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FAMILY PRACTICE BOARD REVIEW MANUAL

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Erectile Dysfunction

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I. INTRODUCTION

Erectile dysfunction (ED) is defined as the inability to obtain or sustain an erection sufficient for sexual intercourse. This condition affects approximately 50% of the male population between the ages of 40 and 70 years and is probably under-reported.¹ The prevalence increases with age, especially after age 40 years.

Although diagnosing ED is fairly straightforward, determining the cause may be more difficult. The cause is often multifactorial and may include primary organic disorders, psychologic problems, or a combination thereof. Most often cases involve a mixture of organic and psychologic causes.

Because treatment for ED frequently depends on etiology, it is important to perform a thorough evaluation of a patient who presents with sexual dysfunction, including a complete physical examination and medical and sexual history. Patients with ED and their partners usually suffer in silence. With new advances in medicine, however, most men will benefit from some form of treatment.

NORMAL MALE SEXUAL FUNCTION

Neurologic Component

The male sexual cycle is divided into 4 phases (Table 1). Erection (phase 2) is preceded by a desire for sex (phase 1), which is thought to be regulated in part by

hormonal and psychologic factors. Visual, olfactory, auditory, imaginative, or physical stimulation leads to involvement of the cortical, hypothalamic, limbic, and sympathetic and parasympathetic nervous systems. This results in the synthesis and release of nitric oxide and guanylate cyclase, which are necessary for the manufacture of cyclic guanosine monophosphate (GMP). Increased levels of cyclic GMP cause smooth muscle relaxation, increasing blood flow into the corpora cavernosa and resulting in erection (Figure 1). Phosphodiesterase type 5 (PDE5) catalyzes the conversion of cyclic GMP to GMP. The resultant vasoconstriction causes an outflow of blood from the corpus cavernosum resulting in detumescence.

Vascular Component

The transformation of the penis to an erect organ requires not only an intact neurologic pathway but also a properly functioning vascular system. Blood is supplied to the penis by the cavernosal artery (a branch of the pudendal artery). Relaxation of the corpora cavernosa (mediated by the parasympathetic nervous system) leads to increasing blood flow into the cavernosal space. Blood trapped in the expanding sinusoidal system causes compression of the cavernosal veins, resulting in occlusion of the venous system. This results in the increased intercorporal pressure that leads to rigidity. Complete rigidity is also caused in part by stimulation of the somatic nerve endings, which leads to contraction of the ischiocavernosus muscle.²