Evidence suggests that arterial hypertension, in addition to being a cardiovascular and renal risk factor, may also be associated with an impairment of male sexual function. Since other cardiovascular risk factors, especially diabetes mellitus, have also been shown to correlate with impaired sexual function it has been proposed that sexual and especially erectile dysfunction (ED) may represent just another manifestation of atherosclerotic vascular disease. Male and female sexual dysfunction is more prevalent in hypertensive than normotensive individuals, and several mechanisms have been implicated in the pathogenesis of sexual dysfunction in hypertensive patients. Several factors affect the sexual function in hypertensives, such as the severity and duration of hypertension, age, and antihypertensive therapy. Older antihypertensive drugs (diuretics, beta-blockers, centrally acting) exert negative results while newer drugs have either neutral (Ca-antagonists, angiotensin-converting-enzyme (ACE)-inhibitors) or beneficial effects (angiotensin receptor blockers). Preliminary data from several randomized and open studies have suggested that angiotensin II (AT) 1-receptor antagonists may even be associated with an improvement of sexual function. Female sexual dysfunction, although more frequent than the male one, remains largely under investigated possibly due to the lack of effective treatment. A better understanding of sexual functioning and appropriate education of doctors at medical schools and specific seminars would result in a more effective approach of sexual dysfunction by practitioners dealing with hypertensive patients.

Keywords: Erectile dysfunction (ED), female sexual dysfunction, essential hypertension, hypertension treatment

Prevalence of Sexual Dysfunction in General Population

The prevalence of ED in the general population varies markedly among different countries. This could reflect different sample populations (age, cohorts of patients with recognized ED), different assessment methods (questionnaires, mail responses and telephone interviews), cultural differences in the willingness of individuals to discuss such issues and accept the social stigma of ED, and ethnic differences (genetic and environmental factors affecting erectile function). The Massachusetts Male Aging Study (MMAS) in 1994 was the first longitudinal, community-based, wide-scale epidemiological study of 1,290 men; the study reported an unexpectedly high rate of 52% ED prevalence. Since then, many studies have reported the prevalence of ED in the general population all over the world, ranging from 15% in Brazil to 74% in Finland. A recent study of 22,839 men in eight countries reported an overall prevalence rate of 15.5% male sexual dysfunction.

WHAT IS SEXUAL DYSFUNCTION?

Sexual dysfunction is defined by the World Health Organization as the various ways in which an individual is unable to participate in a sexual relationship as desired. Erectile dysfunction was formerly thought to be a psychological entity but currently, it is considered to be a disease of vascular origin. Erectile dysfunction is also considered to be an independent predictor of future cardiovascular disease, as it may be viewed as a manifestation of vascular dysfunction, and endothelial dysfunction underlies this symptom (Figure 1).

The relationship between essential hypertension and sexual dysfunction has raised a very important issue: is hypertension per se the cause of sexual dysfunction or do the drugs used in treating high blood pressure impair sexual function? Although several studies have addressed this topic, the question remains largely unanswered.

Figure 1: Endothelial dysfunction as a cause of erectile dysfunction
[Guay AT. Relation of endothelial cell function to erectile dysfunction: Am J Cardiol. 2005;96(Suppl 12B):52M-56M]
Prevalence of Sexual Dysfunction in Patients with Essential Hypertension

Although essential hypertension is widely accepted as a risk factor for ED, available data is in part controversial and indicate that this relationship is not definitely established. Some older studies have reported a similar prevalence of ED in hypertensive patients and normotensive subjects; however, the majority of the available data indicate that ED is more frequent in patients with essential hypertension when compared to normotensive subjects. In a study of 440 impotent men, arterial hypertension was not an independent predictor of vasculogenic ED (after adjusting for diabetes mellitus, hyperlipidemia and smoking), measured by penile arterial flow using duplex ultrasonography. Similar results were obtained in a study of 132 patients evaluated by duplex echo after intracorporeal papaverine injection, where hypertension alone was not an independent risk factor for vasculogenic ED. Larger epidemiological studies have found only marginal effects of hypertension on erectile function; only a “meager” relationship of ED and hypertension was observed in a study of 1,128 patients, aged 16–80 years. Similarly, the 9-year follow-up MMAS study reported that, although hypertension was an independent predictor of ED, its effect was modest (Table 1). However, the majority of epidemiological studies report an increased prevalence of ED in patients with essential hypertension. The relative risk of ED in hypertensives compared to normotensives ranges from 1.3 to 6.96.

The Treatment of Mild Hypertension Study (TOMHS) study was the first large hypertension study to report the prevalence of ED in hypertensive patients. The prevalence of ED at baseline was considerably low (14.4% in men and 4.9% in women); however, this could be due to several factors: (a) the study included only mildly hypertensive patients since diabetic and severely hypertensives were excluded, (b) there was only one question assessing sexual dysfunction without any particular interest or time consumed in that issue, (c) patients’ age ranged from 45 years to 69 years, excluding older patients, and (d) it is a considerably old study and patients were not at that time familiar with the issue and willing to accept it. In a recent open prospective study of 2,130 men with essential hypertension from Spain, ED was detected in 45.8% of hypertensive men. This percentage is significantly high compared to 18.9% that was found in the general Spanish population. However, although the difference of prevalence is too high, these are two different studies and differences in baseline characteristics may account for the difference in prevalence.

Impact of Duration of Hypertension

Duration of hypertension significantly affects erectile function. Studies addressing this issue in hypertensive patients reported that ED is more frequent and more severe in patients with long-standing hypertension (> 5–6 years) compared to patients with recent onset of hypertension.

Impact of Severity of Hypertension

Hypertension severity is associated with ED as well. It seems that as blood pressure increases erectile function decreases, and ED is more prevalent in patients with severe hypertension (Figure 2).

Hypertension in Patients with Erectile Dysfunction

In addition to analyzing the prevalence of ED and identifying comorbid conditions with a possible impact on the incidence and severity of ED, studies have been performed including men with impotence or ED only. Thus, in a study including 154 men aged greater than 55 years with ED, the prevalence of hypertension was 44%. In another study including 472 impotent patients, 117 (24.8%) had a history of hypertension. In a third study, the distribution of four main arterial risk factors (diabetes, smoking, hyperlipidemia and hypertension) was investigated in 440 impotent men (mean age 46.8 years). In this study, the frequencies of hypertension in the group of impotent men and in the general population were not significantly different. In summary, these three studies in patients with ED or impotence add little to the concept of an inter-relationship between ED and hypertension. This could, at least in part, be due to a bias in the selection of patients included in these cross-sectional analyses.

Female Sexual Dysfunction in Hypertension

The most widely accepted system for female sexual dysfunction was introduced in 1988 by the American Foundation for Urologic Disease. In the sexual arousal disorder classification, it is claimed that potential etiologies include hypertension, among others. However, data supporting this claim are substantially weak. It is generally accepted that the prevalence of sexual dysfunction in a disease is higher than the prevalence in the healthy population; however, data in hypertensive women are far from conclusive. No population-based controlled studies exist, capable of clarifying the association between hypertension, antihypertensive treat-ment and female sexual function. Existing data include: (a) the TOMHS study that, though a large double blind controlled randomized trial, has several limitations and (b) a rather small case-control study, addressing

### Table 1: Age adjusted probability of complete impotence in specific medical and psychological conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole population</td>
<td>9.6%</td>
</tr>
<tr>
<td>Treated heart disease*</td>
<td>39%</td>
</tr>
<tr>
<td>Treated diabetes*</td>
<td>28%</td>
</tr>
<tr>
<td>Treated hypertension*</td>
<td>15%</td>
</tr>
<tr>
<td>Untreated arthritis*</td>
<td>15%</td>
</tr>
<tr>
<td>Maximal Spielberger anger suppression or expression scale</td>
<td>16–19%</td>
</tr>
<tr>
<td>Maximum Jackson dominance scale</td>
<td>7.9%</td>
</tr>
<tr>
<td>Maximum Center for Epidemiologic Studies</td>
<td>41%</td>
</tr>
</tbody>
</table>

* Smoking doubled the prevalence in these conditions
female sexual dysfunction in 104 women with mild hypertension (67 on treatment) in comparison with 107 healthy controls. It was reported that hypertensive women experienced decreased vaginal lubrication, less frequent orgasm, and more frequent pain compared to normotensive women. The study revealed that it was hypertension per se resulting in female sexual dysfunction rather than the antihypertensive therapy; moreover, in a recent study examining postmenopausal women with heart disease, antihypertensive medication was not a predictor of sexual problems. In another study sexual dysfunction was found in 42.1% of hypertensive women compared to 19.4% of normotensive women, with an odds ratio of 3.2. Increasing systolic blood pressure, increasing age, and beta-blockers administration were significant predictors of sexual dysfunction, while adequate blood pressure control was related to lower prevalence of female sexual dysfunction. It suggests, rather, that no adequate studies have been performed and appropriate questions have not been asked. Sexual dysfunction must be routinely addressed in hypertensive patients in a very sensitive way, dedicating adequate time and using appropriate tools.

Erectile Dysfunction: An Early Marker for Hypertension?
Recent animal data suggest that ED may be an early marker for hypertension since the onset of ED is detectable before the onset of hypertension. Thus, physicians treating patients with ED need to be aware of the possibility of underlying cardiovascular disease, in terms of a holistic patient approach.

Pathophysiology

Male Pathophysiology in Essential Hypertension
The structural and functional abnormalities induced by hypertension may be implicated in the pathophysiology of ED. Based on the physiology of erection, any factor which impairs the (a) Signal transportation from the central nervous system to penis (b) The basal sympathetic tone in the flaccid state (c) The blood arterial inflow to the corpora and (d) The NO oxide (NO) production and release in the corpora may result in ED. Experimental studies indicate that hypertension results in:

- Structural changes in the penile vasculature, particularly cavernous vessels are affected by high blood pressure in the same way as vessels all over the vascular tree are. Recent data shows that apart from the marked vascular smooth muscle hypertrophy of the cavernous arteries, the smooth muscle layer in the cavernous space is increased in hypertensive compared to normotensive rats.
- Change in extracellular matrix morphology in hypertensive rats, since collagen type III fibers are significantly increased.
- Functional alterations in rat penile resistance arteries have been reported.
- The neurogenic relaxation in response to electrical field stimulation is impaired in hypertensive compared to normotensive rats, due to the attenuated relaxation in response to NO and this is particularly caused by the increased oxidative stress (as indicated by reduced superoxide dismutase activity and increased thiobarbituric acid reacting substance).
- Furthermore, the overproduction of endothelium-derived cyclooxygenase products in the corporal tissue results in increased

Figure 2: Percentage of men at baseline WHO reported problems with obtaining and/or maintaining an erection, by age and systolic pressure. [Grimm RH et al. Treatment of Mild Hypertension Study (TOMHS). Hypertension. 1997;29:8-14.]
vasoconstriction, thus rendering it more difficult for the corporal smooth muscle to relax and achieve an erection. In addition there is an alteration in the responsiveness to alpha-1 adrenergic stimulation of corporal smooth muscle in hypertensive rats, maybe due to a change in the expression of adrenoceptor subtypes.

**Evolving Role of Angiotensin II as a Mediator of Erectile Dysfunction**

Angiotensin II is known to induce contraction of the corporal smooth muscle in vitro and in vivo, via AT1 receptors. The human corpus cavernosum contains 200-fold higher Angiotensin II levels than the human plasma. Angiotensin II increases vascular hypertrophy in hypertension and induces endothelial dysfunction through NO reduction. Thus reticular activating system activation resulting in enhanced angiotensin II production may be responsible for the structural and functional changes in penile vasculature observed in hypertension. Interestingly enough the intracavernosal injection of angiotensin II decreases intracavernosal pressure and terminates spontaneous erection in anesthetized dogs; in contrast, the intracavernous injection of an angiotensin receptor blocker (losartan) increases dose-dependently the intracavernosal pressure. The role of angiotensin II in human erectile function has been recently established by Becker providing in vivo data. Angiotensin II levels in the cavernous blood were higher than in the peripheral blood of healthy volunteers; in addition, angiotensin II levels increased during the detumescence phase of erection, underlining the role of Angiotensin II in the termination of penile erection. Bradykinin is a potent vasodilator peptide that has been recently implicated in erectile function. Functional B2 kinin receptors have been found in the human erectile tissue and activation of these receptors result in NO release and subsequent corpus cavernosum relaxation. Since ACE-inhibitors result in angiotensin II decrease and bradykinin increase, their use in the treatment of hypertension may exert beneficial effects on erectile function, having advantage over utilization of angiotensin receptor blockers.

**Male Sex Hormone, Hypertension and Erectile Dysfunction**

The available data regarding the relationship between endogenous male sex hormones and blood pressure are controversial. Some studies show lower testosterone levels in patients with hypertension, while others show no significant difference between hypertensive subjects and normotensive subjects.

**Endothelin-1, Hypertension and Erectile Dysfunction**

Another possible link between ED and hypertension is endothelin-1 activity. Several studies indicate the importance of endothelin-1 in the regulation of smooth muscle tone of corpus cavernosum. It seems that endothelin-1 augments the contractile responses of other vasoconstrictors (like phenylephrine) present in the human corpus cavernosum.

**Female Physiology and Pathophysiology**

Although sexual problems are common in both sexes, female sexual arousal disorder is less well-characterized, understood and managed than its male counterpart, ED. Arousal disorders are usually organic and can often result from neural and peripheral vascular diseases, pelvic disorders and various medications, including antihypertensive agents (beta-blockers, clonidine, diuretics, calcium antagonists and alpha-blockers). The female genital arousal response is a neurovascular process characterized by genital engorgement, swelling and lubrication. Disorders of arousal include decreased labial and clitoral sensation and engorgement as well as lack of vaginal smooth muscle relaxation. It appears that NO plays a key role in clitoral smooth muscle relaxation, while its role in the vagina remains controversial. Phosphodiesterase-5 inhibitors result in significant increase of genital blood flow and vaginal lubrication. Functional adrenergic receptors are expressed both in clitoris and vagina and mediate norepinephrine-induced genital smooth muscle contraction. Preliminary data indicate that high blood pressure induces structural abnormalities in the female genital tissue that resemble the alterations observed in male hypertensive animals. Moreover, angiotensin II seems to play a pivotal role in the structural and functional changes of the clitoris and vagina while blockade of the renin-angiotensin axis protects the genital tissue from these abnormalities. Thus, the major players in female sexual dysfunction pathophysiology in hypertension appear to be NO, catecholamines and angiotensin II, just like in male sexual pathophysiology. Since research in female sexual dysfunction has lagged significantly, intense efforts are needed for the clarification of sexual pathophysiology in essential hypertension.

**Antihypertensive Medication and Sexual Dysfunction**

The old-generation centrally acting antiadrenergic agents such as methyl dopa and clonidine give rise to male sexual dysfunction, possibly by decreasing sympathetic outflow as well as diminishing libido and ejaculation. Diuretics, can all negatively affect sexual function. The new-generation antihypertensives, including calcium-channel blockers and ACE inhibitors, seem to have a neutral effect. Angiotensin receptor blockers may actually have a beneficial effect on sexual function. Beta-blocker use is associated with an increased risk of sexual dysfunction, and this even extends to some, though not all, of the newer-type beta-blockers particularly carvedilol, but precise data on this are lacking. Diuretics, including spironolactone, are one of the most implicated classes in sexual dysfunction, even when used as adjunct therapy. There are remarkably very little data on combination antihypertensive therapy which is problematic, as the majority of patients will eventually have to take combination therapy.

**Thiazide Association with Erectile Dysfunction**

Thiazide diuretics are commonly thought to disproportionally predispose to ED and sexual dysfunction compared with other antihypertensive agents, but a review of the evidence suggests that may not be the case. In the TOMHS study, chlorthalidone 15 mg was compared with placebo, acetubolol 400 mg, doxazosin 2 mg, amlopidine 5 mg, and enalapril 5 mg over 4 years. In this double-blinded clinical trial, a significant decline in sexual dysfunction was reported at 2 years with chlorthalidone compared with placebo that did not occur with the other comparators. However, there was no significant difference at 4 years. Most often, reports of sexual dysfunction on chlorthalidone did not lead to medication discontinuation. Long-term effects on sexual function were also reported in the Hypertension Detection and Follow-up Program (HDFP) comparing more aggressive “stepped care” therapy versus standard “referred care.” The first drug in stepped care was chlorthalidone 25–100 mg daily. The 4.4% discontinuation rate of chlorthalidone due to impotence and the 1.0% discontinuation rate due to decreased libido were not appreciably different from other drugs used in the HDFP. The Medical Research Council (MRC) trial was an older trial that used a higher diuretic dose than is currently recommended or available. A total of 22.6% of men reported impotence while taking bendrofluazide 10 mg daily compared with 10.1% of men taking placebo. The mechanism by which thiazide produce ED is unclear but it has been suggested that has a direct effect on vascular smooth muscle cells and/or to decrease the
response to catecholamines. Spironolactone, because it also affects the androgen receptors, can cause gynecomastia and feminization in general, testicular atrophy, and sexual dysfunction consisting of loss of libido and ED in males.60

**Beta-blockers Association with Erectile Dysfunction**

First- and second-generation beta-blockers have been widely associated with ED, but again the evidence for this association is not striking. In the previously noted MRC trial, there was no significant increase in ED compared with placebo using doses of propranolol as high as 240 mg daily61 (Table 2). In a large meta-analysis of 15 randomized trials using beta-blockers, which included more than 35,000 patients, the increased risk of sexual dysfunction was of borderline statistical significance, with a relative risk of 1.10 and a 95% confidence interval of 0.96–1.25.62 In four trials where withdrawal of study medication was noted, the annual absolute increase in beta-blocker withdrawal due to sexual dysfunction was 2 per 1,000 patients per year.62 Most of these trials included in this meta-analysis used high doses of first-generation beta-blockers.

In the TOMHS trial, there was no adverse effect on sexual function compared with placebo using acebutolol, a cardioselective beta-blocker. In the VAH 6-drug trial, atenolol 25–100 mg daily was not associated with increased impotence compared with placebo.63

**Psychogenic Factor in Beta-blocker-Associated Erectile Dysfunction**

Psychogenic ED has been estimated to comprise 70% of ED up to age 35 and 10% of ED past age 50, as vascular disease progresses.64 “Nocebo” effects significantly influence patient reaction to medication, and the volume of possible side effects listed in package inserts increase the likelihood of adverse reactions due to such nocebo effects.6566 A study by Silvestri and colleagues67 examined a nocebo effect causing ED in three groups of men aged 52±7 years with newly diagnosed hypertension or angina who were then administered blinded tablets of atenolol 50 mg daily. None had ED at baseline. All patients were advised that they were being prescribed a medication to reduce their chance of heart attack (Figure 3).

Group A did not receive any additional information. Group B was told they were being given atenolol, which was a beta-blocker. Group C was given the same information as group B and in addition instructed that “it may cause ED.” Results of the 90 days International Index of Erectile Function (IIEF) questionnaire showed ED incidence rates of 3% in group A, 15% in group B (P < 0.05), and 31% in group C (P < 0.01). In the second phase of the trial, new cases of ED in the first phase were randomized to placebo versus sildenafil, and there was no significant difference in ED reversal. Therefore, the placebo effect successfully treated the nocebo effect, and ED was shown to be psychogenic in the vast majority of these middle-aged individuals when ED was associated with beta-blocker initiation.

Antihypertensive drugs less likely to cause sexual dysfunction are:
- Angiotensin-converting-enzyme-inhibitors
- Calcium-channel blockers

**Evidence favoring antihypertensive drugs that may improve ED:** A few classes of antihypertensive drugs have been promoted for possible beneficial effects on sexual function, particularly the peripheral adrenergic blockers, angiotensin receptor blockers (ARBs), and nebulol. Angiotensin-converting-enzyme-inhibitors and calcium-channel blockers have been generally regarded as neutral.6869 In the TOMHS study, although there was no significant difference in sexual dysfunction at either 2 or 4 years between placebo and doxazosin, there were a few cases where baseline ED was reversed on doxazosin.69 A few similar cases have been reported in other studies, perhaps due to drug effect reducing penile sympathetic nervous system input.70 However, peripheral adrenergic blockers should not be used as antihypertensive monotherapy,71 and the overall side effect profile affecting quality of life is not as attractive as other drugs. Pharmaceutical industry interest in the area of antihypertensive drug-related sexual dysfunction has been focused on ARBs and nebulol. An increasingly accepted assessment instrument, the IIEF, has been developed with pharmaceutical sponsorship to promote further research in this area.72

Although ARB action against angiotensin II is a reasonable physiologic explanation for improved tumescence, ACE inhibitors act in a similar fashion. It is difficult to explain ARB-related increased “sexual fantasies” in one study,73 and perhaps not so difficult to explain ARB-improved sexual function in an unblended study with pharmaceutical grant sponsorship.74 The Antihypertensive Treatment and Lipid Profile in a North of Sweden Efficacy Evaluation (ALPINE) study was unable to detect a difference in sexual satisfaction between candesartan and hydrochlorothiazide.75 In all, literature favoring ARB antihypertensive treatment for sexual dysfunction is probably insufficient to recommend a proposed algorithm for switching to ARBs when dysfunction arises.76,77 Nebulol is a new third-generation beta-blocker with the additive effect of vasodilatation attributed to generation of NO. There have been a few pharmaceutical industry sponsored studies showing reduced ED in small number of men treated with nebulol, based on the physiologic rationale that the local generation of NO is the final step in the pathway for penile erection.78-80 Larger studies will be necessary to determine whether this potential benefit is in fact significant.

**Phosphodiesterase-5 Inhibitors**

Phosphodiesterase-5 (PDE-5) inhibitors are effective for treating ED. Usually, use of these drugs leads to clinically insignificant reductions in blood pressure, but it can have deleterious effects on patients taking complicated, multidrug antihypertensive regimens. For the majority of such patients, the blood pressure reduction will be minimal, and quite safe. The use of alpha-blockers may lead to a significant interaction with PDE-5 inhibitors, while that of organic
Cardiac Risk of Sexual Activity in Hypertensive Patients
Sexual activity doubles the risk of a cardiac event through sympathetic nervous system activation. However, since the absolute risk for a cardiac event is extremely low in subjects without cardiovascular risk factors (one in a million) it seems rational to assume that sexual activity is rather safe in low-risk individuals. According to the Second Princeton Consensus Conference patients with controlled hypertension are considered low-risk patients and may safely proceed to sexual intercourse. On the contrary, high-risk patients have a 10-fold increased risk for a cardiac event during the sexual intercourse and the following 2 hours. Thus, patients with untreated, poorly controlled, accelerated, or malignant hypertension are considered high-risk patients, and sexual activity should be deferred until the patient’s condition has been stabilized by treatment or a decision has been made by a cardiologist and/or internist that sexual activity may be safely resumed. Since many hypertensive patients have an easy access to phosphodiesterase-5 inhibitors through the internet, without previous medical counseling, hypertension specialists need to pay special attention in this issue in order to avoid undesirable incidents.

Management of Sexual Dysfunction in Hypertensive Patients
Clinicians have to respond to hypertensive patients with sexual complaints requesting treatment. From a practical viewpoint, management of dysfunction should be based on an analysis of its pathogenesis, keeping in mind the following guidelines:

Flow chart 1: Algorithm for management of hypertensive men with and without sexual dysfunction

1. Hypertensive patients
   1. Medical and sexual history
   2. Physical examination

   Sexual complaints

   Receiving drug treatment

   Yes

   No

   Hypotensive drugs can be discontinued

   Yes

   No

   Discontinue drugs
   Sexual complaints

   Blood pressure controlled

   Yes

   No

   Change medications to control blood pressure
   Sexual complaints

   Persistent sexual complaints requires detailed sex evaluation

   Yes

   No

   1. Blood pressure controlled
   2. Normal sexual function

   Receiving drug treatment

   Yes

   No

   Initiate treatment

   Sexual complaints

   Yes

   No
A detailed sexual history should be obtained in patients with new onset hypertension before initiating drug therapy. The sexual complaints of patients on medications are not necessarily a drug side effect. The least number and lowest dose of blood pressure medications should be used to manage hypertensive patients. Patients not taking hypotensive medications because of side effects should be followed-up regularly. The history should be followed by a targeted physical examination, with special attention to peripheral vascular disease, testicular size, fibrotic plaques on the penis, and stigmata of hypogonadism. The algorithm (Flow chart 1) then may be used to render two groups of patients: (1) those with blood pressure controlled and sexual function maintained (this group needs ongoing monitoring), and (2) those with persistent sexual complaints (a) with single or combination drug regimens or (b) without drugs. This group requires detailed sexual evaluation and plans for follow-up care (Flow chart 2).

### Treatment of Sexual Dysfunction in Hypertensive Patients

Therapeutic options remain limited despite the numerous methods available to determine the cause of ED in a given patient. It is quite well-established that lifestyle changes associated with reduced arterial blood pressure, such as initiation of physical activity and weight loss, favorably influence sexual function. These lifestyle changes seem to be most effective at younger age compared with older patients. A subgroup of patients with nonendocrinological organic dysfunction responds to orally administered yohimbine, an
alpha-2 adrenergic antagonist.49 Despite yohimbine modest effect, it may be tried initially due to its ease of administration and safety. In patients not responding to yohimbine, papaverine hydrochloride, a potent vascular smooth muscle relaxant, may be used alone or in combination with phentolamine. The drug or drugs are injected into one of the cavernous bodies on each occasion that sexual intercourse is desired and should be used under close physician supervision. If there is failure to achieve adequate penile rigidity, the possibility of a surgically implantable penile prosthesis should be considered. Implant surgery should be performed after careful psychological screening of the patient and his sexual partner. For patients who refuse to take hypertensive drugs because of their possible effect on sexual function, measures to reduce cardiovascular risks, such as cessation of smoking, normalization of body weight, control of serum cholesterol, and reduction of salt intake should be attempted. Target organ damage in these patients should be assessed periodically.

CONCLUSION

Both male and female sexual dysfunctions are frequent in the general population. Essential hypertension relates to sexual dysfunction, as sexual dysfunction is more common in essential hypertension per se. A significant proportion of hypertensive patients experience sexual problems that impair their quality of life. The older type of antihypertensives all negatively influence sexual activity, while the newer types of drug have a neutral effect. Changing the antihypertensive drug class may improve sexual dysfunction. Phosphodiesterase type-5 inhibitor can safely be administered to hypertensives who are taking antihypertensive drug therapy. Caution is needed with alpha-blockers. Patients who are taking nitrates should not be prescribed a PDE-5 inhibitor. Erectile dysfunction per se is considered to be an independent predictor of cardiovascular risk, and such patients should be evaluated accordingly.

Practitioners should consider choosing an antihypertensive therapy with the lowest possible potential for sexual side effects in order to attain an optimum balance between antihypertensive efficacy and quality of life. Recent studies indicate that ARBs may offer a therapeutic option to prevent or correct ED in patients with hypertension. Angiotensin receptor blockers have been shown to positively impact several indices of sexual function and perceived quality of life, effects possibly attributable to blockade of the effects of ANG II in mediating penile detumescence.

REFERENCES
