent phenomenon in modern medicine. For as long as humans have been studying their sexuality, they have also been documenting the affliction of sexual dysfunction. By 1150 BCE, the ancient Egyptians had described 12 different sexual positions with explicit papyrus drawings that were passed down, studied, annotated, and preserved throughout the centuries. Predating these drawings by several hundred years, the Ebers papyrus contains, among many other remedies, prescriptions for "weakness of the male member" (1700 BCE).[1] Yet it was not until 1992 that the National Institutes of Health held a multidisciplinary consensus conference on impotence and officially defined ED as the inability to achieve and/or maintain erection of sufficient rigidity and duration to permit satisfactory sexual performance.[2] This marked a turning point

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for physicians and patients, making it easier to identify, diagnose, and address ED in common medical practice.

EPIDEMIOLOGY AND RISK FACTORS

The Massachusetts Male Aging Study (MMAS) was the first cross-sectional population study for prevalence of ED, taking a random sample of men in Boston, ages 40 to 70, and using a self-administered sexual activity questionnaire to stratify mild, moderate, and complete ED. Overall prevalence of ED, regardless of type and severity, was 52% in this cohort, and prevalence of complete ED increased from 5.1% in men age 40% to 15% in the 70-year-old group. The probability of moderate ED increased from 17% to 34% with age, and the prevalence of mild ED stayed at 17% regardless of age.[3] More recent data from the Boston Area Community Health Survey, 2005 to 2006, revealed 10% of men in their 30s compared with 59% of men in their 70s had ED.[4] Worldwide prevalence of ED was estimated at 5% to 28% based on the Global Survey of Sexual Attitudes and Behaviors, which included men and women ages 40 to 80 from 29 different countries.[5] The MMAS data from 1987 to 1989 was later compared with new data collected from 1995 to 1997, representing the first reported longitudinal data set for establishing incidence of ED. The study concluded risk of ED was approximately 26 new cases per 1000 men annually. There was a higher incidence in older men (46.4/1000 in men 60–69) and in men with cardiovascular disease (58.3/1000), hypertension (42.5/1000), and diabetes (50.7/1000). Other independent risk factors for ED include smoking, obesity, depressive symptoms, metabolic syndrome, hyperlipidemia, sedentary lifestyle, spinal cord injury, certain medications, neurodegenerative diseases, renal insufficiency, prostate cancer treatments, blunt perineal or pelvic trauma, and bicycle riding.[6–12]

ERECTILE PHYSIOLOGY

Normally, blood supply to the penis arises from the internal pudendal artery, which branches from the internal iliac artery, although there is often collateral circulation with accessory pudendal branches arising from other pelvic arteries, such as the external iliac, obturator, vesical, and femoral arteries.[13] The internal pudendal artery eventually becomes the common penile artery, which then branches into dorsal, bulbourethral, and cavernous arteries. The dorsal artery supplies the glans, and the cavernous artery is responsible for erection as it supplies the corpora cavernosa filling all of the branching helicine arteries, trabecular erectile tissue, and the sinusoids housed as a matrix of elastic erectile tissue within the cylindrical bilayered tunica albuginea. The outer layer of the tunica consists of collagen and elastin fibers arranged in a longitudinal fashion and the inner layer of circular-running fibers. On the periphery of the cavernosal sinusoids, there are subtunical venous plexuses, which drain venous blood and give rise to the emissary veins that penetrate through the tunical layers out to the larger return vessels.[14]

At baseline, the trabecular smooth muscle matrix of the corpus cavernosum is in a state of moderate tonic contraction, thus limiting arterial inflow and maintaining flaccidity. When the smooth muscle is further contracted, as in cold temperatures, the blood flow is further limited, resulting in shrinkage of the phallus. During erection, sexual stimulation via both somatic and autonomic pathways causes release of several neurotransmitters, including dopamine, serotonin, oxytocin, and, most importantly, nitric oxide (NO).[15] NO is responsible for binding and activating guanylyl cyclase, the enzyme that catalyzes the transition of guanosine 5′-triphosphate (GTP) to cyclic guanine monophosphate (cGMP), which relaxes cavernous smooth muscle.