

Review Article

VOIDING AND SEXUAL DYSFUNCTION AFTER CEREBROVASCULAR ACCIDENTS

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ABSTRACT

Purpose: We provide an up-to-date review of the urological manifestations of cerebrovascular accidents and their management, including sexual manifestations.

Materials and Methods: We performed a comprehensive MEDLINE search for peer reviewed articles using key words and incorporated these data with our experience with the treatment of patients in an acute stroke unit, rehabilitation unit and ambulatory care center.

Results: The knowledge of urological dysfunction after stroke is based largely on the evaluation of symptomatic patients. The predominant symptoms are urinary frequency, urgency and urge incontinence. Time after stroke has a significant influence on urological findings. Detrusor hyperreflexia is the most common urodynamics finding. Whereas the site and size of the stroke clearly have an influence on urological findings, to our knowledge the effect of the involved hemisphere is unclear. Urinary incontinence as an initial presentation in acute stroke is associated with a high mortality rate. Sexual dysfunction is common in men and women. Co-morbid conditions, such as diabetes mellitus, benign prostatic hyperplasia and urethral incontinence, may complicate evaluation and management.

Conclusions: A stroke has a profound effect on lower urinary tract function, sometimes resulting in significant morbidity. In well rehabilitated patients sexual dysfunction should be assessed and treatment may be considered to improve quality of life with safety. An overall conservative approach to management is recommended in the initial 3 to 6 months since improvement is common with time.

KEY WORDS: cerebrovascular accident, hyperreflexia, urinary incontinence

Cerebrovascular accident or stroke is a serious neurological event primarily affecting the elderly. Residual effects may be temporary or permanent and they differ in morbidity. Impairment may include the loss of memory, vision, speech, motor function and voiding control as well as sexual dysfunction. The latter 2 sequelae make cerebrovascular accident important to urologists.

A cerebrovascular accident is defined as the acute onset of a focal neurological deficit. It is caused by an occlusive event, such as cerebral embolus or atherosclerotic thrombus, or hemorrhage. More than 500,000 cerebrovascular accidents occur annually in the United States, and a third are fatal, another third necessitate long-term nursing care and the remaining third permit patients to return home, many at their previous level of function.¹ These rates make cerebrovascular accident the third leading cause of death in the United States² and lead to more than \$30 billion yearly in health care costs.³ Risk factors for cerebrovascular accident include hypertension, diabetes mellitus, smoking, high serum cholesterol, alcohol consumption, obesity, stress and a sedentary lifestyle.^{4–7} Recently the incidence of cerebrovascular accident in individuals older than 70 years and the mortality rate have been decreasing,^{8,9} but the disease burden remains high.

A cerebrovascular accident may have a profound effect on the genitourinary tract. A particular problem is voiding dysfunction, which may range from urinary retention to complete incontinence.^{10–14} Significant sexual dysfunction is an

other possible effect. However, because cerebrovascular accidents occur predominantly in the elderly population, evaluation and management are often complicated by coexisting genitourinary dysfunction, such as bladder outlet obstruction in men or stress urinary incontinence in women. Also, various co-morbid conditions, including diabetes mellitus, vascular dysfunction and coronary artery disease, may affect the genitourinary system. Furthermore, changes secondary to aging, such as bladder instability, change in circadian rhythm, hormonal decrease and cognitive impairment, may disturb the function of the genitourinary tract. The effects of any of these conditions may be erroneously attributed to a cerebrovascular accident and increase the difficulty of management.

Some urological findings help when assessing the overall condition of the stroke patient. Particularly urinary incontinence immediately after a stroke implies a poor overall prognosis.^{15,16} Wade and Hewer followed 532 patients beginning shortly after the onset of a cerebrovascular accident and observed that those with urinary incontinence within the first week fared poorly with half dying within 6 months.¹⁶ Incontinence at 3 weeks also predicted a higher risk of death as well as a lesser chance of regaining mobility. Moreover, of the 44% of patients with urinary incontinence 63% had an alteration in the level of consciousness, whereas only 1% of the 300 continent patients had a similar change. Taub et al reviewed 639 registered cerebrovascular accident patients for disability at 3 and 12 months, and noted that initial

incontinence was the best single indicator of future disability with 60% sensitivity and 78% specificity.¹⁵

Given these data it is clear that urologists are frequently called on to evaluate and treat the stroke patient. We review the pathophysiology of cerebrovascular accidents and associated voiding dysfunction. We describe the methods of evaluation and management of stroke with genitourinary sequelae, including sexual dysfunction.

METHODS

The MEDLINE database from July 1966 through February 2000 was searched using the key words cerebrovascular accidents; cerebrovascular accidents and treatment; cerebrovascular accidents, sexual dysfunction and treatment; cerebrovascular accidents and acute and chronic rehabilitative treatment; cerebrovascular accidents and neurogenic bladder; cerebrovascular accidents and voiding dysfunction; cerebrovascular accidents and bladder outlet obstruction; cerebrovascular accidents and urological treatment; and detrusor hyperreflexia and treatment. Preference was given to randomized clinical studies and articles that best describe the diagnosis and treatment used today. Additional nonpeer reviewed articles were obtained. Reports in languages other than English were included when an accurate translation was made. This information was corroborated by our extensive experience with treating stroke patients.

REVIEW OF RELEVANT URINARY NEUROLOGY

The central nervous system has an important role in regulating the ability of the bladder to facilitate urine storage and emptying. These functions may be divided into the suprapontine, pontine and spinal centers. Voiding is coordinated by the neurons of the pontine-mesencephalic gray matter or the pontine micturition center.^{17,18} Voiding depends on the spinobulbospinal reflex through the pontine micturition center after receiving input from the hypothalamus, thalamus, basal ganglia, cerebellum and cerebral cortex. Input from the suprapontine center is predominantly inhibitory but it also has a facilitatory action. The principal inhibitory areas appear to be the cerebellum, basal ganglia and cerebral cortex, while facilitation may be regulated by the posterior hypothalamus and anterior pons.¹⁷⁻²⁴ Most of the suprapontine input to the pontine micturition center is inhibitory and the interruption of this input by a cerebrovascular accident, Parkinson's disease or brain tumor may result in detrusor overactivity or hyperreflexia. This condition may manifest as urinary frequency, urgency and urge incontinence.

The pontine micturition center regulates efferent stimuli to the bladder and external sphincter by its medial and lateral regions. The medial region regulates motor stimuli to the detrusor muscle by overseeing communication between the reticulospinal tracts and sacral intermediolateral cells carrying the preganglionic parasympathetic neurons that supply the detrusor muscle. Electrical stimulation of the median region elicits a decrease in pelvic floor electromyography activity and urethral pressure with a concomitant increase in intravesical pressure. The lateral region regulates the corticospinal stimuli to Onuf's nucleus in the sacral spinal cord, which innervates the levator ani, affecting urethral pressure and levator ani electromyography activity but causing little or no elevation in intravesical pressure.¹⁸

PATHOPHYSIOLOGY OF STROKE AND NEW THERAPIES FOR ACUTE ISCHEMIC (OCCLUSIVE) STROKE

Cerebral angiography performed soon after the onset of symptoms of a cerebrovascular accident detects arterial occlusion in 80% of patients.²⁵ Thrombolytic recanalization of the occluded cerebral arteries in these cases before the in-

farcion is completed may decrease the degree of injury. Various anticoagulant or thrombolytic agents, including tissue plasminogen activator,^{26,27} heparin,²⁸ low molecular weight heparin²⁹ and aspirin,³⁰ have been subjected to randomized trials in patients with acute ischemic stroke with patient condition monitored using objective measures, such as the Barthel Index, modified Rankin Scale, Glasgow Outcome Scale and National Institutes of Health Stroke Scale. In the International Stroke Trial²⁸ and Chinese Acute Stroke Trial³⁰ aspirin administration begun early in hospitalization yielded a small benefit, namely a decrease of 10 deaths or recurrent strokes per 1,000 patients during the first few weeks of therapy. Neither heparin with or without aspirin nor low molecular weight heparin yielded significant improvement in the clinical outcome at 3 months. In 2 open label, dose escalation studies that emphasized therapy within 90 or 180 minutes after the onset of symptoms tissue plasminogen activator delivery produced early neurological improvement.^{31,32} A randomized double-blind trial performed by the National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group showed no statistical difference in the degree of neurological improvement after 24 hours in the groups given tissue plasminogen activator or placebo. Symptomatic intracranial hemorrhage occurred within 36 hours of the onset of stroke in 6.4% of tissue plasminogen activator treated patients versus 0.6% in the placebo arm ($p = 0.001$). Mortality at 3 months was 17% in the tissue plasminogen activator arm versus 21% in the placebo group ($p = 0.3$). However, patients treated with tissue plasminogen activator were 30% more likely than placebo treated patients to have little or no disability at 3 months on the 4 assessment scales. The investigators concluded that despite the higher risk of symptomatic intracranial hemorrhage tissue plasminogen activator therapy initiated within 3 hours of symptom onset provided a significant improvement in the clinical outcome.

The medications ancrod and recombinant prourokinase are currently undergoing registration trials as thrombolytic agents to be administered within the 3-hour window of opportunity after the onset of stroke symptoms.³³ Ancrod is derived from the venom of a pit viper. It decreases the level of serum fibrinogen, which is responsible for clot formation. In an early trial 42% of the patients treated with ancrod during the window period recovered the physical and mental abilities they had before the stroke, whereas only 34% in the placebo group achieved a pre-stroke score. The dose of ancrod depends on patient serum fibrinogen concentration, which is monitored during the initial 3 to 5 days of hospitalization. By maintaining serum fibrinogen concentration between 40 and 70 mg/dl. it becomes possible to reverse the effects of an ischemic stroke while minimizing the chance of intracerebral hemorrhage or recurrent stroke. Similarly patients receiving prourokinase within the first 6 hours after symptom onset were more likely to recover the pre-stroke assessment score than those receiving a 4-hour infusion of heparin. Prourokinase is the first medication that helps to dissolve clots after the 3-hour window of opportunity has elapsed, making treatment appropriate in a larger group of patients. It must be administered directly into the area of the intracerebral clot and it becomes activated after it is released. As with other thrombolytic medications, the risk of hemorrhage is increased.³³ Much less progress has been made in decreasing the sequelae of hemorrhagic strokes.

PATHOPHYSIOLOGY AND PROGNOSIS OF POST-STROKE INCONTINENCE

Pathophysiology. Frontal cortical lesions from a cerebrovascular accident may affect higher cognitive function. This injury may be reflected by patient inability to suppress a reflex detrusor contraction, resulting in urinary inconti-

nence. The precise mechanism of incontinence may be detrusor hyperreflexia, cerebrovascular accident related language and cognitive impairment with normal bladder function or overflow incontinence secondary to detrusor hyporeflexia mediated by neuropathy or medication.¹³ Concomitant dementia, benign prostatic hyperplasia (BPH) or stress urinary incontinence may also contribute. However, it must be emphasized that incontinence in the cerebrovascular accident patient merits evaluation to determine its etiology even if one of these other conditions is known to be present.

Acute urinary retention is common immediately after a cerebrovascular accident. The neurophysiological explanation of this detrusor areflexia is unknown but the condition is referred to as cerebral shock.³⁴ However, acute retention is not necessarily the result of a cerebrovascular accident since it may be a consequence of patient inability to communicate the need to void, impaired consciousness, temporary over distention of the bladder or restricted mobility.³⁵ Comorbidities, such as bladder outlet obstruction and diabetic cystopathy, and the administration of medication with anticholinergic side effects may also be responsible. Urodynamic studies soon after a unilateral cerebrovascular accident demonstrated a 21% incidence of overflow incontinence due to detrusor hyporeflexia.¹³ However, a significant number of patients in this study had diabetes or were receiving anticholinergic medication, and so it is not clear to what extent these findings may be generalized. Our urodynamic evaluation of patients soon after cerebellar infarction has detected a high incidence of urinary retention. In contrast, cerebellectomy in an animal model resulted in hyperreflexia rather than areflexia.³⁶ Thus, the role of the cerebellum in voiding remains speculative, and more prospective clinical and animal studies are needed for clarification.

The symptoms of bladder dysfunction after an established cerebrovascular accident include frequency, urgency and urge incontinence.¹¹ These symptoms are generally a result of detrusor hyperreflexia.^{14, 37-39} Tsuchida et al evaluated 39 patients by urodynamics a mean of 19 months (range 11 days to 13 years) after a cerebrovascular accident and observed that 66% had frequency or urge incontinence, whereas the remainder had dysuria or urinary retention.³⁹ They determined that the symptoms of urgency or frequency are related to detrusor hyperreflexia, which may also contribute to urge incontinence.

Published studies assessing effects on the bladder have been performed largely in a retrospective fashion days to years after a cerebrovascular accident. Detrusor hyperreflexia has been the main finding in patients with voiding dysfunction.^{12, 14, 37, 39} Attempts to correlate the site of ischemic or hemorrhagic injury with urodynamic findings have had inconclusive results.³⁷ Burney et al prospectively studied 60 patients and correlated urodynamic findings with the site of an ischemic or hemorrhagic cerebrovascular accident determined by computerized tomography or magnetic resonance imaging within 72 hours of presentation.⁴⁰ If a patient had a confirmed infarct, he or she underwent filling and voiding multichannel cystometry with simultaneous electromyography recording using a needle electrode. Patients were then separated into 5 groups according to testing results (table 1). The majority of frontoparietal and internal

capsular lesions caused detrusor hyperreflexia and absent external sphincter volitional control, whereas patients with temporo-occipital lesions had normal urodynamic studies (table 2). These investigators also noted that 47% of their patients were in urinary retention and had overflow incontinence. Various lesions were responsible for bladder areflexia, including those of the frontoparietal area, internal capsule, basal ganglia, thalamus, pons and cerebellum (table 2). Of the hemorrhagic infarcts 85% were associated with an areflexic bladder, whereas only 10% of the patients with an ischemic cerebrovascular accident had detrusor areflexia (table 3). Whether the type of infarct determines the type of voiding dysfunction remains to be confirmed but areflexia has been identified after significant strokes of either variety.^{37, 41}

In the majority of patients after stroke electromyography reveals uninhibited relaxation of the external sphincter during or preceding detrusor contractions with resultant urinary incontinence.¹⁵ Interestingly detrusor-sphincter dyssynergia is uncommon in the post-stroke period and because of this low incidence, electromyography is not routinely performed.^{13, 42} However, pseudodyssynergia, characterized by voluntary contraction of the external sphincter during an involuntary detrusor contraction, may be more common in the recovery phase and should not be misinterpreted as detrusor-sphincter dyssynergia.⁴³ To distinguish detrusor-sphincter dyssynergia from pseudodyssynergia, it is important to correlate the onset of electromyography activity with the detrusor contraction. In detrusor-sphincter dyssynergia, electromyography activity precedes or accompanies the initiation of a detrusor contraction, whereas in pseudodyssynergia, the detrusor contraction occurs first. The patient senses the detrusor contraction and promptly attempts to suppress it by contracting the external sphincter. In pseudodyssynergia, electromyography activity occurs only after the detrusor contraction has begun and it acts as a voiding guarding reflex. Because of these confounding clinical possibilities, we recommend that there should be close supervision by the urologist of all urodynamics data and symptoms during a urodynamic study.⁴⁴ Table 4 lists the results of recent studies of the urodynamic effects of a stroke.^{14, 39, 45, 46}

Hemispheric dominance has been well established for language, while the temporal lobe has been linked to musical aptitude. Patients who have had a right hemispheric cerebrovascular accident have been known to incur more sexual dysfunction.⁴⁷ In 1980 Khan et al postulated that patients with a cerebrovascular accident in the nondominant hemisphere were less likely to have urinary incontinence.¹⁴ If this idea is correct, what effects would a dominant hemispheric stroke have on voiding?

Badlani et al retrospectively evaluated 44 symptomatic patients admitted to a rehabilitation unit after a stroke.¹² Mean age was 81.2 years and the time from cerebrovascular accident to urodynamic evaluation was 1 to 12 months. Table 5 lists the urodynamic findings accompanying right or left hemiplegia. The results were similar and Badlani et al concluded, as have others,^{14, 37} that there is no significant difference in the bladder dysfunction produced by a cerebrovascular accident in the dominant and nondominant hemispheres.

Prognosis. The likelihood of early post-stroke urinary incontinence is 57% to 83%.^{11, 35, 47, 48} There have been suggestions that incontinence is often transitory and related to patient immobility and altered mental status. For example, in a study of 151 patients Borrie et al reported an initial incontinence rate of 60% but after 1 month this incidence decreased to 29% and 66% with mild incontinence at 1 month had regained continence by 3 months.³⁵ Brocklehurst et al reported an initial incontinence rate of 39%.⁴⁹ However, by 2 months 55% of patients were continent and at 6 months this incidence had increased to 80%, although 2 and 3-year followup data demonstrated a higher incidence of incontinence than in the general population. Nevertheless, improvement

TABLE 1. Urodynamic findings on cystometry and electromyography⁴⁰

Group No.	Cystometry	Electromyography
1	Normal	Normal
2	Hyperreflexia	Normal
3	Hyperreflexia	Absent external sphincter volitional control
4	Hyperreflexia	Detrusor-sphincter dyssynergia
5	Areflexia	Normal

TABLE 2. Urodynamic findings according to cerebrovascular accident site⁴⁰

Lesion Site	Group 1	Group 2	Group 3	Group 4	Group 5
Frontoparietal	0	0	6	0	2
Occipital	1	0	0	0	0
Temporal	1	0	0	0	0
Multifocal	0	0	0	2	0
Internal capsule	2	0	12	0	4
Basal ganglia	2	2	3	0	4
Combined	0	0	1	3	0
Thalamus	2	0	0	0	2
Pons	0	0	2	0	3
Cerebellum	0	0	0	0	6
Totals	8	2	24	5	21

TABLE 3. Type of cerebrovascular accident and urodynamic findings⁴⁰

Cerebrovascular Accident Type	Group 1	Group 2	Group 3	Group 4	Group 5	Total No.
Hemorrhagic	1	0	1	1	17	20
Ischemic	7	2	23	4	4	40
Totals	8	2	24	5	21	60

TABLE 4. Urodynamic findings after cerebrovascular accidents

References	No. Pts.	No. Detrusor Hyperreflexia	No. Detrusor Hypocontractility	No. Uninhibited External Sphincter Relaxation	No. Detrusor Sphincter Dyssynergia	No. Electromyography	
						Normal	Not performed
Sakakibara ⁴⁵	14	9	3	3	7	7	0
	22	15	3	7	5	11	0
Kong et al ⁴⁶	27	11	1	NA	NA	NA	27
Tsuchida et al ³⁹	39	30	5	7	7	20	5
Khan et al ¹⁴	20	19	0	12	2	4	0

NA, not assessed.

TABLE 5. Urodynamic results according to side of hemiplegia¹²

	Men	Women
Rt. side:		
No.	13	11
Av. age	77	84
Capacity (ml.)	288	317
No. hyperreflexia (%)	6 (46)	6 (55)
No. areflexia (%)	2 (15)	3 (27)
No. normal (%)	5 (39)	2 (18)
Lt. side:		
No.	14	6
Av. age	78	86
Capacity (ml.)	210	416
No. hyperreflexia (%)	6 (43)	2 (33)
No. areflexia (%)	0	4 (67)
No. normal (%)	8 (57)	0

with time is impressive and the possibility of some or a complete recovery should be relayed to the patient and family.

Between 1950 and 1998, 79 studies investigated the predictors of good clinical outcome in patients receiving rehabilitative intervention after a cerebrovascular accident. Because of the heterogeneity of clinical outcome assessment, a meta-analysis was performed to provide a clearer understanding of rehabilitative services outcomes.⁵⁰ Strong predictors of a better outcome at hospital discharge and followup were greater functional skills at admission to rehabilitation, the early initiation of rehabilitation services and rehabilitation in an interdisciplinary setting. A weak correlation with a better clinical outcome was observed for specialized types of therapy and greater intensity of therapy services. The current literature is limited when assessing the outcome of specific types of non-inpatient rehabilitation services. More randomized studies must be done to clarify the effects of specific types of services.

SEXUAL DYSFUNCTION AFTER CEREBROVASCULAR ACCIDENT

Pathophysiology. Autonomic nervous system disturbances caused by a cerebrovascular accident adversely affect cardiovascular regulation,⁵¹⁻⁵⁵ sweating⁵⁶⁻⁶⁰ and sexual function.⁶¹⁻⁶⁷ The latter condition may present as poor or failed erection and ejaculation, decreased libido and coital frequency, and a substantial decrease in vaginal lubrication and orgasm. This sexual dysfunction is multifactorial and may be related to poor post-cerebrovascular accident coping skills, increased physical disability, fear of recurrent cerebrovascular accident with physical exertion, and decreased sensory and cognitive adeptness.⁶¹ Few studies have addressed these changes in a prospective manner with attention to such important factors as the impact on libido, sexual arousal, coital frequency, satisfaction with sexual activity and nocturnal penile erection.⁶⁸

Incidence. Korpelainen et al evaluated 38 men and 12 women 32 to 65 years old (mean age plus or minus standard deviation 53.5 ± 8.2 years) admitted to rehabilitation after a first stroke.⁶⁸ Only married patients with an active sexual life before the stroke and without other peripheral or central nervous system conditions known to affect the autonomic nervous system (diabetes mellitus), severe aphasia or psychiatric illnesses, or diseases affecting daily living activity were included in the study. Patients and spouses were evaluated prospectively for changes in libido, coital frequency, erection, ejaculation, vaginal lubrication, orgasm and satisfaction with sexual activity. After 2 and 6 months scores were reported on the Scandinavian Stroke Scale and Barthel Index. Patients and spouses independently completed the Zung Self-Rating Depression Scale⁶⁹ and a questionnaire describing sexual function and satisfaction before and after the stroke. Male patients at each interval underwent nocturnal penile tumescence studies using an erectometer for 3 consecutive nights and only the maximum measurement was used for statistical analysis. These patients were then divided depending on

penile expansion, including groups 1—greater than 16 mm. or normal, 2—greater than 2 to 15 mm. or impaired and 3—less than 2 mm. or no erection.

At 2 months 60% of the men considered their libido unchanged, whereas at 6 months this incidence was decreased to 49%. Concomitantly 38% of the men at 2 months and 51% at 6 months considered their libido decreased or completely absent ($p < 0.01$). A similar decrease in coital frequency was detected at 2 and 6 months compared with activity before the cerebrovascular accident ($p < 0.01$). At 2 and 6 months 28% and 14% of the men reported having ceased sexual intercourse, 8% and 6% reported complete impotence, and 18% and 12%, could not ejaculate, respectively. Nocturnal penile tumescence studies demonstrated that only 45% of the patients had normal activity. However, none were without nocturnal erections.

Female patients reported decreased vaginal lubrication and ability to achieve orgasm. At 2 months of followup, 46% of the women described decreased or markedly decreased vaginal lubrication and at 6 months this rate had increased to 50%. Before the stroke all female patients attained orgasm but at 2 and 6 months 30% ($p < 0.05$) and 20% ($p < 0.01$), respectively, did not.

Of all patients 90% reported having been very or moderately satisfied with sexual life before the stroke and none reported complete dissatisfaction. However, at 2 months 40% ($p < 0.01$) and at 6 months 42% ($p < 0.01$) of patients reported moderate or complete dissatisfaction with sexual life. The reasons for dissatisfaction included hemiparesis in 55% of cases, spasticity in 29%, decreased libido in 26%, fear of new stroke in 24%, sensory deficits in 19%, impotence in 14% and aphasia in 14%. Changes in sexual function were not statistically related to patient outcomes on the Scandinavian Stroke Scale, Barthel Index or Zung Depression Scale, or to prescription medications that may cause sexual dysfunction, such as antihypertensives, antidepressants and antipsychotics. Patients with sensory deficits had more sexual dysfunction than those with normal sensation with a statistically significance difference in libido, erectile function, ability to attain orgasm and satisfaction with sexual life. Sexual satisfaction of the spouses had also decreased significantly ($p < 0.05$) at the 2 and 6-month followups with 61% reporting decreased (28%), markedly decreased (15%) or absent (18%) libido.

Evaluation and management. The urologist should inquire into the sexual health of male patients who after a stroke are ambulatory with good mental status and manual dexterity and who have been clinical stable for at least 6 months. While making this sympathetic inquiry, a part of the interval history and physical examination may begin to address and improve an important quality of life issue after stroke. Performance status regarding exercise is an important consideration since sexual activity entails physical exertion. Physical limitation may be addressed as part of counseling. The desire for treatment is the basis for evaluation and therapy. Voiding dysfunction, especially incontinence, must be treated before addressing sexual dysfunction. Intimacy rather than intercourse should be the goal. Erectile dysfunction is no different in the cerebrovascular accident patient than in any other patient group. Vascular dysfunction is the most likely etiology. Hormonal evaluation⁷⁰ is indicated with decreased desire, although depression secondary to disability may be a cause of decreased desire.⁷¹

Therapeutic modalities of oral medication,⁷² injectables⁷³ and surgery therapy must be individualized. Cardiac evaluation before sildenafil administration is recommended because these patients are generally on a number of medications. A significant number of patients are on antiplatelet and anticoagulant drugs. This use of vacuum constriction device⁷⁴ is rare, giving injectables is selective and partner training for injection may be helpful. In stable younger pa-

tients after cerebrovascular accident we have done surgical implantation. A semirigid prosthesis is preferable due to physical limitations.

There are many anatomical, embryological and physiological similarities in men and women. However, in regard to sexual dysfunction in women its causes are more multifaceted and treatment remain less clearly defined than in men.⁷⁵ Only recently have its basic science and pharmacological properties begun to be elucidated. Perhaps in the near future sildenafil,⁷⁶ methyltestosterone, L-arginine, prostaglandin E1, phentolamine and apomorphine may have their roles determined.⁷⁵ Currently we limit our intervention in women to offering oral or vaginal estrogen replacement therapy and sexual counseling.⁷⁵ More clinical prospective studies must be performed to evaluate whether sexual counseling or other intervention helps to improve patient and spousal outlook after a cerebrovascular accident.

EVALUATION OF VOIDING DYSFUNCTION IN THE STROKE PATIENT

The 1990s witnessed the implementation of a multidisciplinary approach to the evaluation of stroke patients.⁷⁷ The neurologist, internist and physiatrist have key roles, whereas the urologist is consulted when needed. In most instances urological evaluation begins when a patient is transferred from an acute care facility to a rehabilitation or stroke unit. As in any other disease process, assessment begins with the history predating the stroke and proceeds with a laboratory evaluation and specialized tests, such as urodynamics and endoscopy.

There often are special problems when evaluating the elderly. These problems may be broadly categorized into obtaining an adequate history, technical difficulty with studies, interpreting a urodynamic study, that is distinguishing changes incidental to aging or co-morbidities from those resulting from stroke, and coexisting etiologies. When urodynamic studies are indicated, one should begin by simple screening with uroflowmetry and post-void residual urine measurements, and proceed to complex studies such as multichannel or video urodynamics only when needed. Evaluation and the extent to which it is performed depend not only on the likely diagnosis, but also on patient co-morbid conditions, level of functioning, and patient and family wishes.

History. Diagnosis largely depends on the history of the symptoms. However, elderly patients often under report problems, especially those related to urinary incontinence. Therefore, a patient interview may not provide sufficient information. Difficulty in obtaining a history is often compounded in patients with a dominant hemispheric cerebrovascular accident who have aphasia and in those with dementia. Interviewing the caregiver (spouse, relative or nurse's aide) may be necessary to obtain the desired information. A questionnaire given to the family with a voiding chart that is brought back on the second visit is helpful for assessing ingested fluid and functional bladder capacity. The first visit is used to rule out reversible causes of incontinence, such as infection, stool impaction and medication. Targeted questions are helpful in relation to symptoms such as urinary frequency. It is prudent to ask whether frequency is associated with high or low volume, whether it occurs in the day or night and whether it is associated with dysuria.

Selective nocturia in the elderly may be caused by increased urinary output during the night secondary to a loss of circadian rhythm or peripheral pooling of fluid, which is mobilized with recumbency. More importantly, nocturia may simply result from a poor sleep pattern. Weiss et al proposed a nocturia classification system.⁷⁸ The charts of 200 patients were reviewed for history, voiding data, including day, night and 24-hour voided volumes, post-void residual urine volume and video urodynamics. A 3-tiered classification system was

presented, including 1) nocturnal polyuria, in which urine volume voided during sleep exceeds 35% of the 24-hour output, 2) nocturnal detrusor overactivity, defined as nocturia attributable to decreased bladder capacity during sleep, and 3) mixed nocturnal polyuria and detrusor activity. Polyuria was defined as 24-hour urine output greater than 2,500 ml. and is classified separately. In their series 7% of the patients had nocturnal polyuria, 57% had nocturnal detrusor overactivity and 36% had mixed nocturia, while 23 also had polyuria. This study clarifies that nocturia is often multifactorial and may be unrelated to urological disorders. Prescription and often overlooked over-the-counter medications may have urological implications.

Physical examination. Specific attention should be given to the mobility, visual acuity, hand coordination and mental status of the patient in addition to digital rectal examination for pelvic floor assessment in women and prostatic evaluation in men. A regional neurological examination allows assessment of the patient ability to isolate the pelvic floor and voluntarily contract the muscles. Urinalysis for infection and hematuria follows. When hematuria is present, assessment by cytological study, excretory urography and endoscopy should be considered.

Urodynamic studies. The most useful urodynamic study is cystometry, which should be done as screening with post-void residual urine volume measurement. If complex urodynamic testing is then required, it should be performed with fluoroscopy and the measurement of several other parameters, including the Valsalva leak point pressure. Electromyography is appropriate in relatively few cases since detrusor-sphincter dyssynergia is an uncommon finding after a cerebrovascular accident. Fluoroscopy is helpful for selecting cases for electromyography.

There are several technical difficulties when doing urodynamic testing in the elderly. Studies are tailored to individuals rather than applying the same set of studies in each individual. Often elderly patients have decreased mobility and, thus, may not stand or use the uroflowmetry machine. In such cases only a supine study may be possible. Every attempt is made to have them void before the study because post-void residual urine volume is an important measure in the elderly. Using a rectal pressure balloon is often mandatory to detect straining and for performing the Valsalva maneuver during cystometry. Despite these problems Resnick et al have demonstrated that it is feasible to perform complex urodynamic studies even in frail elderly patients.⁷⁹

The reported incidence of age related uninhibited contractions is 10% in women and 25% to 35% in men. Because uninhibited bladder contractions may occur in continent elderly patients, the significance of bladder instability alone in the presence of incontinence is unclear. An uninhibited contraction may be present independently of associated stress urinary incontinence or outlet obstruction. As mentioned, other age related changes include increased urine output at night, prostatic enlargement and changes in the urethra in women related to the loss of hormonal support. Thus, the urethral pressure profile may change as a normal part of the aging process rather than as a consequence of a cerebrovascular accident. Ultimately it is better not to rely on a single measure, but rather to examine the whole picture in the context of the associated conditions.

Special situations. BPH is a common clinical finding in a male patient who has some degree of incontinence after a cerebrovascular accident. In such cases one must wonder whether detrusor overactivity is secondary to the cerebrovascular accident, bladder outlet obstruction or each condition. A history of voiding disturbances before the stroke is helpful because after prostatectomy, cerebrovascular accident patients report persistent voiding disturbances and unsatisfactory bladder control. It is prudent to evaluate these cases using sophisticated urodynamic techniques at a slow fill rate

of 20 to 50 ml. per minute and rarely video urodynamics when there is a history of mixed incontinence, or urethral, prostate or bladder surgery. Another helpful measure is the flow rate. Patients with BPH generally have a low flow rate. Measuring the voiding urethral pressure profile during the voiding contraction, as described by Yalla et al,⁸⁰ allows one to detect the point of obstruction or ascertain whether the posterior urethra is isobaric with the bladder during voiding. This test requires fluoroscopy and a special catheter. It is also helpful to know the clinical effectiveness of anticholinergic drugs on hyperreflexia in this individual preoperatively. Did they decrease incontinence episodes or urgency and frequency, as attested by a diary? After prostatectomy 30% of patients with hyperreflexia may not respond and are significantly incontinent. Only those with significant detrusor hyperreflexia, decreased bladder capacity and poor compliance undergo urodynamics again after the initiation of anticholinergic drugs. The drawback of giving anticholinergic drugs preoperatively is that urinary retention may develop, and the patient and family must be prepared for this possibility. The use of minimally invasive techniques, such as temporary ContiCath[†],⁸¹ and prostatic stents^{82,83} may also be useful in these scenarios.

Detrusor instability occurs in 60% of men with bladder outlet obstruction.⁸⁴ After prostatectomy detrusor instability resolves in as many as 75% of cases. To our knowledge whether cerebrovascular accident induced detrusor hyperreflexia with concomitant bladder outlet obstruction improves after prostatectomy has not been clearly evaluated. Lum and Marshall,⁴¹ and Moisey and Rees⁸⁵ studied patients who underwent prostatectomy after cerebrovascular accident but they did not specifically examine detrusor hyperreflexia. They reported an incontinence rate of 50% and 60%, respectively, but did not perform urodynamic studies or examine mental status to determine etiology, such as detrusor hyperreflexia, absent external sphincter volitional control, stress urinary incontinence, external sphincter injury or dementia. Our urodynamics experience indicates only a marginal improvement in detrusor hyperreflexia relative to detrusor instability in patients with bladder outlet obstruction after prostatectomy. Many patients require anticholinergic therapy postoperatively.

A diagnostic dilemma in female patients is detrusor hyperreflexia with incontinence secondary to urethral hypermobility or intrinsic sphincter deficiency. Assessing stress urinary incontinence may be difficult when there is low volume bladder hyperreflexia. Conversely detrusor hyperreflexia may be missed when leak point pressure is low and the bladder is not filled to an adequate volume. The latter problem is easy to overcome by occluding the outlet with a Foley balloon. Low volume detrusor hyperreflexia with a coexisting open bladder neck at rest on fluoroscopy may be a difficult problem. Discerning the urodynamic component (detrusor hyperreflexia and/or stress urinary incontinence) is more problematic and it may require repeat testing after the patient has initiated anticholinergic medication. The repeat study often shows clinically significant improvement in bladder capacity and Valsalva leak point pressure may be assessed more accurately to detect concomitant stress urinary incontinence. We often perform video urodynamics in these cases of complicated disorders, such as mixed urinary incontinence, previous anti-incontinence surgery or continuous urinary incontinence. We have not noted that urethral pressure profiles add significant information.

MEDICAL AND SURGICAL MANAGEMENT

Anesthetic precautions and special measures for patients receiving anticlotting agents. Generally patient cognitive function combined with general physical health determines

[†] Conticare Medical Inc., Eden Prairie, Minnesota.

the aggressiveness of interventions after a stroke. Anesthesiologists recommend waiting 3 to 6 months after an ischemic stroke and 6 months to 9 months after a hemorrhagic stroke before giving elective general or regional anesthesia. We most often wait 9 to 12 months after a stroke before initiating any operative therapy. It is important to communicate the operative plan to all patient decision making care providers. If they agree with the plan, these physicians manage the medical problems while the patient is hospitalized.

Most cerebrovascular accident patients receive antiplatelet therapy and less often an anticoagulant. In many antiplatelet therapy is discontinued 1 week before surgery. A bleeding time study is performed on the day of surgery. Antiplatelet therapy may be safely resumed in 1 week. By 6 to 9 months most patients are no longer receiving anticoagulant therapy, and so it does not become a factor in surgical planning. In those who are still on warfarin administration should be discontinued 5 to 7 days before surgery. Heparin may be needed beginning the night before surgery with injections discontinued 3 hours preoperatively. Heparin is reinstated 24 to 48 hours postoperatively. If there is significant hematuria or other blood loss leading to a decrease in hemoglobin of greater than 2 gm./dl. during the first 12 hours, heparin or warfarin should generally be withheld until patient condition is stable. Many new laser treatments for bladder outlet obstruction do not necessitate the cessation of warfarin.

Management of nocturia. Managing nocturnal polyuria in the elderly often focuses on the daytime mobilization of edema from the lower extremities. When the patient reclines during sleep, these fluids mobilize naturally and increase nighttime urine output.⁸⁶ Current treatment for nocturnal polyuria includes evening fluid restriction,⁸⁶ mid to late afternoon or early evening diuretics,^{87,88} compressive stockings,⁸⁶ afternoon sleep or leg elevation,⁸⁶ imipramine,⁸⁹ antidiuretic hormone^{90,91} and nasal continuous positive airway pressure.⁹² When using fluid restriction or diuretics, the urologist or primary care physician should follow the patient closely for signs and symptoms of hypokalemia and dehydration. Oral or intranasal antidiuretic hormone has been useful with few side effects in the elderly.⁹³ However, close monitoring of serum electrolytes for hyponatremia is mandatory and this medication should not be given in patients with a history of congestive heart failure or hypertension.^{86,93} In some patients with obstructive sleep apnea nocturnal polyuria is mediated by plasma atrial natriuretic peptide. Using continuous positive airway pressure considerably decreases nocturia.

Nocturnal detrusor overactivity results in decreased bladder capacity. These patients often have underlying detrusor instability, bladder outlet obstruction, sensory urgency, infection or malignancy. Treating the underlying urological disorder may be expected to improve bladder capacity and decrease nocturia.⁸⁶ In mixed nocturia the predominant cause of nocturnal polyuria or detrusor overactivity may be treated first and the case may then be reevaluated. Another approach is that of Weiss and Blaivas, who recommended the initial treatment of the nocturnal polyuria component of mixed nocturia because the measures are noninvasive.⁸⁶

Retention and obstruction in men. During the period of cerebral shock and urinary retention clean intermittent catheterization every 4 to 6 hours may be a useful minimally invasive therapy. As spontaneous voiding returns and post-void residual urine volume is consistently less than 100 ml., clean intermittent catheterization may be discontinued. When clean intermittent catheterization is not feasible, an indwelling Foley catheter may be placed and changed once monthly.

For documented bladder outlet obstruction new medical and surgical therapies are becoming available. Long-acting α 1-blockers, such as terazosin⁹⁴ or doxazosin,⁹⁵ may be given if the patient is medically stable. These medications selec-

tively antagonize the receptors that mediate prostate smooth muscle tension. As a result, they improve the American Urological Association (AUA) symptom score, increase maximum urinary flow and decrease post-void residual urine volume. Because their efficacy is dose dependent, the dose must be titrated. However, side effects, including dizziness, fatigue, headache and postural hypotension, may preclude their use in the elderly, especially in those on other antihypertensive medications. Tamsulosin is a potent and selective α 1a antagonist that does not require dose titration.⁹⁶ In 3 randomized, placebo controlled studies the co-administration of tamsulosin had no clinically significant effects on the pharmacodynamic action of nifedipine, enalapril or atenolol and produced no clinically significant changes in the blood pressure or pulse rate.⁹⁷ In addition, the new drug did not change electrocardiography or Holter monitor results and did not increase the side effects of blood pressure medication.

Other options are temporary prostatic stents or catheters. The ContiCath, which bridges the space from the bladder neck through the prostate and ends proximal to the external sphincter, may be used as long as 30 days.⁸¹ The ContiCath permits voiding with much less discomfort and there are few contraindications to its use. However, in a multicenter prospective study of 24 patients with nonneurogenic retention lasting greater than 1 week or neuropathic retention only 3 patients voided after ContiCath insertion.⁸¹ Some temporary stents currently under trial have a narrow lumen and, thus, are prone to clot occlusion in patients receiving anticoagulants. Stents rely on active drainage, such that detrusor function is essential. Thus, they are not useful for clot retention secondary to detrusor failure.

In high risk patients a permanent prostatic stent may be placed. The UroLume[‡] is much like a temporary stent and may be placed using local anesthesia with minimal patient discomfort.^{82,83} It may be used in patients receiving antiplatelet therapy but not those on warfarin. Radial forces maintain the stent in the proper position and serum prostate specific antigen is not affected.⁹⁸ Irritative voiding symptoms usually resolve within 60 days. Amon Sesmero et al evaluated 78 patients, of whom 69 were at high risk (American Society of Anesthesiologists score IV) with a mean age of 79.8 years.⁹⁹ All but 4 men required a urethral catheter preoperatively and 4 had a provisional intraprostatic stent. All UroLume insertions were performed using regional or local anesthetic. At a mean followup of 15.3 months the mean flow rate experienced increased to 12.7 ml. per second and the International Prostate Symptoms Score had improved to 6.2. Three patients who presented with hematuria months after stent placement required hospitalization and 3 required explantation due to patient request, significant calcification and stress incontinence in 1 each.

Less invasive endoscopic procedures, such as transurethral microwave thermotherapy,¹⁰⁰ transurethral needle ablation,¹⁰¹ and holmium¹⁰² or indigo^{103,104} laser prostatectomy, may prove useful in high risk cases. Francisca et al evaluated 1,092 patients 5 years after transurethral microwave thermotherapy with the Prostatron 2.0 \S and noted a 2 to 3 ml. per second improvement in the average urine flow rate, a 5 or 6-point improvement in the Madsen symptom score and an absolute re-treatment rate of 26%.¹⁰⁰ Lower energy transurethral microwave thermotherapy achieves a good sustained subjective improvement in patients with moderate symptoms and low grade bladder outlet obstruction but it is less useful in those with severe symptoms, low maximum urine flow, a larger prostate and larger greater post-void residual urine volume. Patients may require more than 1 treatment, and so it is fortunate that this procedure may be performed using local anesthesia.

Issa et al prospectively evaluated 38 patients with a mean age of 65.5 years undergoing outpatient transurethral needle

[‡] American Medical Systems, Minnetonka, Minnesota.

[§] EDAP-Technomed, Lyon, France.

ablation with a transurethral prostatic block.¹⁰¹ There was a statistically significant improvement in the AUA symptom score, bothersomeness score and maximum urine flow. Median sonographic prostate volume was 35 cc and mean necessary volume of 1% lidocaine with 0.25% bupivacaine and 1:100,000 epinephrine was 40 ml.

Matsuoka et al reported on 103 patients with a mean age of 70.2 years who underwent holmium laser prostatectomy for bladder outlet obstruction under regional anesthesia with a followup of 3 years and compared them with 152 undergoing transurethral prostatic resection.¹⁰² Mean International Prostate Symptoms Score improved from 21.7 to 4.3 and the mean maximum flow rate improved from 7.3 to 17.8 ml. per second, while mean post-void residual urine volume decreased from 137 to 35.6 ml. The laser procedure involved longer operative time (83 versus 58 minutes), less estimated blood loss (54.5 versus 336 ml.) and fewer catheter days (2.6 versus 4.9). All 3 differences were statistically significant. The holmium laser prostatectomy group had no complications.

The efficacy of indigo laser prostatectomy in 25 patients was reported by Greenberger and Steiner.¹⁰³ The procedure heats the prostate to the point of necrosis while maintaining the urethral lining intact and preserving antegrade ejaculation. This procedure may be used in patients on anticoagulant or antiplatelet therapy. A catheter remains in place 3 to 7 days postoperatively. Greenberger and Steiner demonstrated an AUA symptom score improvement of 20.2 to 9.8 and an improvement in maximum flow rate of 8.3 to 12 ml. per second at 9 months. Only 6 patients (24%) were in urinary retention, which was transient.

For minimally invasive therapy we perform transurethral microwave thermotherapy and indigo laser prostatectomy. In patients at high anesthetic risk we perform transurethral microwave thermotherapy using local anesthesia, although more than 1 treatment session may be necessary. In patients who are fully anticoagulated we offer indigo laser prostatectomy with general anesthetic. We have not noted any bleeding complications while obtaining a good surgical result.

The urologist should refrain from performing transurethral prostatic resection for 6 to 12 months after a stroke because incontinence and morbidity may be increased. Lum and Marshall identified several variables associated with an unsatisfactory prostatectomy outcome in 50% or more of patients.⁴¹ Variables most closely correlated with poor clinical outcome (higher incidence of urinary incontinence) were patient age older than 70 years, worsening neurological symptoms, a cerebrovascular accident that was bilateral or involved the nondominant hemisphere, absent associated urinary symptoms and surgery within 1 year of a cerebrovascular accident.

Retention in women. Until recently women in urinary retention secondary to detrusor hyporeflexia had only the options of clean intermittent catheterization or an indwelling Foley catheter. Although clean intermittent catheterization offers excellent long range management, its use in cerebrovascular accident is often restricted by patient inability to perform this procedure because of hemiparesis or hemiplegia. Indwelling Foley catheters have the disadvantages of infection risk and discomfort.

An investigational therapy is the In-Flow device,^{||} a 24Fr valved intraurethral catheter with an intraluminal pump designed for an acontractile female bladder.¹⁰⁵ The catheter is fixed at the bladder neck by flexible silicone fins that open like flower petals inside the bladder and by an external tab at the urethral meatus. For urination the patient or caregiver holds a small battery operated magnetic unit near the lower abdomen close to the pubic area. This unit activates a valve in the catheter and drives an intraluminal pump to draw

urine from the bladder at a flow rate similar to that of normal urination. Madjar et al described their experience with this device in 92 patients.¹⁰⁵ At a mean of 7.1 days 49% of patients had had the device removed because of local discomfort or urinary leakage. However, at a mean followup of 7.6 months (range 2 to 26) 47 patients (51%) continued to use the device and were dry with complete bladder emptying. The insert was replaced periodically at a mean of 38 days to prevent salt deposition within and around the intraurethral portion. All 47 patients were satisfied with the insert and preferred it to the previous mode of therapy. Asymptomatic bacteriuria developed in 22 cases (47%). No dyspareunia was reported.

Oral or subcutaneous pharmacological therapy for detrusor hyporeflexia or areflexia. Because a detrusor contraction depends on stimulation of the parasympathetic postganglionic muscarinic receptors by acetylcholine or acetylcholine-like neurotransmitters, bethanechol chloride has been used to improve bladder emptying. Although attractive from a physiological and pharmacological standpoint,^{106,107} clinical efficacy has not been demonstrated in most trials.¹⁰⁸ To our knowledge no study has specifically addressed its use for detrusor hyporeflexia or areflexia after cerebrovascular accident. Finkbeiner extensively reviewed the literature on bethanechol.¹⁰⁸ Although it may increase intravesical pressure^{109,110} while decreasing maximum cystometric capacity,¹¹⁰ it does not significantly decrease post-void residual urine volume regardless of the dose or route of administration.^{109,110} Barrett performed a randomized double-blind study of 48 female patients without neurological disease or bladder outlet obstruction with a post-void residual urine volume of greater than 125 ml.¹⁰⁹ He noted no statistically significant difference among the treatment arms using 25, 50 or 100 mg. of the drug and the placebo, as judged by post-void residual urine volume, percent of post-void residual urine, mean flow rate or intravesical pressure at 100 ml. Wein et al performed a nonrandomized study comparing 12 normal women with post-void residual urine greater than 20% of bladder capacity with 27 without significant post-void residual urine volume who received 5 mg. bethanechol subcutaneously.¹¹⁰ They reported no statistically significance difference in the percent of post-void residual urine or the flow rate. However, a statistically significant decrease in maximum cystometric capacity was evident in each group. Significant increases were also present in intravesical pressure at 100 ml., pressure at maximum cystometric capacity and maximum urethral closure pressure. All patients experienced sweating and flushing but none refused further participation in the study. Other side effects of bethanechol include nausea, vomiting, diarrhea, bronchospasm, headache and difficult visual accommodation.¹⁰⁶ We have not observed that bethanechol is useful for treating detrusor hyporeflexia or areflexia after a cerebrovascular accident. There is anecdotal evidence of the efficacy of other oral cholinergic agonists, including the dopamine antagonist metoclopramide and the synthetic benzamide cisapride, for improving bladder contractility but formal clinical studies are lacking.^{111,112}

Incontinence in men and women. The urologist goal in incontinence is to restore a socially acceptable level of urinary continence while minimizing the risk of infection. Patients with detrusor hyperreflexia may be treated with timed voiding, concomitant fluid restriction and anticholinergic drugs, such as oxybutynin, dicyclomine hydrochloride and hyoscyamine sulfate. However, outlet obstruction in men, and detrusor hyperactivity with impaired contractility¹¹³ in men and women may result in urinary retention with the injudicious use of anticholinergic medication.

Tolterodine^{114,115} is our first choice for relieving detrusor hyperreflexia after a cerebrovascular accident. This agent, which received United States Food and Drug Administration approval in 1998, binds less to cholinergic receptors in the salivary glands than traditional drugs of this class. There-

|| Influence Medical Technologies, San Francisco, California.

fore, patients are bothered less by dry mouth and fewer discontinue therapy secondary to adverse effects.¹¹⁶ Traditionally as many as 43% of patients on oxybutynin terminate its use secondary to dry mouth. Appell reported on 1,120 patients randomized to 2 mg. tolterodine twice daily, 5 mg. oxybutynin 3 times daily or placebo.¹¹⁶ When adverse effects, dose decreases and discontinuation were considered, tolterodine was more efficacious than oxybutynin.

The traditional medication oxybutynin is now available in a once daily, controlled release formulation. In a multicenter, placebo controlled study patients on the long acting product reported fewer problems with moderate or severe dry mouth.¹¹⁷ To our knowledge its efficacy in neuropathy and cerebrovascular accident is not known. In women with detrusor hyperreflexia and stress urinary incontinence several oral medications, such as imipramine or phenylpropanolamine hydrochloride-guaifenesin, are available that increase bladder outlet resistance. However, their use may be limited in hypertension.

If medical and behavioral therapy fails in the female cerebrovascular accident patient with urethral incontinence, a few minimally invasive procedures are available that provide fair to excellent improvement. It is imperative that detrusor dysfunction should be addressed before treating urethral incontinence. Of the newer and refashioned procedures for types II (urethral hypermobility) and/or III (intrinsic sphincter deficiency) stress urinary incontinence that may be performed using local anesthesia are periurethral or transurethral collagen injection with a 20% to 30% 5-year success rate,¹¹⁸⁻¹²³ the Urosurge[®] device, which is an investigational self-detachable balloon system that is placed at the bladder neck under endoscopic guidance to create an effect similar to that of collagen therapy¹²⁴ and a pubovaginal sling with or without bone anchors using autologous rectus fascia or fascia lata,¹²⁵⁻¹³¹ allogenic donor cadaver fascia,¹³² or synthetic polypropylene¹³³ or polytetrafluoroethylene.¹³⁴ These pubovaginal sling procedures were historically reserved for patients with type III stress urinary incontinence or those with failed anti-incontinence surgery. More recently, Chaikin et al evaluated 251 patients with type II or III stress urinary incontinence and at a minimum of 1 year of followup noted a 92% cure or improvement rate.¹²⁶ Today the pubovaginal sling procedure is performed with minimal tension on the sling, and new onset incontinence and permanent urinary retention necessitating urethrolisis have been decreased to 3% and 2%, respectively.¹²⁶ Compared with autologous tissue, cadaveric or synthetic material decreases operative time, postoperative pain and cost without sacrificing patient outcome or increasing morbidity. The success and complication rates associated with polypropylene with bone anchors¹³³ have been excellent in this patient population.

Another new therapy for stress urinary incontinence is tension-free vaginal tape.^{135,136} In a multicenter trial of 131 patients the 1-year cure rate was 91% and 7% of patients improved, while only 2% had failure.¹³⁵ All patients received local anesthetic and mean operative time was 28 minutes (range 19 to 41). Of the patients 90% were discharged home within 24 hours without an indwelling catheter and only 4 patients needed catheterization for 3 or more days. In high risk patients tension-free vaginal tape may become the preferred means of treating each type of stress urinary incontinence.

Other therapies. Sometimes all that is necessary for successful incontinence therapy in the stroke patient is simple behavioral modification, such as fluid restriction and a timed voiding schedule, as are determined by a voiding diary. Many patients with dementia who no longer have socially appropriate behavior also benefit substantially from a prompted voiding schedule. Gelber et al reported that 37% of severely

handicapped (aphasia, dementia or immobility) incontinent patients had a normally functioning bladder and even these debilitated patients benefited from prompted voiding and fluid restriction.¹³ The drawback is that this method of management is labor intensive and most effective in a home environment with 1-to-1 patient attention.

In patients with bladder outlet obstruction or hyporeflexia in whom medical and/or behavioral therapy fails and who are poor surgical candidates there are some less than ideal alternatives. A carefully supervised indwelling Foley catheter may be considered with monthly catheter changes. However, there is a well-known risk of complications, such as increased detrusor hyperactivity, urinary tract infections, bladder calculi, squamous cell carcinoma or adenocarcinoma of the bladder, urethral erosion and fistula formation. Because the suprapubic catheter is more invasive and has a similar complication profile, it should be approached with caution. Condom catheters are available for men but disturbing problems, such as skin excoriation, urinary tract infection and troublesome device maintenance, may limit their usefulness.

Clean intermittent catheterization is another possibility in patients with overflow incontinence secondary to bladder outlet obstruction or detrusor hyporeflexia. The patient or caretaker requires proper instruction, manual dexterity and motivation to perform this minimally invasive procedure with success.¹³⁷ Whereas treatment attempts to cure a condition, management may aim to decrease the impact of voiding dysfunction and improve quality of life.

CONCLUSIONS

The urologist now has an important role in the multidisciplinary evaluation of voiding dysfunction in patients who have had a cerebrovascular accident. An accurate assessment of voiding dysfunction is feasible in most patients for implementing a logical therapeutic plan in conjunction with other caregivers. Accurate diagnosis and implementation of noninvasive or minimally invasive therapy by the urologist may improve patient self-esteem and quality of life as well as decrease morbidity.

This article is dedicated to the memory of my father, Sergio Fabian Marinkovic.

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