International Journal of Impotence Research (2007) 19, 37–42

0955-9930/07 $30.00
www.nature.com/ijir

REVIEW

Doppler evaluation of erectile dysfunction – Part 1

D Golijanin[1], E Singer[1], R Davis[1], S Bhatt[2], A Seftel[3] and V Dogra[2]

[1]*Department of Urology, University of Rochester School of Medicine and Dentistry, Rochester, NY, USA;* [2]*Department of Radiology, University of Rochester School of Medicine and Dentistry, Rochester, NY, USA and* [3]*Department of Urology, University Hospitals of Cleveland, Case Western Reserve University, Cleveland, OH, USA*

Erectile dysfunction (ED) is the consistent inability to achieve and maintain an erection sufficient for satisfactory sexual activity. Erectile dysfunction affects as many as 30 million men in America, with an increasing prevalence with age. Erectile dysfunction is secondary to organic, psychogenic and combined causes. The first part of this review article describes the guidelines for evaluation and treatment plans for men with ED. It also describes the normal sonographic anatomy of the penis, sonographic technique for evaluation of ED and the normal phases of erection.
International Journal of Impotence Research (2007) **19,** 37–42. doi:10.1038/sj.ijir.3901477; published online 20 April 2006

Keywords: erectile dysfunction; impotence; ultrasound; color Doppler; erection; penis

Introduction

National Institutes of Health defines erectile dysfunction (ED) as the consistent inability to achieve and maintain an erection sufficient for satisfactory sexual activity. Erectile dysfunction affects as many as 30 million American men.[1] The disorder is associated with age, with a 39% prevalence at the age of 40 years and a 67% prevalence at the age of 70 years. Massachusetts Male Aging Study reported on 52% of men aged 40–70 years having some degree of erectile difficulty.[2,3]

Erectile dysfunction includes organic, psychogenic and combined causes. Organic causes are found in 80–90% of patients and include vasculogenic (arterial, cavernosal and mixed), neurogenic, anatomic and endocrine causes. Psychogenic disorders with ED are performance anxiety, depression and inhibited sexual desire.

Guidelines for evaluation and treatment plans for men with ED have usually suggested a stepwise model.

Correspondence: Dr V Dogra, Department of Radiology, University of Rochester Medical Center, 601 Elmwood Avenue, Box 648, Rochester, NY 14642-8648, USA.
E-mail: Vikram_Dogra@urmc.rochester.edu
Received 29 December 2005; accepted 14 March 2006; published online 20 April 2006

Basic evaluation

Patients with ED undergo a preliminary assessment designed to address significant diseases such as diabetes, coronary artery disease (CAD) and hypertension before starting therapy, so non- or/and least invasive treatments are to be used first.[4–6] A basic diagnostic evaluation is the first step in ED assessment and is applied for the majority of men, whereas specific diagnostic procedures are implemented in a smaller subset of patients.[7,8] The basic evaluation is often commenced by the primary care physicians. Occasionally, a number of patients are diagnosed and treated by cardiologists, neurologists, endocrinologists and psychiatrists, who treat ED as a comorbidity of an underlying disease.[9,10]

Basic evaluation includes thorough medical, sexual and psycho-sexual history, physical examination and limited laboratory assessment. Radiological imaging in the field of ED has diminished in importance over the past 10 years with the introduction of new effective oral therapies and the recognition that surgical treatment of both penile venous leak and arterial insufficiency have poor long-term clinical outcomes. The introduction of phosphodiesterase-5 (PDE5) inhibitors has revolutionized the therapeutics of ED and radically changed the way in which men with ED are assessed and investigated.[11] Documentation of a good quality erection in response to a PDE5-inhibiting drug confirms grossly adequate arterial flow and patent veno-occlusive mechanisms. However, color

Doppler ultrasound continues to have a role in the evaluation of specific patients with ED and has an increasing role in the detection of silent CAD in men presenting with ED.[12]

Specific evaluation

Vascular evaluation and imaging may be indicated in patients with (a) arterial/arteriolar arteriogenic dysfunction, (b) veno-occlusive disorder, (c) Peyronie's disease, (d) high-flow priapism, (e) penile trauma (fracture) and (f) in patients without symptomatic peripheral vascular disease presenting with ED where penile Doppler is used to assess the risk of cardiovascular disease and need for further cardiac or peripheral vascular assessment. In the last group, significant risk factors such as hypertension, smoking, trauma, hyperlipidemia, diabetes mellitus, obesity and inactivity are closely related to ED and decrease in sexual ability is regarded as one of the first clinical parameters of increased risk of significant CAD or peripheral vascular disease.[13–15] Erectile dysfunction is now being recognized as one of the earliest manifestations of endothelial dysfunction and peripheral vascular disease. Montorsi *et al.*[16] have demonstrated that ED presents about 39 months before CAD possibly because the smaller penile arteries reach critical narrowing and decreasing blood flow earlier than larger vessels. He has demonstrated that a normal penile Doppler test virtually excludes CAD with a 98% negative predictive value, whereas an abnormal penile Doppler test had a 30% positive predictive value for CAD – a value many times higher than 4% found in the general population.[17] El-Sakka and Morsy[18] in evaluation of men with ED found that 77% of those with high-grade ischemic heart disease had an abnormal penile Doppler test with peak systolic velocity (PSV) of less than 25 cm/s. Gazzaruso *et al.*[19] demonstrated in asymptomatic type II diabetics that those with angiographically confirmed silent CAD had over seven times the rate of ED (33.8% vs 4.7%) than control type II diabetics without CAD. As more information accrues confirming ED as an early manifestation of peripheral vascular disease, penile Doppler testing may play a key role in selecting those who do or do not need further coronary artery vascular assessment.

Vasculogenic imaging is usually reserved for men with ED who have a potentially surgically treatable cause. These patients are frequently young, have often suffered traumatic straddle injuries to the penis and may be unresponsive to oral and intracavernosal therapy. In this group of men, the mainstay of investigation is color Doppler ultrasound of the penile vasculature.[20] In addition to penile color Doppler ultrasound evaluation, several other tests of penile function and anatomy are available, including, dynamic infusion cavernosometry, cavernosography, selective penile angiography, penile nuclear magnetic resonance and near-infrared spectrophotometry.[21]

The use of Doppler ultrasound in the assessment of the penile vasculature was first described in 1985.[22] Advantages of penile Doppler and pharmacologic duplex ultrasonography include objective, minimally invasive evaluation of penile hemodynamics at a relatively low cost. Intracavernosal injection of vasoactive substances including prostaglandin E1, papaverine and phentolamine permits testing penile circulation not only at rest when the flow is minimal but also under maximal direct pharmacologic stimulation, when arterial insufficiency may be observed.[23–25] These substances may be administrated as a single drug or in combination,[23,24] with reported efficacy rates of up to 94%. Intracavernosal injections are routinely given with color Doppler ultrasound, but despite this the tests have been less than completely reliable possibly owing to the negative effects of anxiety and adrenergic output on the testing results. A variety of techniques has been used to minimize anxiety and maximize the reproducibility of the investigations, including a quiet and private environment, manual stimulation and visual sexual stimulation.[26] Others have used avoidance of injection giving high doses of PDE5 inhibitors and visual stimulation to promote blood flow and erection during testing, gaining from a less invasive procedure but losing on the certainty of maximal vascular relaxation stimulation.[27–39] In addition to various techniques employed to stimulate penile blood flow and an erection during testing, in order to increase observer ability to precisely assess vasculogenic causes of ED, interpretation of penile Doppler ultrasound findings remain very difficult in some cases. First, there is a gray area in the criteria for identifying arteriogenic ED. The parameter that is most commonly used to define arteriogenic ED is peak systolic blood flow in the penile arteries. Peak flow rates less than 25 cm/s are abnormally low and peak flow rates greater than 35 cm/s are normal, but the range of 25–35 cm/s is equivocal. Recently, Speel *et al.*[40] reported that acceleration time was the most valid parameter to detect cavernous atherosclerotic pathology. Blood flow velocity in the cavernous artery following pharmacostimulation was determined with duplex ultrasonography in 106 patients with ED. The cutoff point for acceleration time to discriminate between atherosclerotic and non-atherosclerotic ED was determined at acceleration time 100 ms or greater. Sensitivity was 66% and specificity was 71%.

Secondly, the criteria for diagnosing veno-occlusive dysfunction are also not very convincing. During a complete erection, the end-diastolic velocity (EDV) of the cavernosal arteries should be zero, or reverse flow may also be observed as a result of the increased intracavernosal pressure in the rigid stage of erection. In men with veno-occlusive

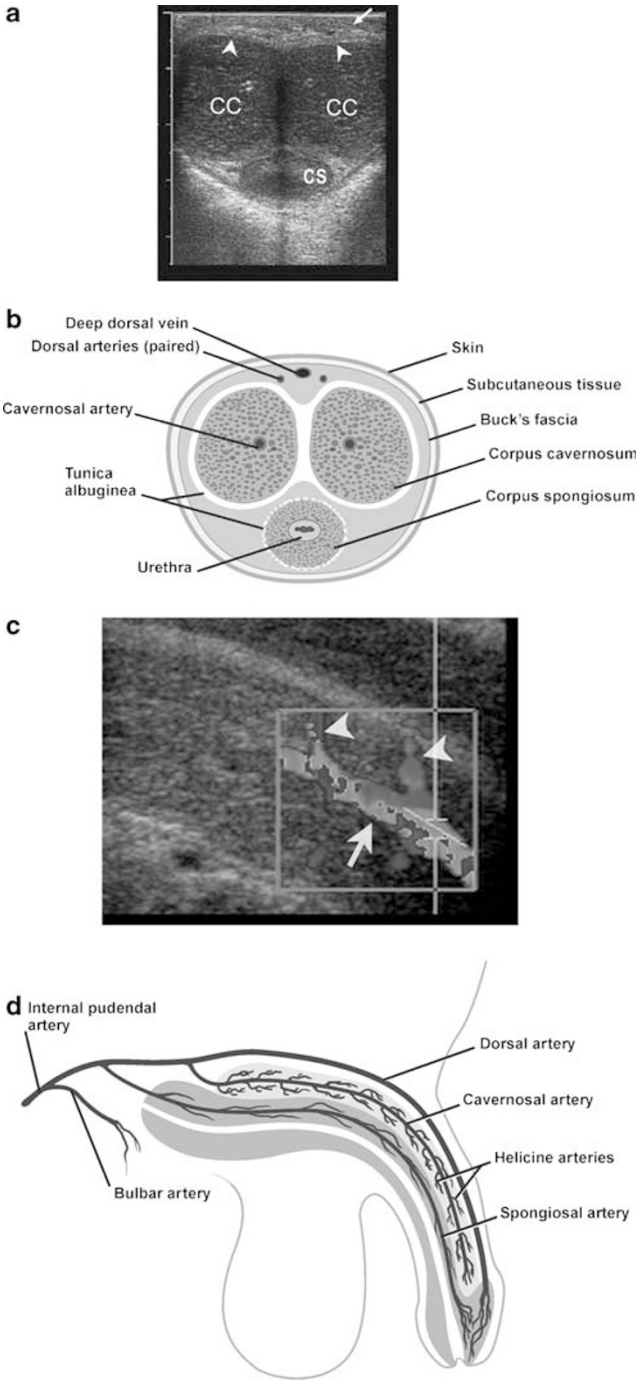

Figure 1 Normal sonogram of penis. (**a**) Transverse gray-scale ultrasound of the penis demonstrates the two corpora cavernosa (CC) surrounded by the tunica albuginea (arrowhead). The corpus spongiosum (CS) is seen inferior to the corpora cavernosa. All three corpora are surrounded by the Buck's fascia (arrow). (**b**) The corresponding line diagram. (**c**) Longitudinal color flow Doppler ultrasound of normal cavernosal bodies and flow through cavernosal artery (arrow) and the helicine branches (arrowheads). (**d**) The corresponding line drawing of the blood supply to the penis.

disorder, the usual criterion for diagnosing veno-occlusive dysfunction has been an EDV greater than 5 cm/s.[41,42] This observation was not confirmed in

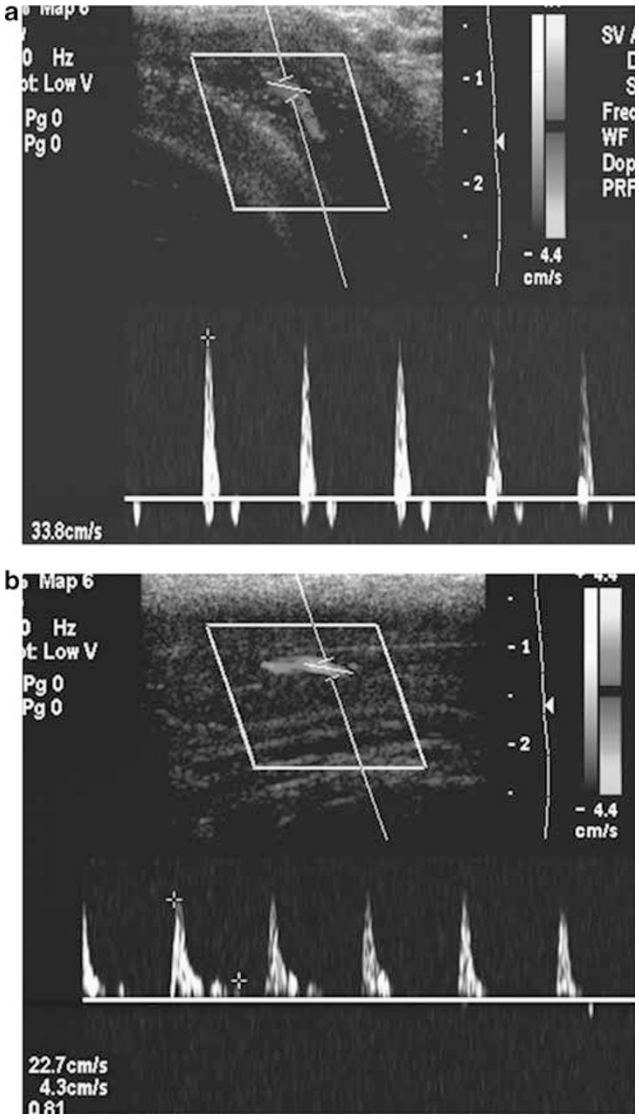

Figure 2 Cavernosal artery peak systolic velocity (PSV). Peak systolic velocity of the cavernosal artery decreases from base of the penis to the glans penis. (**a**) A higher PSV of the cavernosal artery (measured more proximally) as compared to distal measurement in the same patient (**b**).

all studies and there is a poor correlation of color Doppler ultrasound findings and veno-occlusive dysfunction when diagnosis was made using gold standard method, which is cavernosogram with infusion cavernosometry.[39]. Although the accurate diagnostic procedure for venous leak diagnosis is infusion cavernosometry after maximal vasodilatation, this is rarely employed owing to poor surgical results of venous leak repair.

Sonographic penile anatomy

The penis is composed of two dorsal corpora cavernosa and one ventral corpus spongiosum. The

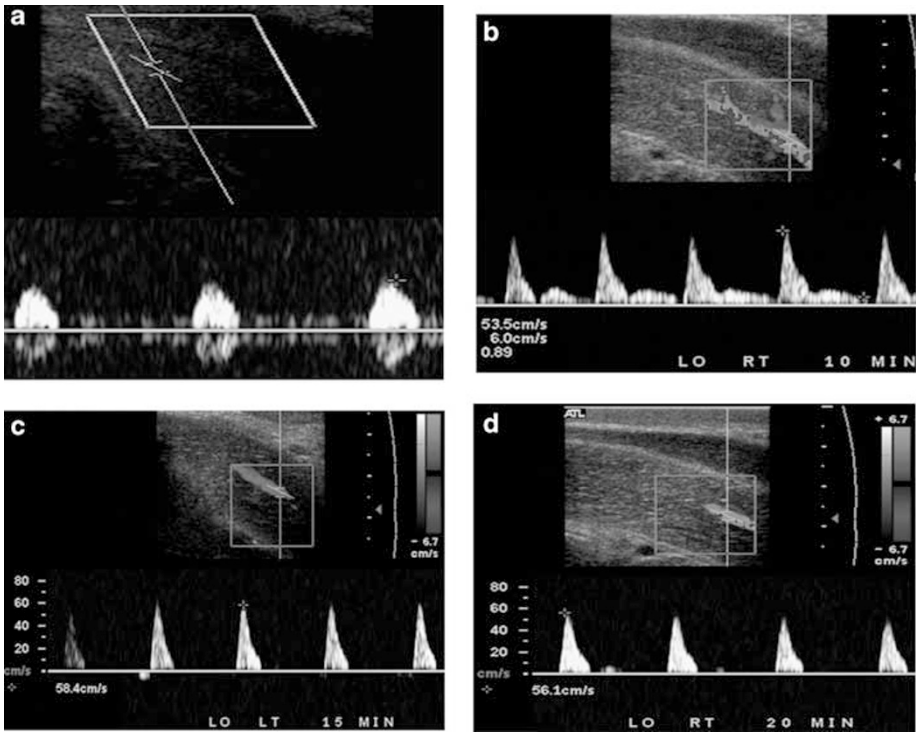

Figure 3 Phases of erection. (**a**) Flaccid phase. Spectral Doppler waveform demonstrates high resistance waveform. Velocities are in the range of 5–15 cm/s. (**b**) Filling phase. Spectral Doppler waveform demonstrates increased systolic velocity and increased diastolic flow. (**c**) Tumescent phase. The systolic velocities stabilize, diastolic flow decreases or becomes reversed. (**d**) Rigid phase. Spectral Doppler waveform demonstrates very high resistance waveform with no diastolic flow and minimal systolic flow. The peak systolic velocities decrease.

two corpora cavernosa are enclosed in a fibrous sheath, the tunica albuginea, which partially covers the corpus spongiosum. The tunica albuginea is composed of elastic fibers that form an irregular, criss-crossing strips network on which collagen fibers rest. The corpora cavernosa are composed of sinusoidal spaces lined by smooth muscles (erectile tissue) and endothelium. The glans penis is formed by expansion of the corpus spongiosum. The corpus spongiosum is traversed throughout its length by the anterior urethra, which begins at the perineal membrane. The corpus spongiosum provides support to the urethra and helps with the expulsion of semen from the urethra. Buck's fascia surrounds both cavernosal bodies dorsally and splits to surround the spongiosum ventrally (Figure 1a and b). The penile blood vessels arise from the internal pudendal artery. The penile artery divides into two main branches, the dorsal penile artery and the cavernosal artery. The cavernosal artery enters the corpus cavernosum on the superomedial surface of the penis. The branches of this artery are called the helicine arteries and subsequently divide into smaller vessels that communicate with the lacunae of the corpus cavernosum (Figure 1c and d). The venous blood is returned by the venous plexus beneath the tunica albuginea. The emissary veins perforate the tunica albuginea, and the blood is drained by the venae circumflexae into the deep dorsal veins.

Applied color Doppler ultrasonography technique

Sonographic examination of the penis is performed with the patient in either the supine or lithotomy (frog leg) position with the penis lying on the anterior abdominal wall or supported with towels between the thighs. High-frequency (7.5–14 MHz) linear array transducers are used to obtain high-resolution images of the penis. A sufficient amount of sonographic acoustic gel should be used on the surface of the penis to obtain good-quality images, and excessive compression by the transducer should be avoided, especially in patients with trauma. The examination is performed in transverse and longitudinal planes starting at the level of the glans and moving down to the base of the penis. A transperineal approach may be used if required to assess the base of the penis. The two corpora cavernosa are homogeneous in echo texture and identified as two hypoechoic circular structures. The tunica albuginea is visualized as a linear hyperechoic structure covering the corpora cavernosa. The cavernosal

artery is visualized on the medial portion of the corpora cavernosa. The corpus spongiosum is often compressed and difficult to visualize optimally from the ventral aspect. Color Doppler examination of the penis should be performed in both transverse and longitudinal planes. Peak systolic velocities of the cavernosal arteries should also be recorded. Owing to the variation in the PSV of the cavernosal artery at different locations across the shaft of the penis (PSV is higher proximally), PSV should be consistently measured at the junction of the proximal one-third and distal two-third of the penile shaft where the cavernosal artery bends (Figure 2). Cavernosal artery velocities in healthy volunteers measure 10–15 cm/s in the non-erect condition. Cavernous arteries should be identified and their action potential dimension measured. When pharmacological testing is required, prostaglandin E1 injection 10–20 μg, is injected to one of the corpora cavernosa laterally in the distal part of the penis with a 30-gauge needle. Injection should always be made in the distal two-third part of the penis on one side only because of the presence of a septum in between the two corpora cavernosa in the proximal part of the penis, which is deficient distally, thus allowing bilateral perfusion of the injected vasoactive substance. Besides prostaglandin E1, other vasoactive agents used are papaverine 30–60 mg and Trimix (combination of papaverine, phentolamine and prostaglandin E1). Trimix is reserved for those patients who do not respond to prostaglandin. An oral vasoactive agent, sildenafil citrate, plus visual sexual stimulation can be used as an alternative to intracavernosal injection of vasoactive agents for penile Doppler evaluation with similar results.

The injection of the vasoactive substance results in physiological response of erection in normal people, and thus helps to differentiate patients with neurogenic or psychogenic dysfunction from those with vascular disturbances.

Similarly, PDE5 inhibitors could be used for pharmacological testing with increased time frame in between examinations. Tumescence and rigidity are observed and recorded, as well as the angle-corrected velocities, in both cavernosal arteries at 5, 10, 15, 20, 25 and 30 min after injection. Velocities in deep dorsal vein should also be recorded.

Phases of erection

The erection process may be divided into four phases. First, the *flaccid* phase during which corporeal volume is small, approximately one-fifth of maximal volume, and corporeal pressure is around 20 mm Hg. In the *filling and tumescence* phase, the corporeal smooth muscle is relaxed owing to reduction in the sympathetic tonus of the arteries allowing brief arterial inflow. The corpora cavernosa are engorged with blood. This results in increase of pressure inside corpora cavernosa, causing pressure at the level of the relaxed veins against the tunica albuginea and preventing outflow. The hemodynamic resistance of the veins increases significantly. This leads to fourth phase of rigidity, where intracavernosal volume grows almost five times baseline and intracorporeal pressure is close to systolic systemic arterial pressure. Final *rigidity* is obtained by contraction of the perineal muscles that generate high-pressure peaks giving the rigidity required for full erection. Doppler ultrasonography accurately divides phases of erection with characteristically different spectral waveforms as seen in Figure 3.

References

1 NIH Consensus Conference. Impotence. NIH consensus development panel on impotence. *JAMA* 1993; **270**: 83–90.
2 Johannes CB, Araujo AB, Feldman HA, Derby CA, Kleinmann KP, McKinlay JB. Incidence of erectile dysfunction in men 40–69 years old. Longitudinal Results from the Massachusetts Male Aging Study. *J Urol* 2000; **163**: 460–463.
3 Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB. Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. *J Urol* 1994; **151**: 54–61.
4 Padma-Nathan H. Diagnostic and treatment strategies for erectile dysfunction: the 'process of care' model. *Int J Impot Res* 2000; **12**(Suppl 4): S119–S121.
5 Gingell C, Wright P, Barnes T, Dean J, Dinsmore W, Eardley I *et al*. Guidance on the management of erectile dysfunction in primary care. *Prescriber* 1999; (Suppl 5): 1–15.
6 Jardin A, Wagner G, Khoury S. Recommendations of the 1st International Consultation on Erectile Dysfunction. In: Jardin A, Wagner G, Khoury S, Giuliano F, Padma-Nathan H, Rosen R (eds). *Erectile Dysfunction*. Plymbridge Distributors Ltd.: Plymouth, UK, 2000, pp 711–726.
7 Wespes E, Amar E, Hatzichristou D, Montorsi F, Pryor J, Vardi Y. European association of urology. Guidelines on erectile dysfunction. *Eur Urol* 2002; **41**: 1–5.
8 Levine LA. Diagnosis and treatment of erectile dysfunction. *Am J Med* 2000; **109**(Suppl 9A): 3S–12S discussion 29S–30S).
9 Levine LA, Kloner RA. Importance of asking questions about erectile dysfunction. *Am J Cardiol* 2000; **86**: 1210–1213.
10 Falk RH. The cardiovascular response to sexual activity: do we know enough? *Clin Cardiol* 2001; **24**: 271–275.
11 Speel TGW, Bleumer I, Diemont WL, van der Maas MC, Wijkstra H, Meuleman EJ. The value of sildenafil as mode of stimulation in pharmaco-penile duplex ultrasonography. *Int J Impot Res* 2001; **13**: 189–191.
12 Barrett-Connor E. Cardiovascular risk stratification and cardiovascular risk factors associated with erectile dysfunction: assessing cardiovascular risk in men with erectile dysfunction. *Clin Cardiol* 2004; **27**(4, Suppl 1): I8–I13.
13 Shamloul R, Ghanem HM, Salem A, Elnashaar A, Elnaggar W, Darwish H *et al*. Correlation between penile duplex findings and stress electrocardiography in men with erectile dysfunction. *Int J Impot Res* 2004; **16**: 235–237.
14 El-Sakka AI, Morsy AM, Fagih BI, Nassar AH. Coronary artery risk factors in patients with erectile dysfunction. *J Urol* 2004; **172**: 251–254.
15 Goldstein I. The mutually reinforcing triad of depressive symptoms, cardiovascular disease, and erectile dysfunction. *Am J Cardiol* 2000; **86**(Suppl): 41F–45F.

16 Montorsi P, Ravagnani PM, Galli S, Briganti A, Salonia A, Dehó F *et al.* Association between erectile dysfunction and coronary artery disease: a case report study. *J Sex Med* 2005; **2**: 575.
17 Enbergs A, Bürger R, Reinecke H, Borggrefe M, Breithardt G, Kerber S. Prevalence of coronary artery disease in a general population without suspicion of coronary artery disease: angiographic analysis of subjects aged 40–70 years referred for catheter ablation therapy. *Eur Heart J* 2000; **21**: 45–52.
18 El-Sakka AI, Morsy AM. Screening for ischemic heart disease in patients with erectile dysfunction: role of penile Doppler ultrasonography. *Urology* 2004; **64**: 346–350.
19 Gazzaruso C, Giordanetti S, De Amici E, Bertone G, Falcone C, Geroldi D *et al.* Relationship between erectile dysfunction and silent myocardial ischemia in apparently uncomplicated type 2 diabetic patients. *Circulation* 2004; **110**: 22–26.
20 Meuleman EJ, Diemont WL. Investigation of erectile dysfunction. Diagnostic testing for vascular factors in erectile dysfunction. *Urol Clin N Am* 1995; **22**: 803–819.
21 Kayigil O, Metin A. Relaxation degree: a new concept in erectile dysfunction. *Int Urol Nephrol* 2001; **33**: 391–394.
22 Lue TF, Hricak H, Marich KW, Tanagho EA. Vasculogenic impotence evaluated by high resolution ultrasonography and pulsed Doppler spectrum analysis. *Radiology* 1985; **155**: 777–781.
23 Virag R, Shoukry K, Floresco J, Nollet F, Greco E. Intracavernous self-injection of vasoactive drugs in the treatment of impotence: 8-year experience with 615 cases. *J Urol* 1991; **145**: 287–292.
24 Montorsi F, Guazzoni G, Bergamaschi F, Dodesini A, Rigatti P, Pizzini G *et al.* Effectiveness and safety of multidrug intracavernous therapy for vasculogenic impotence. *Urology* 1993; **42**: 554–558.
25 Heaton JP, Lording D, Liu SN, Litonjua AD, Guangwei L, Kim SC *et al.* Intracavernosal alprostadil is effective for the treatment of erectile dysfunction in diabetic men. *Int J Impot Res* 2001; **13**: 317–321.
26 Montorsi F, Guazzoni G, Barbierir L, Ferini-Strambi L, Iannacone S, Calori G *et al.* Genital plus audiovisual sexual stimulation following intracavernous vasoactive injection versus re-dosing for erectile dysfunction: results of a prospective study. *J Urol* 1998; **159**: 113–115.
27 Park K, Kwon DD, Oh BR, Ryu SB, Park YI. Efficacy of virtual glasses in audio-visual sexual stimulation during penile color duplex Doppler ultrasonography. *Eur Urol* 2002; **41**: 62–65.
28 Speel TG, Bleumer I, Diemont WL, van der Maas MC, Wijkstra H, Meuleman EJ. The value of sildenafil as mode of stimulation in pharmaco-penile duplex ultrasonography. *Int J Impot Res* 2001; **13**: 189–191.
29 Bacar MM, Batislam E, Altinok D, Yilmaz E, Bacar H. Sildenafil citrate for penile hemodynamic determination: an alternative to intracavernosal agents in Doppler ultrasound evaluation of erectile dysfunction. *Urology* 2001; **57**: 623–626 (discussion 626–662).
30 Bhargava R, Srivastava DN, Thulkar S, Kropman RF, Schiper J, van Oostayen JA *et al.* The value of increased end diastolic velocity during penile duplex sonography in relation to pathological venous leakage in erectile dysfunction. *J Urol* 1992; **148**: 314–317.
31 Furst G, Muller-Matheis V, Cohnen M, Trautner C, Haastert B, Saleh A *et al.* Venous incompetence in erectile dysfunction: evaluation with colour coded duplex sonography and cavernosometry/graphy. *Eur Radiol* 1999; **9**: 35–41.
32 Lue TF, Mueller SC, Jow YR, Hwang TI. Functional evaluation of penile arteries with duplex ultrasound in vasodilator-induced erection. *Urol Clin N Am* 1989; **16**: 799–807.
33 Quam JP, King BF, James EM, Lewis RW, Brakke DM, Ilstrup DM *et al.* Duplex and color Doppler sonographic evaluation of vasculogenic impotence. *Am J Roentgenol* 1989; **153**: 1141–1147.
34 Chiang PH, Chiang CP, Wu CC, Wang CJ, Chen MT, Huang CH *et al.* Colour duplex sonography in the assessment of impotence. *Br J Urol* 1991; **68**: 181–186.
35 Pickard RS, Powell PH, Zar MA. The effect of inhibitors of nitric oxide biosynthesis and cyclic GMP formation on nerve-evoked relaxation of human cavernosal smooth muscle. *Br J Pharmacol* 1991; **104**: 755–759.
36 Benson CB, Doubilet PM, Vickers Jr MA. Sonography of the penis. *Ultrasound Q* 1991; **9**: 89–109.
37 Lee B, Sikka SC, Randrup ER, Villemarette P, Baum N, Hower JF *et al.* Standardization of penile blood flow parameters in normal men using intracavernous prostaglandin E1 and visual sexual stimulation. *J Urol* 1993; **149**: 49–52.
38 Karadeniz T, Topsakal M, Aydogmus A, Gulgun C, Aytekin Y, Basak D. Correlation of ultrastructural alterations in cavernous tissue with the clinical diagnosis vasculogenic impotence. *Urol Int* 1996; **57**: 58–61.
39 Golubinski AJ, Sikorski A. Usefulness of power Doppler ultrasonography in evaluating erectile dysfunction. *BJU Int* 2002; **89**: 779–782.
40 Speel TG, van Langen H, Wijkstra H, Meuleman EJ. Penile duplex pharmacoultrasonography revisited: revalidation of the parameters of the cavernous arterial response. *J Urol* 2003; **169**: 216–220.
41 Cornud F, Amar E, Hamida K, Thiounn N, Hea Leanon O, Moreau JF. Imaging in male hypofertility and impotence. *Br J Urol Int* 2000; **86**(Suppl 1): 153–163.
42 Kropman RF, Schiper J, van Oostayen JA, Lycklama A, Nijeholt AA, Meinhardt W. The value of increased end diastolic velocity during penile duplex sonography in relation to pathological venous leakage in erectile dysfunction. *J Urol* 1992; **148**: 314–317.